# TRANSACTIONS

# AMERICAN ACADEMY
*of*
# OPHTHALMOLOGY
*and*
# OTOLARYNGOLOGY

---

FIFTY-FIRST
ANNUAL MEETING

G. HENRY MUNDT, M.D.
*General Chairman of Arrangements*

---

PALMER HOUSE
CHICAGO, ILLINOIS
October 13-18, 1946

610.5
N3
N17
06

EDITORIAL BOARD

WILLIAM L. BENEDICT, M.D., *Editor in Chief,* Rochester, Minnesota

W. HOWARD MORRISON, M.D., *Associate Editor,* Omaha, Nebraska

HENRY L. WILLIAMS, M.D., *Associate Editor,* Rochester, Minnesota

THE BOARD OF SECRETARIES

*Printed by*
DOUGLAS PRINTING COMPANY, Omaha, Nebraska

PRINTED IN THE UNITED STATES OF AMERICA

Medical Hall Cupboard 1

S. E. Edgerly

# TRANSACTIONS

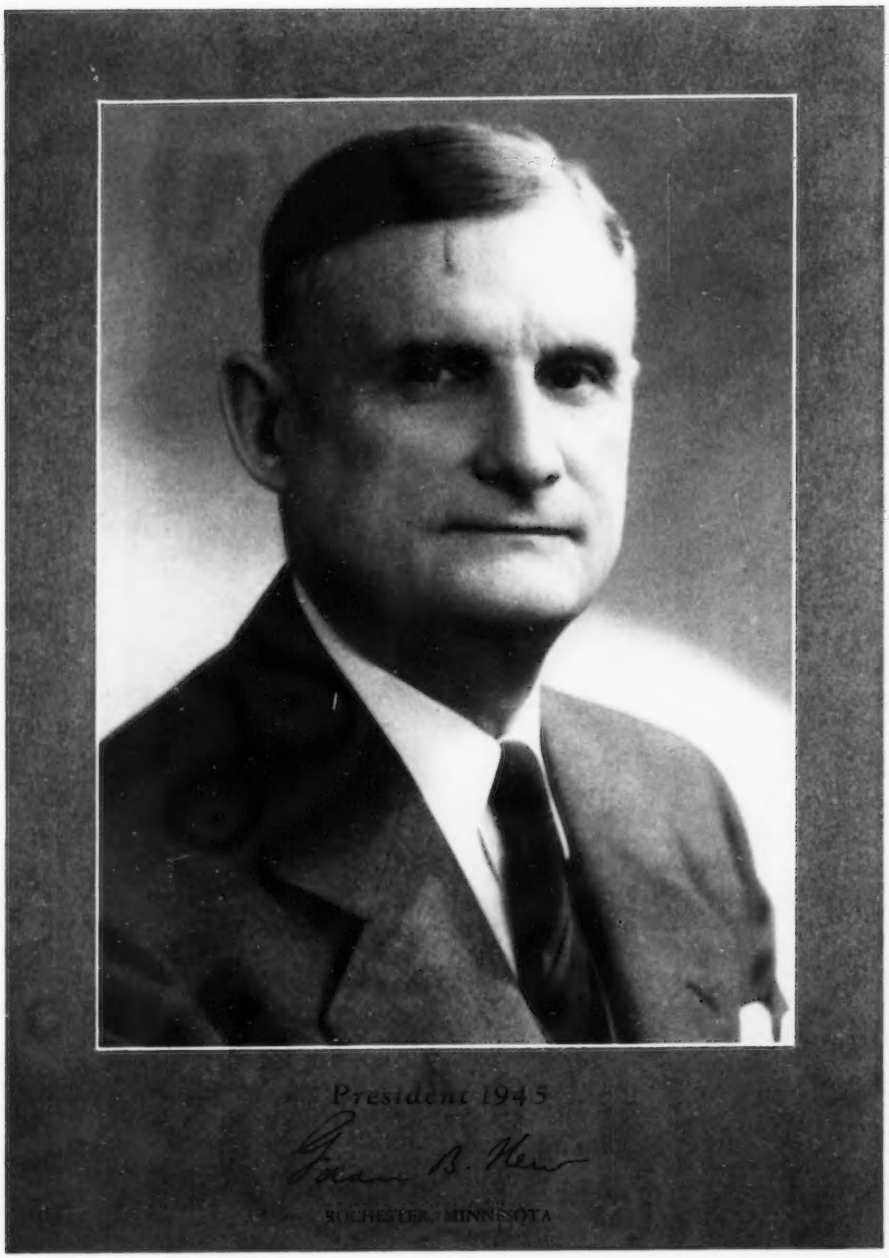
## AMERICAN ACADEMY
*of*
## OPHTHALMOLOGY
*and*
## OTOLARYNGOLOGY

Vol 51 No 1

SEPTEMBER - OCTOBER
*1946*

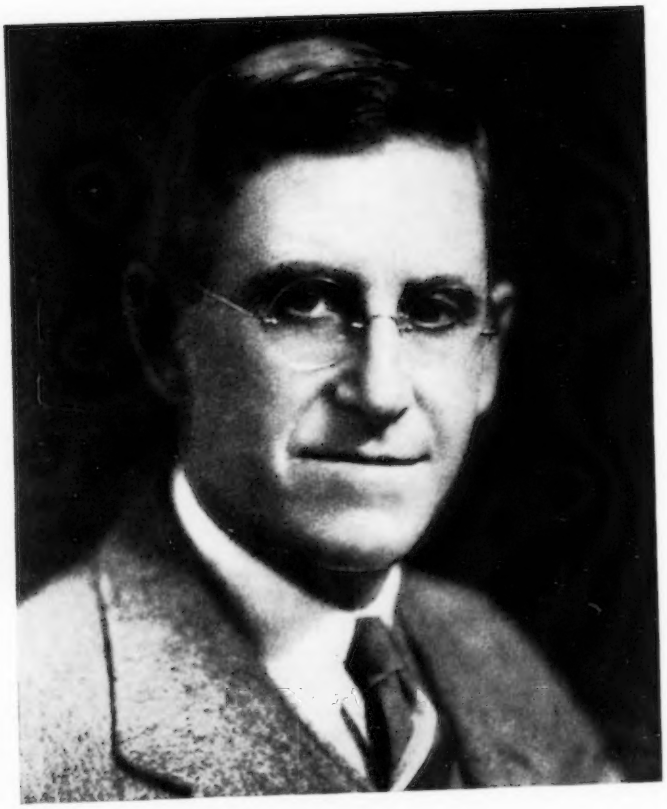
Published bimonthly, February, April, June, August, October, and December, by the Douglas Printing Company, 109 North 18th Street, Omaha 2, Nebraska, for the American Academy of Ophthalmology and Otolaryngology. Editorial and business office, 100 First Avenue Building, Rochester, Minnesota. Subscription price $3.00 yearly to members, $6.00 yearly to non-members. Entered as Second-Class Matter November 28, 1938, at the Post Office at Omaha, Neb., under Act of March 8, 1879.

ANNUAL MEETING OCTOBER 12-17, 1947, CHICAGO



President 1945

ROCHESTER, MINNESOTA



GEORGE MORRISON COATES, M.D.

PHILADELPHIA, PENNSYLVANIA

*Guest of Honor*

*Address of the President*

# CARCINOMA OF THE LARYNX: METHODS OF TREATMENT AND RESULTS

GORDON B. NEW, M.D.
ROCHESTER, MINN.

TEAMWORK is essential between the surgeon and the pathologist in the diagnosis of precarcinomatous and malignant lesions of the larynx. Teamwork among the surgeon, the anesthetist, the surgical assistants and the nurses in both the operating room and the hospital is necessary if the operative mortality rate is to be kept low. Teamwork between the surgeon and the radiologist is indispensable if all the patients are to get the best possible care. I shall return to this subject before I close.

## DIAGNOSIS

Carcinoma of the larynx is being diagnosed earlier today than in the past, due to the fact that physicians in general recognize that hoarseness may mean carcinoma and that the disease is curable. Histopathologic diagnosis is essential in all cases. This can be accomplished by examination of a fresh frozen section obtained at the time of laryngoscopy. It should be done to distinguish among epithelial hyperplasia, leukoplakia, an epithelioma in which the malignancy is of grade 1 or 2 or an inflammatory disease. Many times it is impossible clinically to make a definite diagnosis between a malignant process and a thickened lesion of leukoplakia. A specimen for biopsy should be taken only by the surgeon who is going to operate on the patient, since if it is removed by someone else before the surgeon sees the patient, it is often difficult for the surgeon to determine the extent of the lesion. Such a circumstance could jeopardize the patient's chance of a conservative operation.

In the presence of a suspected malignant process of the larynx, the whole situation should be carefully discussed with the patient and his relatives. Then, after the surgeon has examined the patient and has decided the procedure that probably is to follow, the patient and his relatives are told that when the biopsy is made, the pathologic report will aid in determining the best operative procedure. At times, a specimen for biopsy is obtained under suspension laryngoscopy, and the tumor can be removed at the same time. The physician who assumes the responsibility of treatment of patients who have precarcinomatous and carcinomatous conditions of the larynx should be prepared at all times to treat all types of lesions that present themselves. The surgical pathologist is an essential member of the team and his report on the fresh frozen section at the time of the operation is very important.

## TREATMENT

The patient who has a precarcinomatous lesion or a carcinoma of the larynx usually has but one good chance to get well. Perhaps he is advised to undergo some type of nonsurgical treatment on the assumption that, if the results are not good, surgical treatment can be employed. This is not a good plan to follow. Complete removal of the neoplasm is essential in any operative pro-

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

cedure. Unnecessary sacrifice of the voice, however, should be guarded against. The operation should be one that will best extirpate the disease, depending on the type of lesion, the grade of the carcinoma, its location and extent, the presence or absence of metastasis to cervical lymph nodes and the patient's age and general condition. All these factors must be carefully considered in the determination of just what treatment should be employed. A radical operation should not be employed when a conservative one will remove the neoplasm equally well and will leave the patient with a good functional voice. On the other hand, a conservative operation should not be used when a more radical procedure is indicated because of the activity of the neoplasm and the probability of extension of the disease beyond its apparent limits.

Certain conservative operations are justifiable in the treatment of carcinoma of the larynx, as is shown by the end results in selected cases. Selection of cases for conservative treatment, however, should be made only by a laryngologist who has seen a great deal of laryngeal disease. Small epitheliomas of the vocal cords, of low grade of malignancy can be removed by surgical diathermy under suspension laryngoscopy, with good results. At times thyrotomy is done to extirpate a very small, low-grade carcinoma of the vocal cords that could have been removed by suspension laryngoscopy and diathermy, with much less risk and with just as good a chance of cure. I wish to make it clear, however, that it is only the small lesions of the vocal cords that should be treated in this conservative way; in cases in which the neoplasm cannot be completely brought under inspection and in which there is any question as to its extent, thyrotomy should be done. Carcinoma of the epiglottis can be removed orally. Even in some cases in which the neoplasm involves the aryepiglottic fold and the lateral wall of the pharynx, the oral approach is advisable. After such a neoplasm has been removed with surgical diathermy, radon seeds are inserted into the base of the lesion and into the pharyngeal wall. Preliminary tracheotomy is carried out in all these cases of carcinoma of the epiglottis and in this group the results have been good, as previously reported.[1] Thyrotomy is done for epithelioma of the larynx which involves the anterior three fourths of the vocal cords, when the epithelioma is of low grade of malignancy. Surgical diathermy is used in all such cases. In a case of epithelioma of the larynx in which there is any question of extension of the neoplasm into the thyroid cartilage, the anterior third or half of the cartilage is removed on one side, or both sides, depending on the extent and the grade of the tumor. Half of both vocal cords, along with the cartilage, can be removed; this leaves a rounded opening posteriorly, through which the patient can breathe. A voice of fair quality will remain, so that in this respect the result is much better than it would have been had the patient undergone laryngectomy. Thyrotomy frequently is performed as surgical exploration of the larynx to render the neoplasm visible, since it is often impossible to determine otherwise, before the operation, whether the growth should be removed in a conservative way or whether laryngectomy should be done. Supraglottic neoplasms can be removed by thyrotomy if they are too extensive to be extirpated by laryngeal suspension. They do not all require laryngectomy, although, sometimes it is stated that they do require it.

Laryngectomy is used for growths that are fixed and which, by their fixation, give evidence that the muscle is involved. Also, for highly malignant tumors laryngectomy should be performed if there is any question of the extent

of the lesion. Removal of cervical lymph nodes is effected by block dissection if they are involved and if the neoplasm is of low grade of malignancy. In the presence of active lesions of high grades of malignancy, if cervical nodes are involved more is accomplished by irradiation than by surgical measures.

From 1934 to 1943, inclusive, 568 patients were treated for carcinoma of the larynx at the Mayo Clinic. Of these 446, or 78.5 per cent, were operated on and 122 or 21.5 per cent were treated by irradiation. One of the operations employed in the former group was laryngeal suspension together with insertion of radon seeds and the removal of the growth by diathermy. In some cases, irradiation was applied externally but the primary procedure was a surgical one. Other operations employed were thyrotomy and laryngectomy. Cases of the second group were those in which my colleagues and I felt that the patients would be benefited more by irradiation than by surgical measures. Patients in this group are not comparable to those for whom some physicians elect irradiation even though surgical measures could have been used. In many of the cases in which we ordered irradiation, the neoplasms were extensive and cervical nodes were involved.

Of the 446 cases in which surgical operation was performed, in 213, or 47.7 per cent, the operation was laryngectomy; in 184 cases or 41.2 per cent, it was thyrotomy and in 49 cases or 11.1 per cent, it was laryngeal suspension, removal of the growth by diathermy or insertion of radon seeds. Of the 446 patients who underwent operation, 90.4 per cent were males and 9.6 per cent were females; 80.9 per cent were between the ages of 45 and 69 years; 12.6 per cent were less than 45 years of age and 6.5 per cent were more than 69 years of age.

Of the traced patients who were subjected to laryngectomy, 60.2 per cent lived five or more years after the operation. Of these cases, in 46 per cent the neoplasms were epitheliomas of which the grade of malignancy was 1 or 2 and in 54 per cent, epitheliomas of malignancy grade 3 or 4.

Of the traced patients who were subjected to thyrotomy, 83.6 per cent lived five years or more after the operation. The neoplasms of 61 per cent of these were epitheliomas of which the malignancy was of grade 1 or 2; of 39 per cent, the neoplasms were epitheliomas of grade 3 or 4. Of the patients treated by means of suspension laryngoscopy, diathermy or insertion of radon seeds, 91.7 per cent lived five years or more after the operation. Of these, 82 per cent had epitheliomas grade 1 or 2.

The high percentage of cases in this group in which the neoplasms were of lower grades of malignancy, accounts for the very high survival rate. Of all patients subjected to operation and traced, 73.5 per cent lived five or more years after operation.

Of patients who we did not feel would be benefited by surgical measures and whom, consequently, we sent for irradiation, 46 per cent underwent preliminary tracheotomy and 6.9 per cent lived five or more years after treatment. Roentgen rays administered in fractional doses have been of great aid in treatment of carcinoma of the larynx; the condition has been cleared up by use of such treatment, in many cases in which surgical operation would not have been of any value. Some of the patients have stayed well for several years. None of them could have undergone a surgical operation such as laryngectomy secondarily, as is necessary in many cases, in which patients are referred to us after having received irradiation elsewhere.

Carcinoma of the larynx is a curable disease if the teamwork advocated at the beginning of this paper is applied. To make this point, of necessity I must

continue for a moment, to relate the experience of my colleagues and me over a period which includes that which has been the subject of immediately preceding paragraphs but which includes, also, the years 1944 and 1945.

Recently a study was made of 608 patients who had carcinoma of the larynx and who were operated on by Drs. Figi, Havens, Erich and me from 1934 to 1945, inclusive. During this period the operative mortality rate was 1.5 per cent. During the last seven years, 428 patients were operated on with only two deaths—both from coronary occlusion—an operative mortality rate of less than 0.5 per cent. The five year survival rate of the traced patients was 73.5 per cent.

If the results just recorded are considered extraordinary, the surgeons concerned do not attribute them to any extraordinary knowledge or skill possessed by them. Such results spring from intimate co-operation of pathologists, anesthetists, radiologists, surgical assistants, nurses and surgeons.

## REFERENCE

1. New, G. B. and Dorton, H. E.: Suspension laryngoscopy in the treatment of malignant disease of the hypopharynx and larynx. Surg., Gynec. & Obst. 72:930-935 (May) 1941.

*Address of the Guest of Honor*

# TWO PROBLEMS: CARE OF THE DEAFENED AND RHINOPLASTIC SURGERY

GEORGE MORRISON COATES, M.D.
PHILADELPHIA, PA.

To be elected a Fellow of the American Academy of Ophthalmology and Otolaryngology is in itself an honor eagerly sought by every young ophthalmologist or otolaryngologist. To attain this fellowship is one of the prime reasons why he seeks certification from either of the examining Boards, at the expense of years of hard work, financial sacrifice and strenuous effort, not unaccompanied by worry and trepidation. The honor of fellowship in this great Society, the most important and useful in its given field, in the world, is, thereafter cherished fondly throughout life. Because of our large membership, the honor of holding office in the Academy can, of necessity, be attained by a comparatively small number and, as a rule, is earned by long and arduous work in the affairs of the organization, membership on committees and in the strictly scientific work of the sessions and instructional courses. To be your Guest of Honor is the highest gift that the Academy can bestow and this recipient, however unworthy he feels himself to be, nevertheless has great pride in the award that you have made, which he attributes in great measure to your friendliness—that spirit of friendliness, cooperation and brotherhood, one of the outstanding characteristics of this great organization of ours of which we are all so proud. Thank you from the bottom of my heart.

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

What is it that has made our Academy the great driving force that it is and has given it priority over all other groups concerned with the sciences of ophthalmology and otolaryngology? There are many reasons for this but it seems to me that the chief one is that it has developed into a great postgraduate teaching institution, whose unique methods have been and are being adopted successfully by smaller societies far and wide. For that is just what it is, where thousands of our specialists congregate yearly to keep up to date on what is going on in the profession. This amazing teaching program offers instructional courses in literally everything pertaining to our twin specialties, including borderline subjects, taught by experts in their respective fields where the young resident or the man in practice for fifty years will find his greatest difficulty in choosing from so many fascinating subjects, from the basic sciences to advanced research work. Supplemented by the film-teaching program and the papers of the scientific assemblies with their competent discussions, he is a poor man indeed who will not keep every minute of the entire session occupied in collecting invaluable information, besides the stimulus achieved by meeting old and new friends and former teachers or students. The arduous work of the various scientific committees and the reports of research projects sponsored by the Academy add greatly to the knowledge of all those who attend the meetings and take the courses or who, unable to attend, read the Acad-

emy TRANSACTIONS. Few indeed of this latter group fail to plan to be present at the next convention.

Some years ago, Mosher, in discussing the inroads on the established practice of otolaryngology caused by the newer knowledge in the etiology and treatment of upper respiratory tract disease, warned that the field of the otolaryngologist was becoming more and more contracted, and advised the members of this specialty to broaden their interests to include more neck and possibly chest surgery, and to become more proficient in neuro- and intracranial surgery. After all this is a reversion, and a proper one, to conditions existing many years ago, for laryngology and rhinology developed from the chest upward. But even aside from this, the opportunities for the otolaryngologist today are far wider than might at first glance be thought, in spite of the fact that allergic studies and methods, as well as the spectacular advances of chemotherapy and biotherapy, have greatly reduced the surgery of middle ear and sinus infection and their formerly frequent complications. A glance at the titles of the instructional courses offered in the program of this meeting will convince the most skeptical that there is still plenty of work to be done. In the field that is broadening now rather than contracting, I only wish to speak briefly on two of the many subjects that might well be included and on only a few phases of these. Each one of them might well, and indeed frequently does, constitute a sub-specialty in itself.

### CARE OF THE DEAFENED

For many years studies for the prevention of deafness have been sponsored by the Academy but the results of this work have lagged far behind the work of the ophthalmologists for the prevention of blindness. For many reasons, the plight of those unfortunates who had been deprived of the inestimable boon of sight has had a greater emotional and sympathetic appeal than those with hearing deficiencies, which as a rule come on slowly and are not readily apparent to the casual observer. The work of our former president, the late Dr. Horace Newhart, laid the foundation for the present greatly increased interest in not only the prevention of deafness, but the care, if not the cure, of deafness already established. During the last two years, under the energetic leadership of Dr. Dean M. Lierle, great strides have been made towards the nationwide organization of otologists for not only the prevention of deafness but the care of the vast number of stricken individuals, young and old, in our ever growing population. Valiant cooperation in these efforts is being given by the American Society of Hearing, with its various Leagues or Chapters, under the inspired leadership of our fellow-member, Dr. C. Stewart Nash. This work has become one of the most important activities of the Academy and it should be the duty of every otologist amongst us, not only to familiarize himself with these activities but to join heartily in the program of his own state and community. It is acknowledged that the deafened individual is often a difficult person to work with, but if otologists everywhere would take an active interest in this effort, the mobilization of great numbers, working enthusiastically, would soon result in greatly alleviating the condition of these sufferers and enable the Committee to do in a short time what otherwise would be the work of years. Once a nationwide program is adopted and put in effect with a sufficient number of competent workers, there is no question that results will speedily follow. It is a fact that relatively few otologists are conversant with this program, are familiar with the work of the otologists' committee on audiometers and hearing aids of the Council on Physical Medicine of the Ameri-

can Medical Association or understand the mechanism and necessary upkeep and interpretation of results of the various audiometers on the market, or the now numerous hearing aids, good, bad and indifferent, offered by the manufacturers. To do these things takes time and constant study, but the up-to-date otologist will have to learn them if only for self preservation. Granting that, in the long run, little can be done for many advanced cases of progressive deafness (excepting otosclerosis for which surgery now offers great help) and that many of these cases come to us only when irreversable pathology has occurred, it is on the management of these cases in the private office or in the hospital clinic that I would now like to remark briefly.

War, in spite of its horrors, brings some blessings in its train. Research of all kinds takes on a quicker tempo and this applies in great measure to medicine in all its branches. The care of the sick and wounded is necessary not only for humanitarian reasons but in order that the solder may, if possible, return to duty in order to keep up the effective combat strength of his unit. If this is not possible, or when the fighting ceases, a more or less grateful community owes the disabled veteran everything that can be done in order that he may be rehabilitated to the greatest extent possible. The induction into the Armed Forces during the last two great wars of many cases of defective hearing, and even suppurating ears, is well known to every otologist in the Army and Navy, and to those who saw cases for the Veterans Administration after World War I. This was due in large measure to the careless and obsolete methods of testing the hearing of the inductees and against the protests of the Committee on Otolaryngology of the National Research Council. Part of it was unavoidable because of the necessity for speed and the shortage of audiometers and trained audiometrists. Many veterans with hearing defects in need of rehabilitation therefore had pre-existing hearing losses though frequently aggravated by service-connected acoustic trauma. Largely through the urging of civilian otologists in our national societies, a program for rehabilitation for these acoustically disabled veterans, whether or not service connected, was established well before the cessation of hostilities, and their most successful programs both in the Army at three centers, namely at Butler, Pa., at Chickasha, Oklahoma, and at Santa Barbara, California, and for the Navy at Philadelphia, have taught lessons that can be used, with necessary modifications, in civilian practice. Every otologist who has not done so should read the accounts of these service operations.

These four clinics operated under advantageous conditions in that their patients could be segregated in institutions, complete with most modern installations and facilities, and retained until the desired or optimum training could be accomplished. The patients lived together and were trained as a group with proper attention devoted to improving their existing morale, adequate psychiatric care, often needed, the immediate construction in the institution of individual ear moulds by trained technicians, the most careful and accurate testing of hearing for pure tone losses and for speech reception, both before and after the fitting of hearing aids, the careful training of the patient in the use of the hearing aid selected (and incidentally presented to him), a large selection of various types of hearing aids and training in lip and speech reading, which it is so difficult to persuade the average civilian patient to submit to but which is of inestimable value to the deafened patient either with or without a hearing aid.

How can this very successful program be put into practice by the otolo-

gist in civil life? How can the civilian otologist attempt to prescribe a hearing aid satisfactorily in a busy office and hospital practice, and find the time to educate his patient in its successful use? It is surely not enough to have an office assistant take a routine audiogram and tell the patient to try one or more aids fitted commercially and with little follow up. Often he purchases the first instrument offered to him, finds it unsatisfactory, frequently because he has not been trained in its use, even if it may have been of the proper type for his particular case, struggles with it for a while, and finally puts it in a bureau drawer and gives it up as a bad job. The hearing aids offered today are marvelous improvements over those of a decade ago before the advent of the vacuum tube and here again military-induced research has contributed greatly, but the fact remains that the vast majority of otologists have neither the time nor the facilities to properly care for those unfortunates. On the other hand, it is inconceivable that they should be turned over to the commercial concerns marketing the hearing aids, invaluable as their work and cooperation is.

In certain instances for some time past, local Leagues for the Hard of Hearing, located in large cities, have endeavored to help in the work of fitting hearing aids by having a central clinic, with more or less trained personnel and a number of different types and models of aids on hand for trial—these aids supplied by the manufacturers. Is not the time ripe to establish in all large cities, and in smaller cities or towns serving areas of considerable dimensions, rehabilitation clinics similar to those of the Army and Navy? Several articles have appeared advocating this course. The financial outlay would be considerable but might be met by contributions from philanthropic foundations or individuals, or by funds raised by cooperation of the local units of the American Hearing Society. Where possible University or Hospital facilities should be used and proper compensation given to the operating personnel, professional or lay. To such clinics patients should be referred by the otologist, and the operating cost covered by proper fees except in the case of the indigent. The staff need not be large at first and can be increased as the idea becomes better known and more popular. Equipment for careful pure-tone testing and for speech reception and intelligibility, a laboratory for making ear moulds, a good assortment of trial hearing aids including, of course, the latest Council accepted models would be necessary.

The staff should consist of one or more otologists, trained technicians for making the ear moulds, a psychiatrist who could be on a visiting or part-time basis, a lip or speech reading instructor, a speech correctionist and possibly, in a larger clinic, an electronic engineer. The patient, referred by an otologist, would go through a regular course of examination and indoctrination which the service clinics have found so useful for the morale of their rehabilitation cases, have the ear mold properly made and a general explanation given of the deficiencies as well as the merits of hearing aid wearing, and instruction as to its use, care and upkeep. All of this takes time that the otologist, busy in private practice, cannot afford to give, cannot contrive the space for the technical part of the work, or keep trial hearing aids on hand. In some few instances otologists such as the late Walter Hughson, have confined their practice to this type of work and have succeeded. Where no such practitioner exists in a given community and where there is no clinic of this type available, I commend to the otologist the report of your committee on the Conservation of Hearing, to be found in the first pages of the last issue

of the TRANSACTIONS. This report, as the result of careful study and deliberation, summarizes what the otologist, dependent on his own resources, should do for his hard-of-hearing patient, what instruction he can give him and how he can maintain the supervision necessary for a satisfactory result.

That this subject, at long last, has made its appeal to the otological branch of our profession cannot be doubted as witness the program arranged by the committee on the Conservation of Hearing and the numerous instructional courses offered on various phases of this problem, nine in all. These courses should be oversubscribed because I feel strongly that we all need whatever training we can ourselves get in putting this most important work on a sound practical foundation.

### RHINOPLASTY

With the advent of the sulfa drugs and penicillin, with more and better remedies of this nature in the offing, the surgical work of the otologist undoubtedly became much more restricted, and some pessimists felt that the heyday of the specialty was over and that we would revert to the status of the spraying specialist of the '80s and '90s. That they were wrong can easily be shown, although our instructional program is not overloaded with the technic of surgical procedures. Surgery for otosclerosis and for Meniere's disease is being increasingly employed by numbers of trained young otologists, and here the field is literally enormous. Surgery of the neck, of the larynx, trachea and bronchi (via endoral procedures), neoplastic surgery of the upper respiratory tract, surgery for chronic ear infections and their complications, will always be with us and surgery for acute mastoiditis will always be needed although in decreased numbers. Intracranial surgery when associated with ear infection falls within our province. Many of these types of surgery are borderline, done by the otologist, the chest surgeon, the neuro-surgeon or the general surgeon, depending largely on individual preference and special training. More and more in recent years the rhinologist has invaded the field of the general plastic surgeon by giving especial attention to reconstructive surgery of the nose and ears, two of the most prominent features of the face and head. Is he stepping out of his field in performing this type of surgery? Certainly not, since he has been closely identified with its development ever since the specialty of otorhinology was recognized. Indeed what otological surgeon does *not* do plastic or reconstructive surgery? We have all made minor or major corrections of nasal contours, employed skin grafts in aural and rhinologic surgery, done submucous resections of the nasal septum (a plastic procedure) or in older days corrected septal deviations by the methods of Asch, Gleason, Watson and others. We have used plastic procedures on the nasolacrimal duct, upon the external auditory canal, have corrected alveolar and postauricular fistulae and the defects caused by radical sinus surgery and have been pioneers in cleft palate and cleft lip corrections for many years. There has always been a lack of proper training opportunities for the otolaryngologist who desires to perfect himself in this branch of the specialty and here I enter a plea for improved facilities for this type of work. Look for a moment at the partial list of otolaryngologists who have made history in the evolution of plastic surgery of the head: John O. Roe, Mackenty, the senior Ballenger, Morley, Marshall, our distinguished President Dr. New, Figi, Beatty, Peer, Sheehan, Carter, Metzenbaum and Lee Cohen, to mention only a few Americans.

The general plastic surgeon has done a marvelous piece of work, in civilian

practice and both World Wars and as physicians we are proud of him. Often a dental or oral surgeon, he is superbly equipped for plastic work on the oral cavity and by further training for plastic procedures in any part of the body, including rhinoplasty. For the latter operation, however, the rhinologist through his training in and familiarity with nasal anatomy, physiology and instrumentation, definitely fits into the picture and with proper teaching is able to do excellent work in this field. What is the definition of rhinoplasty and what is its purpose? In former times, from ancient history to a comparatively recent era, it consisted in making a new nose to replace one destroyed by disease or trauma. The modern idea is indeed to correct unsightly nasal deformities that are conducive to lowering of morale and therefore the production of unhappiness, but more especially to improve or restore lost or impaired normal physiology, the normal functioning of the nasal structures in their properties of transmitting the proper amount of warmed, moistened and filtered air being paramount.

The newer studies of physiologists and anatomists within our own group have made it of prime importance that the operation should not ordinarily be done for simply beautifying purposes, although in certain cases this is justifiable, but especially to improve function by widening the nose, narrowing the nose, lifting the tip, narrowing or replacing the columella, making proper correction of the septum, correcting the action of the alar cartilages and opening the vestibule. Frequently these procedures alone, when done to improve physiological function, result in improved cosmetic results also. The introduction of the submucous resection of the nasal septum by Killian, Ballenger, Freer and others was a tremendous advance in the effort to obtain normal nasal breathing through obstructed nasal chambers, but it is not always successful unless accompanied by some of the rhinoplastic procedures just mentioned. Conversely, in many instances, a submucous resection is also needed to supplement the rhinoplasty.

A colleague of mine has defined rhinoplasty as follows. It is the specialty which deals with the study of normal and abnormal contours and their corrections, to be performed by removal, replacement and shifting of tissues. It has to do with the restoration of defects of nasal contour, whether congenital or acquired and in many instances the correction of nasal contour also improves nasal physiology. Almost always an improvement in breathing results if this is required. The point made here is that nasal physiology must always be respected by the rhinoplastic surgeon and in order to accomplish this he must in the first place know his nasal physiology and in the second place how to accomplish his aim surgically without harm to or with improvement in physiological conditions. The surgeon operating for chronic sinus infection or the removal of neoplasms has frequently, often of necessity, violated this rule. The rhinoplastic surgeon should never do so. He is working on displaced structures, not diseased ones, and when removal is indicated it is only because of redundancy.

In view of the undoubted growing interest in rhinoplasty by rhinologic surgeons, the question of providing proper training for young aspirants arises. The type of plastic surgery involved in otorhinolaryngological practice should receive more detailed consideration in our graduate courses, and in the instruction given to residents. A comprehensive knowledge of the anatomy and physiology of the parts involved is, of course, a sine qua non. The fundamental principals of plastic surgery should be taught

and the student given opportunity, not only to observe, but to assist in and finally conduct actual operations on the living subject, preceded by carefully conducted practice on the cadaver. This is exactly parallel to the teaching of all branches of surgery in our specialty. The American Board of Otolaryngology is urging that this training be extended to all candidates preparing for Board examinations, whether in postgraduate or graduate schools of medicine, in hospital residencies or under a preceptorship. Otolaryngologists desiring to do this special type of plastic surgery, or any other for that matter, should have opportunities for proper preparation, and not be forced or allowed to acquire this technic on the trial and error basis. So may this work be extended with greatly increased surgical opportunities of benefiting those in need and they are many.

I thank you all for listening to this somewhat rambling discourse, which will have little interest for our ophthalmologists, although the remarks dealing with plastic surgery may well apply to their specialty also. Thank you all again for allowing me to be your guest and best wishes for the wonderful convention which I know is now about to start.

*Symposium: Irradiation Therapy in Otolaryngology and Ophthalmology*

# GENERAL FACTORS IN IRRADIATION THERAPY

CURTIS F. BURNAM, M.D.
BALTIMORE, MD.
BY INVITATION

FOR all practical purposes a general review of the facts known about x-ray and radium is all that is profitable in this brief review. While it is true that there are other and perhaps very valuable methods of irradiation most of these have no general use at present and are still under investigation. To this category belong neutrons and the radioactive substances produced by neutron bombardment.

The essential difference between irradiation and other physical and chemical instruments rests on two properties; first, the rays penetrate solid substances such as living tissues and can be concentrated at any depth in the living body, and second, cells and tissues differ widely in their tolerance to irradiation. Irradiation may be so weak that no demonstrable changes in cells or tissues can be observed. It may be so strong that all cells and tissues which it strikes are completely destroyed. Therapeutic irradiation lies between these two extremes. Where two or more tissues exist side by side or are actually intermingled and one is pathologic and the others normal it may be possible to entirely destroy the pathologic tissues without serious injury to the normal tissues. It is probable that all cells and tissues are injured by irradiation. However, if the amount of irradiation is not lethal the tissue or cell has the power of recuperating and returning to normal.

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

THE NATURE OF ROENTGEN AND RADIUM RAYS:

X-rays and gamma rays of radium, rays of ordinary visible light, ultraviolet rays and infra-red rays, as well as radio waves, all belong to the same order of things. They all have the same speed, 186,000 miles per second. They have no electrical charge. They can be polarized, reflected and diffracted. All of these phenomena can be explained by the wave theory of light. However, there are other observed characteristics that are much better understood by the assumption that all of these waves are made up of extremely small particles of matter traveling at enormous speed. As a matter of fact, the two theories have to be combined for an adequate explanation of all the observed effects. The particular theory is not new but in its modern form is known as the quanta theory.

The only difference between these different waves is their frequency or wave length. The shortest wave lengths are obtained from the gamma rays of radium and from extremely high voltage x-ray. There is a direct relationship between the wave lengths and the energies of the quanta. The shorter the wave length the greater the energy.

The unit of measurement for wave lengths is one ten-thousandth of a centimeter and it is called the Angström unit. Gamma rays from radium vary from 0.02 to 0.1Å, roentgen rays vary from 0.01 to 5.00Å.

From radium preparations, in addition to gamma rays, there are two

other kinds of rays known as alpha and beta rays or particles. A large part of the energy emitted in the form of irradiation by radium is in alpha rays. These rays have so small a penetrating power that they are completely absorbed in the first few centimeters of air or by a thin sheet of paper. Owing to this failure of penetration alpha rays have only a very limited use in therapy. The alpha rays from various ray-emitting substances have different speeds, varying from 9,000 to 20,000 miles per second. From any one substance, however, all have the same speeds. Alpha rays have been identified as helium nuclei.

Beta rays vary greatly as to speed. Some closely approach that of light. They carry a negative charge. They have considerable power to penetrate matter. A small fraction of an incident beta ray irradiation is present after passing through two centimeters of tissue. Approximately seven per cent of the initial beam is present after penetrating one cm. of tissue. All beta rays are absorbed by .5 mm. of platinum or 2 mm. of brass or monel metal. As will be discussed later, beta rays are very valuable in treating many superficial conditions amenable to ray therapy.

HOW ARE X-RAYS PRODUCED:

From the crude apparatus which Roentgen used to discover x-rays in 1895 there has been almost an unbelievable development of x-ray apparatus and tubes. As fascinating as an account of this development would be it must be omitted here. The x-ray machine takes the ordinary street current, reduces the amperage and raises the voltage to any desired level. It removes the reverse current and as a direct pulsating current is passed through a highly vacuumized tube. In the tube is an anode, usually called a target, and a filament called the cathode. The cathode is heated and when a current is passed, rapidly moving ions fly from the cathode against the target. The impact of these particles produces x-rays. Only about two per cent of the energy of the employed electric current is transformed into x-rays. This is the reason that cooling of x-ray tubes is necessary. Most of the energy is dissipated in heat.

The shortest wave length emitted by an x-ray tube is directly proportional to the voltage used in producing the x-rays. In ordinary deep therapy from 250 kv. to 400 kv. is now employed. For very superficial therapy peak voltages as low as 10 kv. are used. For a number of years 1,000 kv. apparatuses and tubes have been in use. At present 2,000 kv. outfits are available and indeed arrangements for using voltages all the way up to 100,000 kv. can be constructed. Little, however, is known as to the therapeutic value of these very high voltage apparatuses.

MEASUREMENT OF THE QUANTITY AND QUALITY OF X-RAYS:

By far the most accurate method of measuring the quantity of x-rays is by the ionization method. At the present time it is possible to secure measuring instruments which are easily used and extremely accurate. The unit of x-ray quantity which has been universally adopted is represented by the small letter, r, and is called a roentgen. Ordinary air when subjected to irradiation becomes ionized and due to the fact that free ions are liberated from the atoms composing air, the air becomes electrically conductive and the strength of the current is directly proportional to the amount of ionization. Under fixed conditions of temperature and pressure a roentgen is the amount of ionization which is produced in one cubic centimeter of air. With the remarkable stableness so far as output of x-ray is concerned of our apparatuses of today and of the methods employed in mea-

suring x-ray, there is very little or no excuse for x-ray burns and injuries.

The quality of the x-rays is usually expressed in stating the thickness of a metal filter which reduces the quantity of irradiation to one half. This is ordinarily called the half value in copper, aluminum, etc.

FILTERS:

Filter is the term applied to any material placed between the x-ray tube and any object being irradiated. Primary filters are usually metals, while secondary filters are organic substances.

When an x-ray tube is in operation the rays are emitted in a continuous spectrum. The shortest wave length is directly proportional to the peak voltage. Extending downward from this shortest wave length, every wave length to those corresponding to very low voltages can be observed. The quantity in different sections of the spectrum is about the same, except in the region of the characteristic radiations. Here there is a great increase in the intensity of the radiation. This K radiation, so far as position in the spectral band is concerned, varies with the metal employed in the target of the x-ray tube. In the case of tungsten, the K radiation corresponds to voltage of 80 kv.

What a metal filter does is to remove the longer wave lengths completely and to reduce all the wave lengths. It is obvious that this reduces the quantity of rays but increases the penetration due to the fact that the average wave length is shorter. In ordinary deep therapy a filter thick enough to remove all of the K radiation and everything below this level is employed. This first metal filter is called the primary filter. However, a secondary filter should also be used and this should be of some light organic material, like rubber, or bakelite. The reason for this secondary filtration is that ions are liberated from the filter when x-ray is passed through it. These particles have but little power of penetration but can produce irritation of the skin if a human being is being irradiated. The quantity, as well as the energy, of these secondary ions is directly proportional to the density of the filter.

As the density of metals increases so does the capacity of absorption. Roughly the absorption is proportional to the cube of the densities.

FILTERS EMPLOYED IN USING RADIUM RAYS:

We have already commented on the very low power penetration of alpha rays. If a preparation of radium or radon is surrounded by a filter of 2 mm. of copper or brass or the equivalent thickness of any other metal, all the beta rays are absorbed by the filter. When high penetration is desired it is very important to remove by filtration all the beta rays and some of the softer gamma rays. Gamma rays from radium have a capacity of penetrating great thicknesses of very dense metals; for example, there are still present a small percentage of these rays after passing through twelve inches of lead. Beginning with a filter which removes all the beta rays the filter may be made thinner and thinner and with each reduction in thickness the quantity of beta rays is increased. More will be said about this in succeeding paragraphs.

WHAT IS MEANT BY PEAK AND EFFECTUAL VOLTAGE:

Peak voltage has already been defined. Effectual voltage represents an average voltage or the voltage which would give the same penetration if a continuous homogeneous voltage is applied to a tube. The effectual voltage is usually estimated at about one-half of the peak voltage. The effectual voltage corresponding to the wave lengths of radium is approximately 1,000 kv.

HOW ARE RADIUM RAYS PRODUCED:

Radium rays are emitted spontaneously from certain elements. Practically all elements with an atomic weight of 208 or more possess this property. There are three groups or families of such elements. Radium belongs in the uranium family. The other two groups are thorium and its decomposition products and actinium. There are at least forty or more of these elements known.

The parent of radium is uranium. Uranium in small quantities is present in the entire crust of the earth and in sea water. In much larger quantities it is found in a number of minerals.

Radium can be separated from uranium by chemical and physical procedures. It is usually sold as a salt of one of the well known acids. Radium chloride, bromide and carbonate are soluble in water. The sulphate is insoluble.

Starting with uranium, whose atomic weight is 238, and ending with lead, whose atomic weight is 206, there is a continuous series of transformation.

A radioactive element is in equilibrium with its products when the amount of each product is always the same. Radium for instance is in equilibrium with uranium when there is one gram of radium to 2,830,000,000 grams of uranium. There are two kinds of transformations; first, those in which a helium nucleus is removed from the atom. When this occurs an alpha ray is liberated and the atomic weight reduced by four. The other change is where only beta and gamma rays are emitted. These are always together. The atomic weight is not changed but a new substance formed which is termed an isotope. Between uranium and radium there are six of one or the other of these transformations and between radium and lead there are ten such changes. The atomic weight of radium is 226. Consequently in going from uranium to radium there are three alpha ray transformations.

The disintegration of a radioactive element is perfectly uniform. The same number of atoms are affected in a unit of time. What determines why this should be is not clear. None of the ordinary laboratory methods have any capacity to change the rate of disintegration. It occurs in liquid nitrogen or at very high temperatures without any change whatever. Bombardment with neutrons would undoubtedly produce alterations in this rate of disintegration.

The more rapid the disintegration of an element the greater the amount of radiation. The rate of disintegration is usually expressed by stating in time the half value period. For example, the half value period for uranium 2 is 2,000,000 years; for radium 1,690 years, for radon 3.85 days, and for radium A, B and C minutes.

Freshly prepared radium gives off only alpha rays and when it is placed in a glass or metal container all these rays are absorbed by the container itself. The same is true of radon. All of the useful rays from a radium preparation come from radium A, B and C.

When freshly prepared radium is sealed in an air-tight container it reaches equilibrium with its products in thirty days and unless lost by breakage or other accident remains essentially constant for long periods of time.

SEPARATION OF RADON FROM RADIUM:

It is obvious from what goes just before that the rays from radium and radon are identical. Radon may be separated from radium in a number of ways. We have found that a highly efficient and convenient method is to put radium chloride into solution in slightly acidulated distilled water. This solution is placed in a flask which is joined to a vacuum pump or pumps. Every day there is produced a definite quantity of radon, helium, oxygen and hydrogen and water vapor. By means of the

pump or pumps these gases are removed from the flask. First, the water vapor is taken up by potassium hydroxide. Then the hydrogen and oxygen are combined by sparking in a special chamber. In this way several hundred cubic centimeters of gas are reduced to four or five cubic centimeters. This residual gas is passed into an inverted tube and transferred to another apparatus which has been completely evacuated. The gas is drawn into the vacuum and by means of a mercury column brought into a special spiral tube which is surrounded by liquid nitrogen. By this means the radon is frozen to the walls of the tube. The other gases can be removed as they are not frozen. Finally by removing the liquid nitrogen the radon is thawed out and transformed from a solid back into a gas. This small quantity of radon is then pushed into any desired container and sealed off. Usually in our work it is placed in a glass bulb with an internal diameter of 3 mm. Into such a bulb at least two hundred times as much radon can be placed as the radon which is found in equilibrium with a radium salt. We have frequently made bulbs containing 2,000 mc. of radon.

COMPARISON OF RADIUM AND RADON APPARATUSES:

Each has advantages and disadvantages. The advantage of a radium apparatus is that the output of radiation is constant as long as the apparatus is intact. When a treatment is completed all that need be done is to place the radium apparatus back in the place where it is stored until the next time it is used. The disadvantages of radium apparatuses are that there is always the danger of loss by breakage, by sterilization and that insurance rates are relatively high.

The advantages of radon are that there is very little danger of loss, that there is great plasticity as to the strength, shape and size of apparatuses. It is very ideal for all kinds of experimental work. Sterilization is more adequate and much less hazardous and the insurance rates are low.

The disadvantages of radon are that it demands operating a radon plant, developing technicians, daily measurements as to quantity and tables showing the changes in an apparatus from day to day or from hour to hour.

Where work is in a limited field, such as for example treating adenoids in the nasopharynx, a radium applicator would seem to be much simpler and more convenient unless a very large number of patients have to be treated each day. On the other hand, in an institution where all kinds and conditions of problems are met, a radon plant is most convenient.

MEASUREMENT OF RADIUM RAYS:

The measurement of an unknown quantity of radium is by comparing its ionization to that of a known quantity. This can be done in a number of ways but the simplest method is with an ordinary gamma ray electroscope. The first standard was made by Madam Curie in Paris. A perfectly pure radium salt was obtained, accurately measured as to quantity and its gamma ray output determined. From this standard subsidiary standards were made for a number of governments. Our standard in this country is at the Bureau of Standards in Washington. It is highly desirable for those operating radon plants and using the radon apparatuses to have a standard made to correspond with the standard at Washington.

The amount of gamma rays emitted by one milligram of radium and one gram of radium are as one to a thousand. Radon is compared with radium by determining its gamma ray output. When the output is exactly the same as that of one milligram of radium it is called a millicurie and when ex-

actly that of a gram of radium a curie. As already stated, the quality of radiation from radon and radium are identical. Both are the rays given off by radium B and C.

HOW TO RECORD RADIUM OR RADON DOSAGE:

We have already stated that x-ray is measured in r units. Measuring gamma rays in r units has proven quite difficult. However, one milligram of radium at a distance of one centimeter gives approximately 7.75 r per hour. It is not practical, however, to use the gamma rays on the r basis. What has to be annotated is the amount of radium, the filtration, the distance from the surface and the duration in time of the exposure. Where it is desired to give the dosage in a volume in the depth of the body, in addition to the data already stated, the actual size of the volume and its shape, its distance from the surface and the distribution of the sources of radiation must all be stated.

WHAT HAPPENS TO RADIATION WHICH PENETRATES A SOLID SUCH AS THE HUMAN BODY:

Some of the rays pass straight through unchanged. Certainly these have no biological effect. The same may be said of the scattered radiation which emerges from the body in various directions unchanged. Thinking of the rays as quanta some may be completely absorbed. Others lose only a part of their energy and proceed as waves or quanta of longer wave length and less energy. It is practically certain that the rays which are absorbed, either in whole or in part, produce ions and that these ions cause the biological changes in tissues. These particular ions are called photo-ions and their energy depends on the energy of the x-ray or gamma ray quanta. A single photon can dislocate ions from a number of atoms.

There is no direct way of measuring ionization in the living body. What takes place can be approximated by taking measurements in phantoms of the same specific gravity as human tissue. Water and various other substances have been used for this purpose. Careful measurements can be thus made at various distances from the surface of the body and the measurements correlated and annotated in isodose curves. Exactly similar curves may be made for radium in considerable quantity used at from five to 15 centimeters from the surface of the body. This particular type of radium radiation is known as tele-radium. It apparently has little or no superiority to x-ray and has been almost entirely replaced by x-ray treatments.

CHEMICAL AND BIOLOGICAL CHANGES WHICH FOLLOW THE ABSORPTION OF ROENTGEN AND RADIUM RAYS:

Both clinical and experimental observations indicate that irradiation is always an injurious agent and never a direct stimulant. It is true that radiation which is not lethal and where recovery is rapid may be followed by overactivity in growth and other functions. This is not peculiar to irradiation but often follows other types of injury. Two factors, first, the intensity of irradiation and second, the capacity of the body to repair injuries incident to irradiation, determine in large measure what is to be expected from a given exposure to the rays. If the radiation is short of being a lethal amount one can expect ultimately a return to normal and the effects are quite different as the time of exposure varies. A given amount of irradiation given in a few minutes may produce a violent reaction on the skin, whereas if the same amount be given in a period of three weeks no reaction may occur. It is certain that any malignant tumors which happen to be less tolerant to

radiation than the normal tissues which surround them can be much better treated by some method of prolonged irradiation than by a single short massive dose. It seems that the tumor in such cases has less power of repair than the normal tissue. This rule, however, does not hold for all clinical effects and also cannot be relied on in all laboratory experiments. This is evidenced in experiments both on fruit fly and Ascaris eggs.

The changes both gross and microscopic which take place in cells and tissues after irradiation are not specific and are observed following various other injuries. Following irradiation the first morphological changes are observed in the nucleus of the cells. Mitosis and cell division are brought to a halt. The protoplasm of a cell body soon becomes granular. With the ideal irradiation there is not destruction of the cell or perhaps even shortening of the cell life but prevention of reproduction. It is possible that this is the effect observed in treating the nasopharynx with the nasal applicator to remove adenoid tissue and secure better ventilation of the middle ear. Some of the early laboratory work suggested that cells were more sensitive at the time of mitosis than at other times and also that in the case of tissues the sensitivity was inversely proportional to the degree of differentiation. Up to the present time these facts have not aided in clinical treatment. Many tissues which are actively growing and show marked mitosis are very insensitive to irradiation and exactly the reverse is found in other tissues.

Certainly there are great variations in radiosensitivity between different cells and tissues and indeed individual cells of the same type may vary greatly. A very moderate irradiation may destroy a half or more of the cells of a given tissue but to destroy every cell may require four or five times the amount of radiation. At the present time there is no adequate explanation for these variations in sensitivity. They are observed in all experimental material such as fruit fly eggs for example.

The latent period is a term applied to the period of time which elapses after an irradiation before definite morphological changes can be observed. The length of the latent period varies inversely with the intensity of the irradiation. If we take the human skin as an example after a mild radiation a slight erythema may occur as late as three weeks whereas with a very intense radiation it may develop in a few hours. In highly sensitive cells and tissues definite morphologic changes may be found by microscopic examination after only a few hours even when the radiation has been very mild. These first changes involve both the nucleus and cell body. Physiologic changes are more difficult to observe and evaluate. However, definite changes in metabolism, increased permeability of cell membranes leading to edema and increased acidity are all common occurrences.

EFFECTS OF DIFFERENT WAVE LENGTHS:

Reasoning on the analogy of the effects of ordinary light, television, etc. it was believed that very different effects might be observed depending on the wave lengths employed. The preponderant weight of opinion today is that there is no qualitative difference in effect between wave lengths and both the morphologic and physiologic changes will be the same if the same amount of radiation is absorbed. This of course does not mean that the character of the radiation inside the body as produced by different quality of radiation is the same. However, there seems to be very little, if any, advantage in clinical results by increasing the voltage from 300 to 400 kv. to 1,000,000 kv.

PROTECTION OF THE PERSONNEL USING RADIUM AND X-RAY FOR TREATING PATIENTS:

It is easily possible where x-ray is used with suitable thicknesses of lead to cut off all radiation from the persons who operate the x-ray machines. This should always be arranged for. The construction details and the protection necessary can readily be found in any modern work dealing with x-ray therapy. Before considering protection from radium or radon preparations it is well to recall that the time of exposure, the strength of the applicator, the distance of the applicator from the operator are all facts which it is essential to know. To these factors must be added the protection afforded by lead screens and covers.

The best way of protecting the hands and fingers against beta rays is afforded by cutting the time of exposure to a minimum and using forceps to secure as much distance as possible. The radium applicator should be kept in a hollow lead cylinder whose wall thickness should be from 1½ to 3″ across. This cylinder should be kept on a movable hand truck. The truck can be rolled up to the treatment table.

Gamma rays are more likely to cause internal injuries. The earliest changes observed are in the blood count and in the effects on the generative organs. In addition to the precautions already listed, the length of service of nurses and technicians who make up the applicators should be limited as to time.

It may be of interest to calculate the actual exposure in roentgens. A thousand milligrams at one centimeter in one hour give 7,750 r. Like all other forms of light the intensity varies inversely with the square of the distance from the source of radiation. For example, if a gram of radiation is 100 cm. from the point of measurement then the radiation produced by the one gram in one hour would be only .78 r, at 200 cm. only .19 r per hour. It is obvious from these calculations that even when dealing with large quantities of radium that over-radiation is almost out of the question provided the protective measures outlined be followed. Five cm. of lead reduces the radiation to 7.5 per cent which means that if two applicators are used, each containing 50 mg., the actual radiation is the same as that of 7.5 mg. when no lead is used. Behind the two inch lead screen at two feet only .1 r is given.

Just how much irradiation the human body will tolerate is an open question, but taking .03 r per hour which is about as much as any one operator would get in a single day using it every day for a year the dosage would only be about 90 r.

At the clinic where I am associated there has never been a serious general injury. In the early days moderate reduction in the white blood count did occur but on removal from the work promptly returned to normal. There have been no cases of sterility, no cases of genetic change in children.

It might be of interest to know that we have been using from five to six grams of radium for approximately thirty years and in the first ten years had nothing like the protection that we have today.

USE OF X-RAY AND RADIUM IN THE CLINIC:

My part of this program, as I understand it, is not to consider details of treatment and clinical results obtained but to outline some of the guiding principles which should be followed in employing irradiation as a therapeutic procedure.

While the results are the same if an equal absorption is obtained with radium and x-ray rays, in actual clinical practice the application may be much simpler with one or the other or even impossible with one and readily ac-

complished with the other. We have already covered the essential reason for these differences. Spherical dispersion which accounts for the inverse square law is at the basis of many of these differences in usage. The aim of treatment always is to cure and not injure the patient. In ophthalmology it is particularly vital not to injure any of the essential structures of the eye upon which vision depends. In the nose and throat work injury of bone, cartilage and nerve tissues must be considered and the radiation planned so as to reduce to a minimum the possibilities of such injuries. In otolaryngology, as well as ophthalmology, the beta radium applicators are particularly valuable in treating superficial lesions. Where deep-seated and extensive lesions are concerned such as may occur deep in the orbit, in the sinuses, in the tonsils, in the larynx, in the pharynx and nasopharynx, x-ray is the best agent. By careful location of the pathologic lesion, its depth from the surface, its shape and size it is possible by using multiple portals of entry through the skin and cross-firing to give quite intensive dosage where this is needed. I would like to point out here that in the treatment of malignant neoplasms x-ray should be considered as a surgical instrument. This is particularly true of the more ray-resistant tumors. There are only a few of these growths that can be eradicated by a general spraying of the entire field. These are of course the supersensitive tumors such as lymphosarcoma and lymphoblastoma. This viewing of the necessity of using portals just large enough to take in the entire lesion being treated cannot be emphasized enough. To actually carry out exact cross-firing with small portals demands time and patience. It can't be turned over to a technician as is so often the case. One frequently sees portals 10 cm. in diameter employed in treating a growth which is less than 1 cm. in diameter. This inevitably means over-radiation of normal tissues and injuries which may not show up for years.

Inflammatory conditions when treated by x-ray never require heavy dosage. In general the benign tumors can be better treated by some surgical procedure. Most of them are quite ray-resistant. Benign lymphomas and angiomas are an exception to this rule.

Radium by direct application or implantation in suitable locations is much more desirable than x-raying. The inverse square law makes it possible to give direct radiation of any intensity decided on to the pathologic tissues and at the same time give very little radiation to surrounding normal elements. Implants are very small tubelets of radon which can be left in permanently or easily removed as seems best. The filtration, usually .3 mm. of platinum, removes the beta rays almost entirely. Where there is an accessible solid mass the ideal method of treatment is to divide it into spheres and attempt to implant in each sphere a radon point strong enough to give adequate treatment to tissues on the surface of such a sphere. Implants can readily be used in the treatment of lip, tongue, floor of the mouth, cheek and tonsil conditions, as well as in the glands of the neck which have been secondarily involved. In general implants should only be used for treating malignant new growths. Radon or radium can be used in conjunction with a surgical approach for treating malignant conditions of the head sinuses and of the orbit after removal of the eye.

X-ray and radium can often to great advantage be combined to secure adequate radiation therapy. It should also be constantly kept in mind that certain conditions which may not be suitable for operative treatment become so after the effects of radiation have fully developed. While not a cure-all for any type of lesion, irradiation can bring relief to

many conditions and cure where no other agent can accomplish such a result. The advantages and disadvantages of radiation as compared to surgery where both may be effectual must be carefully weighed and determined as to the most suitable by experience. Certainly in the malignant tumor class all of those cases which obviously can't be cured by surgery should be given the advantage of ray treatment and some of them will be cured.

BRIEF DESCIPTION OF NASAL APPLICATOR AND OF THE BETA RAY APPLICATOR:

Dr. Crowe and I originally used a nasal applicator with a wall thickness of 1 mm. Our feeling was that a better distribution of the radiation in the nasopharynx could be secured by such an applicator which emitted only gamma and a few very hard beta rays. The disadvantage of this applicator was that it required for short treatments a great deal of radon and the treatment could not be accomplished at all where radium was used except by long exposures. By reducing the wall thickness from 1 mm. of brass to .3 mm. the time of the application has been reduced to about one sixth of that required when 1 mm. of brass is used. Monel metal which has the same absorption power as brass but has other qualities that are better than brass was selected as the screen. This made it possible to make a radium containing applicator and yet not unduly prolong the time of treatment. In addition to this there is the economic factor. Radium is an expensive material and it means a good deal to cut the essential amount needed to one sixth of what would be required with the thicker screens.

The so-called beta ray applicator devised for superficial work in ophthalmology will be described by Dr. Iliff and whereas approximately a gram twenty-three seconds is required for a unit dosage with the nasal applicator with this applicator an approximately similar dosage can be secured in a gram two seconds if there is no screening except the glass containing the radon. It is possible, however, to use screens of various thicknesses of aluminum if it is desired to remove the softer beta rays. The best method of determining the safe method of dosage where any change is made in the applicator is to determine the time necessary to produce a mild erythema on the skin. Physical methods of measurement are not very satisfactory.

REMARKS ON ANNOTATIONS:

We have already remarked on how radium and x-ray treatments should be recorded. Usually radium applications are recorded as so many milligram hours, milligram minutes and milligram seconds. We have found it more convenient to express the dosage in gram hours, minutes and seconds. For instance where gram seconds are considered the amount of radium or radon used is divided into 1,000. Let us say the amount of radium is 50 mg. Then the quotient will be 20 and 20 seconds exposure with the applicator used will give 1 gram second. This 1 gram second can be again multiplied to give any desired number of seconds.

GENERAL CONCLUSIONS:

I trust I have made it clear that irradiation can be used to advantage in a great many clinical conditions both in otolaryngology and ophthalmology, that it is simply a method of treatment just as is surgical procedure and the use of bactericidal and bacteriostatic drugs. The specialists in ophthalmology and otolaryngology should master the principles of ray therapy and either carry out themselves the treatments or have them carried out in the way that is likely to produce the best results. Initially practically all the patients suffering

from new growths, benign and malignant, and from various infections come to the specialist. He is in position to study and assess the value of therapeutic procedures and as experience is accumulated to change and adjust methods to secure better and new results. So far as the beta ray applicators are concerned their use is so simple that any competent man can take them up with very short study and instruction and apply them clinically. With x-ray and with implanting a joint and unison work between a competent radiologist and a specialist is perhaps the best method of procedure.

Finally for those who decide to take up this form of therapy it is urged that all the suggestions made as to protection of patients and of personnel be most carefully and scrupulously carried out. If this is done there should be no method of treatment safer or simpler in its employment.

*Symposium: Irradiation Therapy in Otolaryngology and Ophthalmology*

# IRRADIATION OF THE NASOPHARYNX

SAMUEL J. CROWE, M.D.
BALTIMORE, MD.

UNTIL recently text books on otolaryngology devoted very little space to the underlying causes and prevention of upper air passage infection, but chiefly were concerned with the treatment of infection. Particularly was this true in the case of recurring attacks of otitis media, sinusitis and allergic bronchitis in children. However, our increasing success in reducing the frequency and severity of upper respiratory infections and preventing chronic sinusitis and changes in the bronchial mucosa that are irreversible and lead to chronic asthma, has placed the emphasis upon removal of the cause rather than treatment of the result. The ever increasing routine examination of school children proves more than any other hopeful sign, that at last we recognize a great opportunity for preventing conditions which may lead to later trouble. One result of years of intensive study is the conviction that many of the hearing disorders of adult life begin in childhood, and if we are to learn more about the causes, treatment and prevention of deafness we must concentrate our studies on children. Almost every type of middle and inner ear deafness seen in adults is found in children, with this important difference: in children the impairment is just beginning and opportunities are offered for study of the life history of the disease and for developing some kind of effective and even preventive therapy. In adults the malady has been in the making for many years, and the hearing apparatus usually is irreparably damaged. There is much less chance of curing deafness in the adult, and so we must seek in children for a clear understanding of the causes and best methods of prevention.

This principle of search for underlying causes led to the founding, in 1924, of the Otological Research Laboratory of the Johns Hopkins University, with the aid of the Rockefeller Foundation and interested friends. For twenty years we have used in this laboratory the method that has been employed so successfully in studying diseases of the heart, lungs, kidneys and other parts of the body, i.e. correlation of history, clinical examination and functional tests with the histologic appearance of the organ after death. By making for research purposes, accurate audiometric and tuning fork tests and other examinations of the ears and upper air passages on patients in the neurosurgical, medical and tuberculous wards, whose general condition made it likely they would come to autopsy, we have been able to collect and section serially more than 2000 temporal bones. In a good proportion of these the hearing and histologic findings were normal, which made them most useful as controls.

The first publication of this correlation study was made after ten years spent in collecting material and preparing sections. The group selected for study had impaired hearing for high tones, but good hearing for low tones and for most of the frequencies in the

Presented as the Wherry Memorial Lecture at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

speech range. At that time this type of hearing impairment was universally thought to be due to an inner ear or nerve lesion, and in the majority of our cases this was found to be true. In a few, however, the nerve and organ of Corti were normal histologically and the only changes seen were in the middle ear. This finding made it very clear that impaired hearing for high tones is not always due to a nerve lesion and led us to re-examine patients, particularly children thought to have an early stage of nerve deafness. When examined from this new point of view, we began to evaluate the history, changes found in the tympanic membrane, middle ear mucosa, nasal passages and nasopharynx. Many of these children had opacity and retraction of the tympanic membrane, hyperemia of middle ear mucosa, and a dark purple or bluish discoloration of the drum membrane, which could only be due to a small hemorrhage in the mucosa, or to red blood cells and serum lying free in the middle ear cavity. Previously, we had thought these findings, in the absence of pain or symptoms of infection, could not cause deafness in which high tones were more impaired than low tones. It soon became evident, however, that we were dealing with the condition we now call aerotitis, and that the changes we saw could be due only to partial or intermittent eustachian tube obstruction. It was also evident that we must find some method of seeing the nasopharynx and orifice of the eustachian tubes more clearly than was possible with the laryngeal mirror. Here I wish to acknowledge my gratitude to Dr. Edgar M. Holmes for devising the nasopharyngoscope, an instrument which to me just at this time seemed like an answer to prayer. Examinations with this instrument showed an overgrowth of lymphoid tissue in and around the pharyngeal orifice of the eustachian tubes in many of these children, and convinced us that some measure, other than surgery was necessary in order to remove this tissue without further damaging the tubes.

It had been known for thirty years or more that lymphocytes are extremely sensitive to irradiation, so we turned to Dr. Curtis F. Burnam for advice. He suggested treatment with a radon applicator, constructed the instrument, advised us about placing the applicator, and the lenth of time it could be left in the nasopharynx with safety. Under this treatment the lymphoid tissue around the tubal orifices gradually disappeared, marked improvement or complete return of hearing followed, and in many the bluish discoloration of the tympanic membrane also disappeared. No drugs, inflations or other forms of local treatment were used. The marked improvement in these cases showed conclusively that certain types of middle ear deafness could be cured by this simple, safe, painless and inexpensive method, and also that middle and inner ear deafness cannot always be differentiated with audiometer and tuning fork tests alone.

This was the first of a long series of clinical observations and treatments that has led us to the conclusion that oft-repeated attacks of eustachian tube obstruction are a common cause of impaired hearing in children. Recurrence of adenoids after operative removal is so frequent that it must be regarded as normal. Repeated examinations for research purposes of 1365 unselected public school children showed that adenoids recur in more than 75 per cent of those whose tonsils and adenoids have been removed before puberty, and that nearly 40 per cent of these had either impaired hearing for high tones alone, or for all tones in the speech range. In addition to loss of hearing, the regrowth of lymphoid tissue after operation predisposes to recurrent upper respiratory infections, attacks of suppurative otitis

media, sinusitis and bronchial infections. For several years we have supplemented every tonsillectomy with irradiation of the nasopharynx. This does not interfere with healing, and is desirable, since it aids in the removal of lymphoid tissue in all parts of the nasopharynx and thus helps to reduce the incidence of colds and their complications. The first treatment is given about two weeks after the operation, and two additional treatments are given at intervals of two weeks. With the 50 mg. monel metal applicator the dosage for all treatments is 8½ minutes on each side. Recurrence of adenoids is not always due to incomplete operation, but chiefly to the fact that lymphoid tissue is an integral part of the mucous membrane of the posterior and lateral walls of the pharynx and nasopharynx, and that in some children it hypertrophies as a result of infection, allergy and other causes we do not understand. In the majority of children however, it regresses spontaneously soon after puberty, without permanent damage. Our task is to recognize early the children who are in need of treatment to help tide them over the childhood years, and thus prevent permanent damage that may interfere with their physical development, education and even their career.

Operative removal of a very large adenoids, followed by a series of radium treatments, is always more satisfactory than irradiation alone, due to the mechanical difficulty of applying adequate irradiation to a large mass in this area with the nasopharyngeal applicator. The radium-containing chamber of the applicator is 2 cm. in length and most of the beta and gamma rays are emitted from its sides and not from the end, which is only 2.3 mm. in diameter. When inserted through the nose, the end of the applicator comes in contact with the large mass of adenoids at the choanae. This prevents its further passage into the nasopharynx and leaves most of the radium-containing chamber in the nose. In this position the majority of the rays go into the posterior part of the septum and turbinates; very few of them reach the adenoids. If patients with large adenoids are treated in this way the results will be disappointing. It is decidedly dangerous to plunge the applicator into the mass of adenoids, to bend the applicator, or to construct it with a hinge so the angle of the radium-containing chamber may be changed. This introduces a hazard that the radium-containing part may some day break away and be swallowed or aspirated. If adenoids are relatively small however, and it is possible to introduce the applicator far enough into the nasopharynx to permit sufficient irradiation of the tubal orifices, the fossa of Rosenmüller and the lateral walls, then operation is not necessary.

Most infections of the upper air passages begin in lymphoid tissue. It is for this reason that operative removal of tonsils and adenoids is so beneficial in many cases. Lymphoid tissue however, is not limited in its distribution to the tonsils, the middle of the nasopharynx and base of the tongue, but is also an integral part of the mucous membrane of the lateral and posterior walls of the pharynx and nasopharynx. In many children lymphoid tissue in these locations hypertrophies after operative removal of tonsils and adenoids and gives rise to the condition known as "granular pharyngitis". Often these compensatory hyperplastic nodules, although small in size are so located that they interfere with the function of the eustachian tubes, and cause acute aerotitis media in military aviation, in "escape hatch training" in the submarine service and in other occupations in which men are subjected to extreme pressure changes. The same mechanical abnormality causes in many children a mild and often symptomless aerotitis, which if oft-repeated leads to hyperemia,

edema and eventually to scar tissue in the mucous membrane of the middle ear. This impairs the hearing and in the presence of an acute nasopharyngitis, causes repeated attacks of suppurative otitis media.

We wish to make it very clear that we do not advocate indiscriminate use of radium as a treatment for deafness. The hearing may or may not improve, depending on the cause and duration of the impairment. Radiation is of great value in re-establishing the lumen and function of a eustachian tube partially occluded with hyperplastic lymphoid tissue. In other words it is used to correct a condition that predisposes to impaired hearing and to attacks of otitis media. The relationship between chronic or oft-repeated episodes of partial eustachian tube obstruction and impaired hearing is so definite, that irradiation of the pharyngeal orifice of the tubes is justifiable as a prophylactic measure in all patients in whom hyperplastic lymphoid tissue is seen in or near the orifice of the tube. Even if the hearing is normal, irradiation is indicated as a preventive measure when nasopharyngoscopic examination reveals the above conditions. It is also called for in all patients with this condition who have impaired hearing due to any cause whatever, such as developmental abnormality of the cochlea or auditory nerve, chronic suppuration of the middle ear, or otosclerosis, in order to prevent superimposed or additional impairment from an entirely different cause.

Nodules of hyperplastic and infected lymphoid tissue in the nose, near the posterior end of the middle turbinate, may cause symptoms resembling vasomotor rhinitis. We believe lymphoid tissue in this location is a common cause of ethmoiditis and sphenoiditis in children. The acute attacks usually subside with local treatment, but unless the underlying cause is recognized and eliminated the symptoms recur again and again, and result in chronic sinus infection. Lymphoid nodules in the nose and in and around the tubal orifices are often so small that they can be seen only with a nasopharyngoscope. They should always be looked for, and if present, removed with irradiation rather than operation or a cautery. Irradiation is the ideal treatment for chronically infected or hyperplastic lymphoid tissue in locations inaccessible to operative removal. Edema is a constant change following radiation. Therefore, it is advisable to omit treatment when the patient has an acute upper respiratory infection, and in subacute or chronic infections always to give the treatments at three or four week intervals. Otherwise irradiation may precipitate a complicating otitis media or sinusitis. The radium applicator should not be used in the nose itself for any condition except nodules of lymphoid tissue. Polyps are extremely resistant, and the applicator is not suitable for the treatment of malignant growths. It was designed for one purpose alone—the removal of lymphoid tissue.

It has long been known that infection plays an important role in the development of respiratory allergy, and that children of allergic parents or those with food sensitization or eczema, who are subject to upper respiratory infections should be considered as allergic suspects, and prompt corrective measures instituted. Especially is this true of children subject to frequent winter colds, in whom cough and sometimes wheezing is a prominent if transient symptom. In the very young and in early adult life, lymphoid tissue in the pharynx and nasopharynx is the primary focus of these infections. Later in life when the nose has become filled with polyps and the sinuses and bronchial mucosa are chronically infected, the results of surgery, chemotherapy, and the administration of antibiotics are much less satisfactory. In children com-

plete surgical removal of tonsils and adenoids, together with protection against airborne or environmental pollens, dusts and danders often effect a cure. In others the cough and wheezing recur a few months or a year later.

When asthmatic symptoms of a child improve following operation and recur at some later date, the otolaryngologist rarely has any difficulty in finding more infected tissue to remove. Through the accident of a shortage of beds, particularly free beds, we were forced to postpone operations on these and patients with other maladies. This led to the establishment of a radium clinic in the otolaryngological dispensary, so that at least their adenoids could be treated. To prevent over-exposure of the doctors, the radium treatments were limited to the nasopharynx. For several years an average of 150 patients a month were treated. Among them were many with asthmatic bronchitis waiting for operation, and others in whom the asthmatic symptoms had recurred following removal of tonsils and adenoids. Sometimes following the second, but usually about a month after the third irradiation, the parents would report marked improvement or complete disappearance of asthmatic symptoms. These reports were so positive and frequent, that a co-ordinated study was begun in 1942 by the pediatric, allergy and nose and throat departments. A full and detailed report will soon be published, but I can say now it is an established fact that the results following irradiation of the nasopharynx alone are as good and often better than those following operative removal of tonsils and adenoids. This has given us proof that the greater number of upper respiratory infections have as their focal point the nasopharynx. All children having these treatments in the nasopharynx should be examined at least once a year, and if lymphoid tissue recurs, additional treatments should be given. Irradiation with the nasopharyngeal radon or radium applicator is a method of treatment that can be used on patients of all ages to remove lymphoid tissue in locations inaccessible to ordinary surgical measures. It is effective, safe, painless, inexpensive and has proved particularly valuable for prevention of certain ear, sinus and bronchial condition in children.

The technic of examination with the nasopharyngoscope and treatment with the radium applicator is simple, even in the case of children of school age. After a few preliminary questions about the symptoms, we select the side of the nose that is least obstructed, and by spraying lightly with 5 per cent butyn begin the anesthetization. We then take a careful history, test the hearing with voice and tuning forks, and make a routine examination of the tympanic membranes and upper air passages. An audiometric test if indicated is made later in a sound proof room. At intervals the examination is interrupted and the lower half of the septum, the inferior turbinate and the posterior wall of the nasopharynx are further anesthetized with a small cotton-tipped applicator, moistened with 20 per cent cocaine. By thus prolonging the anesthetization period the minimum amount of cocaine can be used, and at the conclusion of the routine examination we can at once proceed to pass the nasopharyngoscope. To make a satisfactory examination with the nasopharyngoscope, all thick mucus on the floor of the nose and in the nasopharynx must be removed with a suction tip. In young or very nervous children, it is impossible to examine the nasopharynx with a nasopharyngoscope under local anesthesia. Even if the head is held firmly, the excess mucus that results from crying obscures the view. In these patients we must decide whether the nasopharyngoscopic examination is important enough to justify general anesthesia, or whether to treat them on the

basis of history and such examinations we can make, without upsetting the child psychologically and thus rendering all future examinations and treatments even more difficult. If irradiation of the nasopharynx is indicated by the history, we treat these children without examination, since we know from twenty year's experience that if properly used, the radium applicator can do no harm.

I have found it easier to keep the radium-containing chamber in contact with the tissue to be removed, if the patient lies on a couch. Especially is this true when the pharyngeal orifice of the eustachian tube is being treated. When irradiating the right eustachian orifice the head is turned to the left, and vice versa. For protection of the doctor and office personnel the applicator should be left in the lead cylinder until everything is in readiness for its insertion into the nasopharynx. If many treatments are being given each day, we must realize that the applicator is out of the lead much of the time. This exposes our own office force, and even those in other offices separated by a partition, but who are within a few feet of the applicator. To guard against this constant danger, distance is the best protection, since the intensity of the radiation varies inversely with the square of the distance. At twenty feet the radiation is 1/400 of that at one foot. It is of the utmost importance for the radium to be stored and the treatments to be given at least twenty feet from those who are daily exposed to its effects. The 2 inch lead cylinder that comes with the applicator is intended to make transportation practicable, but for storage in an office the lead should be at least 6 inches in diameter. A cylinder of this size and weight should be mounted on a table that can be wheeled from room to room.

The tissue reactions and the therapeutic value of a radium treatment depend entirely on the absorption of beta and gamma rays. All beta rays emitted by the 50 mg. monel metal applicator are absorbed by tissues in the nasopharynx, and approximately 75 per cent are absorbed in the first 3 mm. This is another reason for removing greatly enlarged adenoids at operation and irradiating the relatively small amount that is left, rather than treating with radium alone. Every variety of cell in the body has a specific range of sensitiveness to beta and gamma rays. The sensitiveness of cells depends on their life cycle and mitotic activity. In adenoid tissue the germinal center cells are much more sensitive than the mature lymphocytes, and their sensitiveness to irradiation varies with the degree of mitotic activity at the time of the treatment. For this reason the response of the adenoids to irradiation is often more satisfactory in children than in adults. As we all know a heavy dosage is used to irradiate a malignant growth, since the object is to injure all cells so seriously that they will never recover. The dosage used in treating hypertrophied lymphoid tissue however, is just enough to stop mitosis in the germinal centers. As the mature lymphocytes complete their life cycle and are discarded, no new ones are being formed to replace them, and the entire mass gradually decreases in size leaving only the fibrous framework. With the dosage we use, the degree of tissue injury is small and some of the germinal center cells may recover. If they do, lymphoid tissue will eventually regenerate, in which case further irradiation may be necessary.

In treating the nasopharynx it is always essential to use a dosage that we know will not injure adjacent normal structures. We advise three treatments of 8½ minutes on each side, with the 50 mg. monel metal radium applicator in direct contact with the tissue to be removed; then a wait of thirty to sixty days after the third treatment before considering further irradiation. The ob-

ject of the treatments is to remove the particular lymphoid tissue which is causing the patient's malady, not to remove all lymphoid tissue in the nasopharynx. If symptoms do not return, it is unnecessary to give further radium treatments, but if needed they can be given with perfect safety. During the war approximately 25,000 treatments were given with the monel metal radium applicator by medical officers of the Army Air Forces and at the New London Submarine Base, in order to prevent recurrent aerotitis and thus conserve man power. The average result was 90 per cent effective. Not a single instance of radium burn or radium poisoning, with a drop in the white count, has been reported. The author has given treatments with the nasopharyngeal applicator almost daily for twenty years and has suffered no ill effects. Years of experience, together with histologic study of adenoids irradiated and then removed surgically at varying intervals, have shown that 1 gram 25.5 seconds, or 8.5 minutes on each side of the nasopharynx at intervals of two weeks, is the safe and most effective dosage for the 50 mg. monel metal radium applicator. The only requirements are an accurate examination with the nasopharyngoscope to establish the indication for radium treatment, to place the applicator so that the side, not the end of the radium-containing chamber is in contact with the tissue to be treated, and finally that the greatest care be taken to time each treatment accurately.

In closing I wish again to emphasize the great value of irradiation in otolaryngology, if intelligently and carefully used. May I solemnly urge those of you who have an applicator, or intend to use one in your practice, to surround yourselves and your office personnel with protection essential to its safe employment.

*Symposium: Irradiation Therapy in Otolaryngology and Ophthalmology*

# BETA IRRADIATION IN OPHTHALMOLOGY

CHARLES E. ILIFF, M.D.
BALTIMORE, MD.

BY INVITATION

THE majority of reports on radiation therapy of the eyes are concerned with gamma rays. The purpose of this paper, however, is to discuss the use of the less well-known beta rays in the treatment of nonmalignant lesions of the lids and anterior ocular segments.

Beta rays possess the same qualities as other rays in the power of selective tissue destruction but in contrast to gamma rays they penetrate about three millimeters of tissue only. This makes them ideally suited to treatment of the anterior ocular segment. In no single case where this therapy has been used has any damage to the lens been observed. This has been true clinically, as well as in animal experiments where in one instance a rabbit's cornea was given 300 gram seconds of beta rays without a resultant cataract.

The ease of application is a great advantage of the beta rays. In the removal of certain small benign tumors of the lids or conjunctiva, the facts that pain is absent, hospitalization is not required, and the cosmetic result is excellent, all make beta therapy peculiarly acceptable both to children and adults, especially to those who object to surgery. In the treatment of anterior ocular tuberculosis a certain analgesic effect has been noted after treatment and the course of the disease seems to be shortened. Superficial vessels of the cornea can be occluded, and new vascularization inhibited, thus, beta irradiation is ideal as postoperative therapy in lamellar keratectomes, in preventing postoperative granulation and inhibiting the overgrowth of connective tissue.

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.
Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University, Baltimore, Md.

## TREATMENT

Beta irradiation is applied with a special radon applicator designed by Curtis F. Burnam. This consists of a five millimeter soda glass bulb containing from two hundred (200 mc.) to five hundred millicuries (500 mc.) of radon, enclosed in a brass cylinder with walls two millimeters thick. From a four millimeter window at one end of the applicator, beta and gamma rays pass unfiltered; the alpha rays being stopped by the soda glass container.

The dosage of beta irradiation is calculated in gram seconds (the product of the weight of radium in grams or its equivalent in curies of radon, and the exposure time in seconds). Since the skin erythema dose of unfiltered beta rays is 18 gram seconds while that of gamma rays is 8 gram minutes, it is possible to give adequate beta therapy with such short exposures that the effect of the gamma rays is negligible.

The applicator is placed as close to the lesion as possible (approximately 1 - 3 mm.) and moved slowly back and forth across an estimated square centimeter of field, giving a spray action to the rays. Application distance is important as the strength of the delivered dose varies inversely with the square of the distance from the source. Care should be taken that the overlap-

ping effect does not exceed the calculated optimum dose. In the treatment of small tumors, pterygiums, granulation tissue, or blood vessels invading the cornea, the applicator is held in contact with the lesion.

Beta irradiation has a cumulative effect if repeated doses are given within a period of two weeks. The total dose to any one area should not exceed 18 gram seconds during this period. With adults not more than 12 gram seconds is given to an area at one treatment; with children the dose is started at 3 - 4 gram seconds. Before administrating a maximum dose, the individual sensitivity to beta rays should always be tested. The course of treatment consists of 18 gram seconds in a two week period, followed by a two to three week rest period. At the end of this time another course of 18 gram seconds may be given. When the case is not acute, the patient may be placed on a monthly maintenance dose of 8 - 10 gram seconds.

When contact therapy is used or when photophobia, pain and lacrimation make adequate exposure impossible, local anesthesia with pontocaine is helpful. The treatments themselves cause no pain, but an exacerbation of the existing symptoms frequently occurs in three to four hours and may last twenty-four hours. In these cases a mild sedative is very helpful.

In some instances a punctate stippling of the cornea (clearly visible when stained by fluorescein) has appeared within twenty-four hours and lasted four to five days. Loss of cilia is rare but does occur in heavily treated areas of the lids.

## VERNAL CONJUNCTIVITIS

Throughout the literature on vernal conjunctivitis there are many reports on the excellent results with radium therapy. However, due to sparsity of data and to varied technic, evaluation of this material is difficult. An adequate review of the literature to 1929 has been given by Kumer and Sallman, and since that time outstanding reports have been made by Quick, Stallard, Robinson, Burnam, Bowing and Fricke and Pendergrass and Andrews.

*Material*

Sixty patients with vernal conjunctivitis were treated with beta rays. All were followed for at least one full season of nine months and some, for as long as six years. The majority of white patients developed symptoms before the age of thirty; colored patients before the age of ten.

The total dosage producing a cure ranged from 7 gram seconds in an early lesion to 258 gram seconds spread over three and one-half years in a case of longstanding palpebral lesion. Most patients (70 per cent) showed marked symptomatic improvement with less than 40 gram seconds of spray therapy.

An example of the course of the disease and the rate of clearing under therapy is W. D., a 5 year old colored boy, first seen in July, 1941. Typical symptoms of lacrimation, photophobia, itching and discharge had been present for the previous season, and at the time of examination the lids had a velvety appearance but no follicles. The limbal vegetations were present around the upper half of each cornea from three to nine o'clock.

Two treatments totaling 10 gram seconds were given to the limbus, and four treatments to the upper lids totaling 20 gram seconds. After these, he became symptom-free, and the vegetations flattened within three weeks. The following year there was a slight recurrence, and 9.4 gram seconds were given to the limbus and 7 gram seconds to the lids. The child had no further symptoms until the summer of 1943, when there was a mild recurrence with lacrimation and itching. At this time the limbus of the left eye was treated with 9 gram sec-

**TABLE I.**

**Results of Beta Irradiation Therapy in Vernal Conjunctivitis.**

| | Cured | | Improved | | Unimproved | |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % |
| Palpebral 41 | 8 | 20% | 31 | 75% | 2 | 5% |
| Limbal 12 | 5 | 42% | 6 | 50% | 1 | 8% |
| Combined 7 | 5 | 71% | 1 | 14.5% | 1 | 14.5% |
| 60 | 18 | 30% | 38 | 63% | 4 | 7% |

onds, and the right eye was used as a control. The left eye promptly cleared while the right eye continued to give symptoms until it also received proper therapy. For checking purposes the child was seen in the summer of 1944 and again in the spring of 1945; no recurrence of symptoms or signs was visible on either of these occasions.

The most severe case of limbal vernal conjunctivitis seen here was that of C.O., a 7 year old colored boy. The disease had been present four years and vision was reduced to 10/200 in each eye. For three seasons the boy received therapy totaling 80 gram seconds. Improvement was slow but by the end of the first season there was some definite gain, and by the end of the third, his vision had improved to 20/40. He is still under treatment.

An example of a palpebral lesion in a 6 year old colored boy is seen in M.W. The symptoms had been present two years. He was given nine treatments at two to three week intervals, totaling 88 gram seconds. The follicles disappeared and he became almost symptom-free. This child, if available will be given two prophylactic treatments next season.

A control was run on a 9 year old colored boy, L. J., who had symptoms for two years. The left upper lid, which had the more severe lesion, was given 25 gram seconds while the right upper lid was not treated. Zinc and adrenalin drops were given for both eyes. For the next two years the right untreated eye continued to give symptoms and follicles and discharge persisted; the treated eye was asymptomatic and had no follicles. After two years the right eye was treated and it also became asymptomatic. The third season follow-up, the right upper lid was white and conjunctiva smooth, the left upper lid which had been treated three years before now looked slightly injected so it was given 10 gram seconds prophylactic treatment. In this patient, 25 gram seconds gave freedom from symptoms for two years during which time the control eye was extremely annoying.

*Results*

The results of beta irradiation therapy on 60 patients with vernal conjunctivitis are presented. Forty-one patients had palpebral lesions, 12 limbal, and 7 a combination of palpebral and limbal. Patients were considered cured if no signs or symptoms remained after treatment and if there was no recurrence the next year. Improved cases were those where both subjective and objective improvement occurred, but a few granulations or slight discharge persisted. Patients apparently healed after treatment who did not return for a check the sec-

ond season, were also placed in the improved group. Patients were considered unimproved, when severe symptoms continued and the follicles persisted after adequate therapy.

Thirty per cent of the 60 patients were healed, 63 per cent were improved, and 7 per cent were unimproved by beta ray therapy.

Limbal and combined lesions usually were brought to the clinic at an earlier stage in the disease than the palpebral variety and thus responded more readily to treatment. In only one case was the limbal lesion of long standing (C.O.) and it showed the same resistance to therapy as the long standing palpebral lesions. The slightly better results in the combined group are not significant due to the small number of cases.

ANTERIOR OCULAR TUBERCULOSIS

Encouraging results in the treatment of anterior ocular tuberculosis with beta irradiation were reported by Woods in 1939. Fifty-two patients representing 72 eyes have been treated.

The duration of the disease before treatment had ranged from one week to as long as thirty years. All cases were characterized by remissions and exacerbations. A few of these patients had been followed in the Wilmer Dispensary for ten to fifteen years and from these an attempt was made to evaluate the length and severity of exacerbations with and without beta therapy. In any such problem, controls were extremely difficult. In the overall picture, however, it seemed clear that treatment shortened the course of the attack and lessened its severity.

In one instance, it was possible to treat one eye and use the other as a control. This patient, A. R., had a severe bilateral central keratitis which was at first thought to be due to congenital syphilis. However, intensive penicillin therapy caused no improvement of the lesion, and it gradually assumed the appearance of a tuberculous keratitis. Beta irradiation was started on the more severely affected left eye December 28, 1944. Seven applications at two week intervals, using spray technic at the limbus, with a total of 52 gram seconds were given. Both eyes remained markedly inflamed for eight weeks. By the tenth week, the right eye was almost quiet; the left eye still showed slight pericorneal congestion but its cornea was clearer than that of the right. At the end of four months the treated left eye was the better one with less deep scarring and vascularization. A slit lamp picture of the right eye shows the corneal nebulae and deep vessels which were not present in the treated left eye.

An example of improvement in milder lesions is that of I. M., a 50 year old woman who was first seen October 20, 1944, with a typical sclerokeratitis of the right eye of three month's duration. She had been hospitalized for one month by her local doctor for typhoid and sulfonamide therapy and the usual local treatments had been used without improvement. The first treatment was of 5 gram seconds contact treatment in three areas at the site of the scleral inflammation three to six o'clock. Symptoms were markedly reduced after the first treatment and disappeared completely after the second one of 5 gram seconds sprayed over the limbus at 3 to 5 o'clock. A third spray treatment of 5 gram seconds was given in the sixth week. When checked, six weeks after the last treatment, the eye had already been asymptomatic for nine weeks and the lesion had remained clinically unchanged for five weeks. The pre- and post-treatment vision was 20/30. The eye has remained quiet for one year to date.

Another patient, J. V., suffered a violent keratitis, for three months. There was sclerosing of the superficial vascular trunks by the irradiation, with

**TABLE II.**

**Seventy-two Eyes with Anterior Ocular Tuberculosis Treated with Beta Irradiation**

| | Healed | | Improved | | Unimproved | |
|---|---|---|---|---|---|---|
| Eyes 72 | Number | Per Cent | Number | Per Cent | Number | Per Cent |
| | 38 | 52.8 | 28 | 38.9 | 6 | 8.3 |

**Criteria**—(1) **Healed** quiet eyes, no corneal infiltrates, negative aqueous ray, no nodules in the iris, no K.P. followed for one year.
(2) **Improved** asymptomatic, no more than residual conjunctival hyperemia. A-R negative, corneal infiltrates inactive or healed eyes followed less than one year.
(3) **Unimproved** decrease in vision due to disease, no alleviation of symptoms, progression of the disease in spite of adequate therapy.

**TABLE III.**

**Comparison of Initial and Final Vision in Seventy-two Eyes with Anterior Ocular Tuberculosis Treated with Beta Irradiation.**

| | Improved | | Maintained | | Decreased | |
|---|---|---|---|---|---|---|
| 72 Eyes | Number | Per Cent | Number | Per Cent | Number | Per Cent |
| | 30 | 42% | 33 | 46% | 9* | 12% |

*The decrease in vision in 2 of the 9 eyes was due to the progress of senile cataracts.

a clearing of the cornea as the active process quieted down. The total spray therapy was 40 gram seconds given in two courses. Vision improved from CF ft. to 20/40.

*Results*

Table II gives the results of treatment in the 72 eyes with anterior ocular tuberculosis.

Eyes were considered healed if they were quiet, with no corneal infiltrates, a negative aqueous ray, no iris nodules, and no K.P. These eyes were all followed for at least a year. In 7 eyes the above criteria for healing were present with the exception that the per cent of observation is less than one year. They are therefore, placed in the improved group. Eyes were considered improved if after therapy they were asymptomatic and showed no more than residual conjunctival hyperemia, a negative aqueous ray, and inactive corneal infiltrates. Eyes classed as unimproved showed no alleviation of symptoms or a progression of the disease in spite of adequate therapy. Fifty-two and eight tenths per cent were considered healed after treatment. Thirty-eight and nine tenths per cent of the eyes were improved. Eight and three tenths per cent showed no improvement in spite of adequate therapy. Since any clinical classification such as *healed, improved* and *unimproved* presents obvious difficulties, a comparison of vision before and after treatment is especially interesting.

Vision was improved two lines or more in 42 per cent of the eyes, maintained in 46 per cent and decreased in 9 eyes or 12 per cent. The decrease in vision in 2 of the 9 eyes was due to the progress of senile cataracts.

*Comments:*

A comparison of the clinical and visual results is interesting as it shows a close correlation between the total eyes considered healed and improved, and the total eyes in which vision was either improved or maintained. This suggests that the clinical observations of amelioration of symptoms and shortening of the duration of the disease were indications of an actual decrease in severity of the lesion with less permanent damage to the eye.

In general, active treatment was continued as long as rapid improvement occurred. Healing occurred in one case after a total of 31.9 gram seconds had been administered, in another 223 gram seconds of treatment over a four year period was given during which time there were two exacerbations. This eye has now been healed for two years.

As a rule in the treated eyes subjective improvement preceded objective improvement by about two weeks. Objective improvement was usually noted in about four weeks and eyes were either quiet or markedly improved in six to eight weeks. In extremely severe lesions, longer treatment was required.

While beta irradiation seemed to have a beneficial effect on the immediate attack, it did not prevent future attacks. On this basis, a long term therapy in the form of tuberculin treatment was advised in all patients.

The mode of action of irradiation in ocular tuberculosis is unknown. It has been pointed out that both beta and grenz rays increase the antibody reaction in the cornea, and it may be in this way that the beneficial results are obtained.

### BENIGN TUMORS OF LIDS AND CONJUNCTIVA

The removal of small benign tumors of the lids and conjunctiva is usually carried out for cosmetic reasons, thus a form of therapy which will produce the least residual scar is important. In some cases the surgical approach is more expedient and more satisfactory; in others, irradiation is more desirable. It is important for the ophthalmologist to differentiate between benign and malignant lesions, and in case of any doubt a biopsy should be done. Most malignant lesions usually require deeper and more intensive therapy and should be referred to the radiologist.

The data on 24 cases of benign tumors of the lids and conjunctiva are given with the dosages and the length of time necessary to effect a disappearance of the lesion. Angiomas as a rule respond well to irradiation except the nevus flammeus, or port wine stain type. With these, we have uniformly had very little success and therefore no longer recommend it. In contrast, the nevus vasculosis, or strawberry mark, frequently seen at the caruncle or involving the cul-de-sacs responds dramatically. J. G., a 3 month old baby, had a lesion involving the caruncle, nasal upper and lower cul-de-sacs, and nasal portion of left upper lid. Five treatments, totaling 42 gram seconds, were given in contact with the lesion over a period of six months time. Under this therapy there was progressive improvement.

A 4 year old boy, A. Z., had a thickening of the lid and partial ptosis from an angioma cavernosus. These are usually completely subcutaneous and consist of dilated vessels producing an elevation and bluish color to the skin. Five months later, after three treatments totaling 19.8 gram seconds, he was much improved. The response with this type of lesion is excellent but not so dramatic as in nevus vasculosis.

The pedunculated papillomas of the lids usually dry up after one, or, at most, two treatments. One patient with this type lesion is shown before treatment and after 5 gram seconds contact.

Papillomas of conjunctiva respond rapidly and leave little residual scar. D. B., a 10 year old colored girl, developed a mass in the upper cul-de-sac which on section was a papilloma. Three contact treatments were given totaling 37 gram seconds at two week intervals.

Papillomas of the bulbar conjunctiva present the most frightening picture of the benign tumors. Their growth is usually rapid and they frequently extend so far over the cornea that they affect the vision. With these, biopsy is

**TABLE IV.**
**Benign Tumors Treated with Beta Irradiation.**

| | Name Age | Lesion | Dosage | Result |
|---|---|---|---|---|
| Angiomas | J. G. 3 mo. | nevus vasculosis caruncle, upper and lower cul-de-sac, nasal portion of left upper lid causing partial ptosis. | 5 treatments over 6mos. 42 gm. sec. total to different areas in contact with lesion. | Slow resolution of lesion |
| | A. Z. 4 yrs. | angioma cavernosus, bluish discoloration and thickening of skin of right upper lid with partial ptosis. | 3 treatments 3 weeks apart, totally 19.6 gm. sec. Contact with lesion both sides of lid. | Complete resolution within 2 months |
| | H. L. 5 mos. | angioma vasculosis, left upper lid and left hand. | 4 treatments over 5 mos. 40 gm. sec. just short of contact with lesion. | Complete resolution in 5 months |
| | G. P. | small mulberry angioma, bulbar conjunctiva. | 2 treatments, 14 gm. sec. | Lesion completely disappeared, 2 mo. |
| | Mrs. G. | angioma of caruncle. | 2 treatments, 6 gm. sec. each | Lesion completely disappeared, 4 weeks |
| | J. D. 4 years | angioma inner canthus | 1 treatment 8 gm. sec. contact | Lesion disappeared 3 weeks. |
| Papillomas, lids | J. K. | small pedunculated papilloma, right upper lid margin. | 1 treatment, 14.3 gm. sec. | Tumor fell off in 2 weeks |
| | J. W. | papilloma, right lid marmargin. | 1 treatment, 6.9 gm. sec. | Tumor fell off in 3 weeks. |
| | H. W. | papilloma, upper lid margin. | 2 treatments, 9.3 and 7 gm. sec. 1 week apart. | One month lesion disappeared. |
| | J. N. | small papilloma, left lower. | 10 gm. sec. 9.75 gm. sec. 1 week apart. | Two weeks lesion disappeared |
| | Mrs. B. | flat papilloma, right upper lid. | 8 gm. sec. contact. | Lesion $\frac{1}{2}$ size in 3 weeks, patient became ill and could not return for further treatment |
| | S. C. | flat papilloma, upper lid. | 4 treatments, 3 week intervals, 7, 9, 6, 8 gr. sec. | Lesion did not change, treatment discontinued |
| | Mrs. W. | pedunculated. | 1 treatment, 10 gm. sec. | Lesion disappeared 2 wks. |
| | Mrs. R. | flat. | 3 treatments, 10 gm. sec. | Lesion flattened 2 months. |
| | Mrs. P. | flat papilloma right upper. | 3 treatments. 6 gm. sec. each. | Lesion did not respond, was surgically removed. nevus on section. |
| Papilloma Conjunctiva Bulbar | P. U. | papilloma of caruncle. | 1 treatment, 7 gm. sec. contact | Lesion disappeared in 2 weeks. |
| | F. R. | papilloma of caruncle. | 3 treatments, 26 gm. sec. in contact with lesion. | Lesion disappeared in 1 month. |
| | M. W. | papilloma with granulation tissue at caruncle. | 7.9 gm. sec. in contact with lesion. | Lesion flattening in 4 weeks. |
| Papilloma Bulbar Conjunctiva | D. B. | papilloma of upper cul-de-sac. | 3 treatments 2 weeks apart in contact with lesion. 10, 15, 12 gm. sec. | Lesion disappeared 2 months. |
| Papilloma Bulbar Conjunctiva | H. C. | papilloma bulbar conj. LE. | treatment, 75 gm. sec. in 8 treatments. | Lesion completely disappeared. Vision 20/40, seen 2 years later. |
| | M. S. | papilloma bulbar conj. LE. | 4 treatments in contact with lesion, 4 areas, each treatment, total 57 gm. sec. | Lesion completely disappeared. Vision 20/20 3 years. Later no recurrence. |
| | W. G. | papilloma partially covering both cornea. RE lesion surgically removed, base irradiated. LE just irradiated. | RE 2 treatments, total 7 gm. sec. LE 5 treatments, total 40 gm. sec. | Both lesions completely disappeared. |
| | A. G. | papilloma marking left cornea. | dosage contact 5 treatments 45 gm. sec. total. | Lesion cleared and remained clear. Pt. wrote 1 yr. later. |
| | D. S. | papilloma at limbus early. | 32 gm. sec. 3 treatments. | Disappeared in 8 weeks. Followed 8 months. |

routinely done to rule out epithelioma. Both the benign and malignant forms, however, respond well to beta therapy.

A 64 year old woman with a typical bulbar papilloma of three months duration was seen first on May 8, 1943. Biopsy confirmed the clinical diagnosis. Contact treatment consisting of 57 gram seconds was given in four doses over a period of six weeks. In ten weeks the lesion had completely disappeared. The patient has been followed now for two and one-half years, and the eye has remained quiet, with vision 20/20.

A similar lesion occurred in a 60 year old man. After a total of 75 gram seconds contact and spray treatment, the resulting vision was 20/30. There has been no recurrence in three years.

We wish to emphasize the fact that the removal of benign tumors of the anterior ocular segment for cosmetic reasons is in the province of the ophthalmologist and that beta irradiation is practical and gives excellent results.

### CORNEAL LESIONS

*Punctate Keratitis*

Five patients, with a typical punctate keratitis which had responded to no other therapy, were sent to the radium clinic for treatment. Notwithstanding the fact that a punctate keratitis can be produced by beta irradiation, 2 of these were given low doses, 3 - 5 gram seconds; 3 were given a full course of 18 gram seconds. One in the latter group improved then relapsed. The other 4 showed no improvement whatever.

*Corneal Ulcers*

Eleven patients with corneal ulcers, due either to staphylococcus or mixed infections which had not responded to local therapy, were given one course of treatment. One patient showed marked improvement but shortly developed a marginal infiltrate in the other eye. The picture became typically that of a tuberculous keratitis which rapidly responded to irradiation therapy. The first eye was probably also on this basis with a superimposed secondary infection.

In 6 patients the treatment seemed to make the ulcers definitely worse and 3 were admitted to the hospital. On 2 of these, conjunctival flaps were pulled as the cornea became so thin that perforation was feared. The third seemed to improve on local therapy in the hospital, and the ulcer slowly healed. In 4 eyes where the purulent discharge was not so marked, only very questionable improvement occurred after the first course of treatment. However, after the discouraging experience with 3 cases that required admission to the hospital a second course of therapy was not given. Here again the result is as would be expected, in that irradiation hastened the break down of the already damaged cells.

*Chronic Ulcers*

Nine patients with corneal ulcers considered of an acne rosacea keratitis origin have been treated. These consisted of stromal infiltrates usually in the lower corneal quadrants into which went deep and superficial vessels. The epithelium usually was intact but on some occasions staining occurred. Treatment at the limbus produced occlusion of the vessels and with the decrease in vascularity the corneal lesions improved in 6 cases. In the 3 cases where there was no improvement the purulent discharge and conjunctival factor were prominent features.

The corneal lesion of L. G., a 50 year old woman, with an acne rosacea keratitis of six months duration was treated in three courses of spray and contact therapy totalling 45 gram seconds. In a follow-up of two years, there has been no recurrence.

A second patient, A. R., was given 115 gram seconds of therapy during sixteen months. Both spray and contact therapy were used and a few very

large vessels were occluded with actual cautery. The lesion has now been quiet for two and one-half years.

*Corneal Scars*

A great variety of corneal scars have been treated and the results are extremely difficult to evaluate. Old dense scars or those with calcium deposits show no change even after prolonged therapy.

F. G. had a herpetic corneal lesion with a resultant heavy corneal scar. He was given 155 gram seconds over six years time. This man had adequate treatment, yet showed absolutely no change in the density of the scar. Old fibrous tissue is relatively resistant to irradiation and this result could have been anticipated.

Recent corneal lesions with thickening of the stroma and vascularization on the other hand do seem to show clearing with treatment although here a control is difficult. An example is C. S. whose left eye suffered a sulphur dioxide burn. For a year, many kinds of local treatments had been tried without signs of improvement. Vision was reduced to 4/200 not improved with pinhole. The eye was extremely painful and there was marked photophobia and lacrimation. The temporal half of the limbus was treated with 5 gram seconds contact therapy and the vessels in this area occluded. Vision improved to 20/200. The lower nasal quadrant of the limbus was also treated; then the whole limbus. Therapy was given at four week intervals with a total dosage of 5 gram seconds contact to each area and 10 gram seconds spray. Vision one year later was 20/70 and with pinhole 20/50; the eye was asymptomatic. In this case vascularization played a large part and with beta irradiation it was possible to occlude the vessels. Young fibroblasts are sensitive to irradiation and their activity inhibited. In considering this feature, however, the relative sensitivity of the stromal cells is important and to date we have no experimental evidence to show they are more resistant than the invading fibroblasts.

Lamellar keratectomies are routinely treated at the limbus with beta rays to prevent vascularization. The best results have been obtained with a course of treatment on the fifth to seventh day and again on the twenty-first day. The applicator is applied as close to the limbus as possible, and the circumference is treated by quadrants using the spray technic. An attempt is made to confine the treatment chiefly to the limbus and conjunctiva, rather than over the new corneal epithelium. When irradiation is delayed until new vessels have already entered the cornea, larger doses are required to cause occlusion. This is produced most satisfactorily by contact treatment over the vessel at the limbus.

L. K. was a 28 year old woman with a fatty degeneration of the cornea. After keratectomy vascularization at the limbus was well controlled by beta rays. The vessels developed clubbed ends and did not extend further.

In keratoplasties beta is immediately used if there is any evidence of new vessel formation or old vessels opening up, for the superficial vascular process can in most cases be controlled by this method. Deep vessels are more difficult to treat and in some instances it is necessary to use actual cautery. Corneal clouding due to other causes, is not helped by irradiation and treatment is contraindicated.

*Pterygiums and Psuedoterygiums*

Eighteen patients with pterygiums have been treated and in only one did treatment seem slow in obtaining results and operation was performed. In cases where a recurrence is seen after operation, beta irradiation is especially valuable. Contact therapy gives the more satisfactory results.

Conjunctival flaps over the cornea, and granulation tissue can be flattened and made avascular. B. C. suffered a severe corneal ulcer following an injury. A conjunctival flap was pulled over the cornea to save the eye. Irradiation was subsequently used to flatten the flap. In N. V., a child of 7, a neglected chalazion produced a horn of granulated tissue on the skin surface of the lower lid. One treatment of 10 gram seconds caused shrinkage and healing of the lesion.

*Other lesions*

One case of blastomycosis of the outer canthus was successfully treated by contact and spray beta irradiation therapy and the lesion completely disappeared.

Nine patients with sarcoid involving the cornea and iris were treated without benefit.

In corneal dystrophies, lupus erythematosus and pemphigus, no improvement was noted after adequate therapy.

## CONCLUSION

1. In the treatment of vernal conjunctivitis, beta irradiation gives excelcelent results. Recurrences are less frequent after treatment and when these do occur, they are less severe. Sixty patients were treated, with 30 per cent cured, 63 per cent improved and 7 per cent unimproved.
2. In anterior ocular tuberculosis, beta therapy seems to shorten the course and decrease the severity of the symptoms with less residual scarring of the cornea. Irradiation, though, did not prevent future tuberculous recurrences.
3. Small benign tumors of the anterior ocular segment can be removed by beta irradiation easily and safely and give excellent cosmetic results. Since it is extremely easy to use in an office and requires neither hospitalization nor anesthesia, most patients prefer it to surgery. These advantages make it particularly satisfactory for work with children.
4. In corneal scars, where vascularization is a prominent feature and where there is still activity, irradiation appeared to be beneficial. In old, calcified, and dense scars, treatment is not successful.
5. Beta irradiation is ideal for preventing superficial vascularization after keratectomies and keratoplasties.
6. Granulation tissue, conjunctival flaps and pterygiums can be made avascular with contact therapy.
7. Encouraging results are obtained in the treatment of acne rosacea keratitis, with limbal application to obliterate vessels.
8. Dystrophies, sarcoid, lupus erythematosus, pemphigus, punctate keratitis and corneal ulcers due to pyogenic infections are not improved by beta rays and may be made worse by their use.

# PLASTIC OCULAR PROSTHESES IN UNUSUAL SOCKETS

BENJAMIN F. SOUDERS, M.D.
READING, PA.
BY INVITATION
HARVEY J. FORESTNER, D. D. S.
ST. LOUIS, MO.
BY INVITATION

THE occurrence of injuries in the military service necessitating enucleation and resulting in unusual and deformed sockets has frequently demonstrated the inadequacy of the glass ocular prosthesis. This problem has led to consideration of a plastic (acrylic resin) compound for the fabrication of ocular prostheses. The primary purpose of this paper is to present a preliminary report of a method of procuring a satisfactory socket impression, especially in deformed sockets from which functionally and cosmetically adequate plastic prostheses can be produced. The impression-molding procedure to be described has been evolved by us during the past few months and has yielded encouraging results in the fabrication of ocular prostheses.

## METHOD

The impression material which we have employed with satisfaction consists of an alginate gel and is commonly used in dental impression technics. This material is marketed in the form of a very fine powder with the consistency of talc, which, when mixed with distilled water at room temperature creates a semi-liquid mass which "sets" or gels after a period of from two to five minutes. The resultant setting of the impression material produces a fairly solid mass which possesses a consistency ideal for proper impression of any surface with which the material has been in contact.

An impression tray has been produced from acrylic resin which has the general form of a small ocular prosthesis without the trochlear notch. This tray has a scleral radius of curvature of 12 mm. and a corneal bulge with a radius of curvature of 7.5 mm. located slightly nasally to the center of the tray. A perforation at the corneal vertex accommodates a screwhead dental mandrel, attachment of which was found to facilitate the taking of the impression and its removal from the socket. Perforations paralleling the periphery secure the impression to the tray, which because of its form becomes an essential part of the finished impression. An inner ring of perforations 11.5 mm. in diameter and centered on the vertex has also been found to be of assistance, as will be discussed later.

A plaster bowl, graduate, Jaeger spatula, iris spatula, and muscle hook comprise the remainder of equipment required for the impression technic. (Fig. 1).

The patient is seated in a chair with headrest in order that the head be held in a fairly firm, upright position. A topical anesthetic agent may be instilled into the socket, but does not appear to be essential, since no great discomfort seems to accompany the steps to follow. Two grams of the impression material are then mixed with 9 cc. of distilled water at room temperature. The mix-

Presented at the Forty-Ninth Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 8-12, 1944, Chicago, Ill.
From the Eye, Ear, Nose and Throat Section of the Surgical Service and the Dental Service, Bushnell General Hospital.

ture is spatulated from 60 to 90 seconds and a large portion of it is introduced into the socket by means of a Jaeger spatula. An iris spatula is then employed to remove air pockets and cause even spread of the material throughout the socket. Impression material is then placed on the posterior surface of the tray and the latter is positioned over the previously inserted material. At this point the patient is instructed to look straight ahead. By manipulation with the aid of the mandrel, the 11.5 mm. circle of perforations in the tray is positioned in the bed of impression material so that future alignment of the prosthetic iris with the remaining eye will be reasonably accurate. The tray is then impressed into the socket until the future desired width of the palpebral fissure and "fullness" of the future prosthesis is secured. Any excess of impression material will find exit through the perforations in the tray or around its margins. (Fig. 2). The excess is allowed to remain until setting of the impression material ensues. The afore-mentioned procedure usually consumes about 4 minutes from the time the impression material is mixed and can be performed without haste after brief experience. Setting of the material can usually be judged by testing the excess with the iris spatula. After setting, the excess impression material is cut away with the iris spatula, following which a muscle hook is placed in the lower fornix and is insinuated between the posterior surface of the impression and the conjunctiva. With gentle manipulation of the mandrel the impression is delivered from the socket, usually beginning with the outer aspect of the lower lid. The impression is then inspected (Fig. 3) and obvious artifacts are removed with a sharp knife or scalpel. Excesses beyond the margin of the tray are pared carefully so that a generalized transition into the entirely bared anterior surface of the tray is effected. Any herniation of the impression material through the perforations in the tray are cut away, adhesion to the main body of the impression being assisted by obliquity of the perforations. With completion of the trimming process a total impression of the future prosthesis is obtained, the tray creating

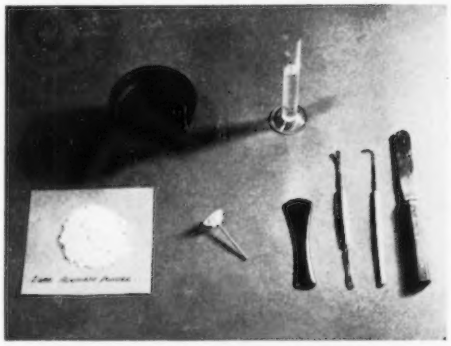


FIG. 1—Items used in taking impression of socket. Acrylic impression tray (center) has detachable screw-head dental mandrel.

FIG. 2—Mixed impression material is placed in socket and tray is superimpressed and centered.

FIG. 3—Total impression of socket for future prosthesis.

FIG. 4—Impression is invested in dental store. After investment of anterior curvature the tray is removed from the mold by removing screw which secured mandrel to tray.

the anterior "exposed" surface and the impression material creating the unexposed anterior surface and the posterior surface.

The impression is washed in cold water, placed in a hardening solution for 10 minutes, and the posterior surface is invested in dental stone in the lower half of a small flask with the screw portion of the mandrel remaining with the impression, the shank having been removed. (Fig. 4). A separating agent is applied to the stone, and the upper half poured. After setting, the two halves of the flask are separated, the screw and tray removed, and upon the application of tinfoil to the stone the equivalent of a metal mold is obtained which will assure an accurate and detailed reproduction of the impression. (Fig. 5).

The actual fabrication of the prosthesis is essentially the reproduction of the impression in methyl methacrylate, using a molding technic similar to that employed in making dentures, the resultant form being further contoured to secure the desired size and shaped fissure. (Fig. 6). The iris perimeter is described with a caliper (Fig. 7), and a recess is prepared to receive the iris. The iris may be obtained by painting oil pigments on a disc of photographic film base, using the good eye as the

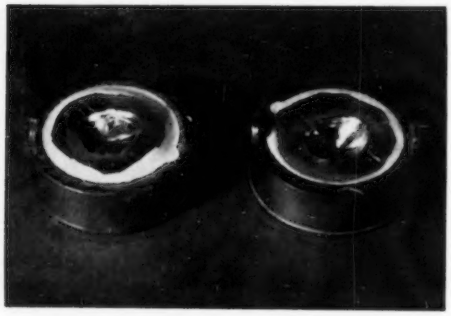


FIG. 5—Mold of impression after tin foiling.

FIG. 6—Reproduction of impression with methyl methacrylate ("acrylic," "plastic") by pressure-molding and curing in boiling water.

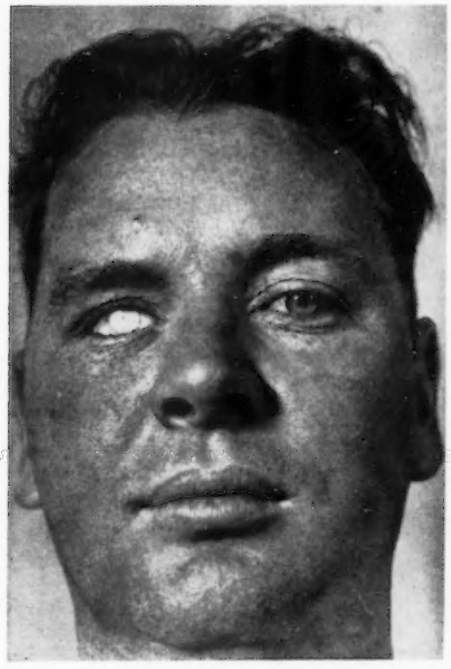


FIG. 7—Location of future iris perimeter.

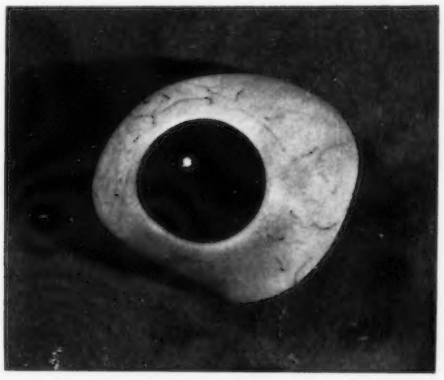


FIG. 8—Semi-finished prosthesis with recess accommodating a glass iris disc.

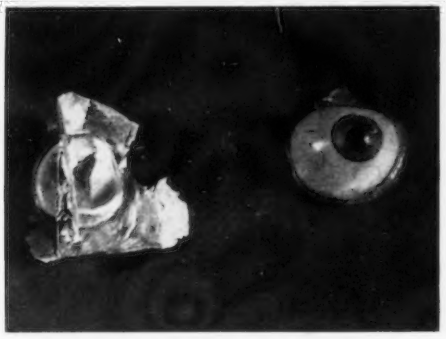


FIG. 9—The prosthesis after final curing. Excess clear acrylic is ground away and a high polish over entire prosthesis is produced.

model. If this procedure is not feasible a glass iris may be salvaged from an old or new glass prosthesis. The surface of the iris does not necessarily have to be perfectly smooth, since it will be completely encased in plastic, thus nullifying surface irregularities because of similar indices of refraction of plastic and glass. Between 1 and 2 mm. of material is then removed from the anterior surface of the semi-finished prosthesis to provide for a layer of optically clear plastic. (Fig. 8). After stains, pigments, and simulated blood vessels have been applied to the scleral portion, clear methyl methacrylate is pressed over the anterior surface and, after curing, (Fig. 9), the prosthesis is finished and polished. Usually only minor adjustments are necessary to secure an acceptable prosthesis.

### CASE REPORTS

The following cases are briefly reported to illustrate the possibilities inherent in plastic ocular prosthesis derived from the impression-molding technic just described:

Case No. 1—B.P., a 25 year old soldier had an apparent evisceration of the right eye following a crushing injury of the right maxillary bone, sustained when he fell from a railroad car in 1933. A marked bony depression of the inferior lateral quadrant of the orbital margin was noted when the patient was first seen by us and the socket was markedly distorted. (Figs. 10 and 11). The patient stated that he had never worn a satisfactory prosthesis, custom-made or stock. An acrylic prosthesis was produced by impression-molding technic which was completely comfortable and produced an excellent

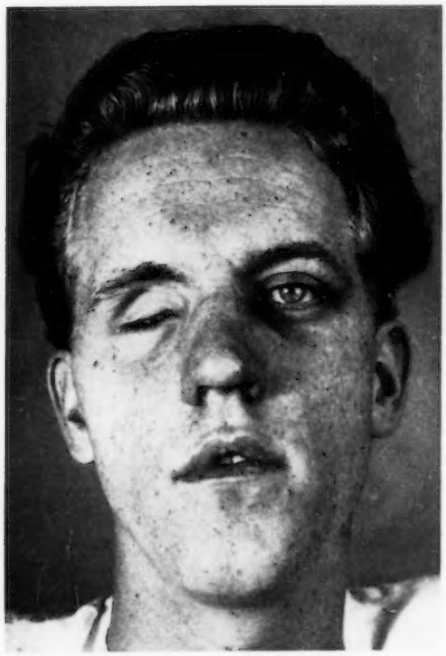


FIG. 10

FIG. 11

FIGS. 10 and 11—Patient with considerable bony deformity of right infra-orbital region and irregularity of socket.

FIG. 12

FIG. 13

FIGS. 12 and 13—Patient wearing prosthesis derived from impression-molding technic.

FIG. 14—Prosthesis noted in Figures 12 and 13.

cosmetic result. (Figs. 12, 13, and 14).

Case No. 2—H.S., a 21 year old Korean-American and a member of the Rangers, was injured by an enemy sniper bullet which entered the left maxillary region, traversed ethmoid cells, nasal passages and right orbit, to emerge over the right malar region. Simple enucleation was performed immediately following his injury. When the patient was seen at Bushnell General Hospital, two months following his injury, he demonstrated severe contraction of the socket, and absence of any motor function of the extraocular muscles apparently due to orbital disorganization produced by the bullet wound. He also demonstrated complete paralytic blepharoptosis as well as bony deformity of the nasal orbital wall.

Following the revision of the bone deformity a Thiersch graft was applied to the socket, thus creating an adequate cavity for accommodation of a prosthesis. (Fig. 15). Partial correction of the patient's blepharoptosis was accomplished after an acrylic prosthesis had

FIG. 15—Large, irregular socket obtained after scar excision and skin grafting for contracted socket.

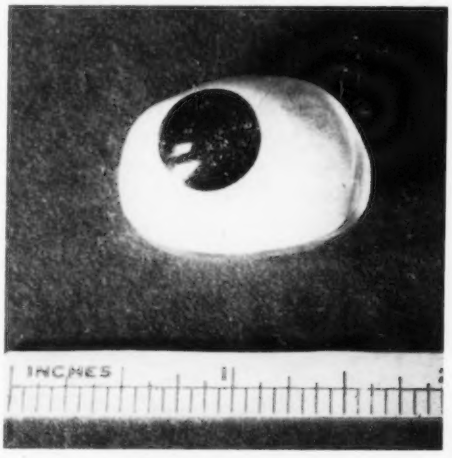



FIG. 16—Unusual form of prosthesis created by impression-molding. The prosthesis is viewed exactly as it reposed in socket.

been produced. The prosthesis is depicted in Fig. 16 exactly as it was disposed in the patient's right socket. In spite of its unusual shape and its fixa-

FIG. 17

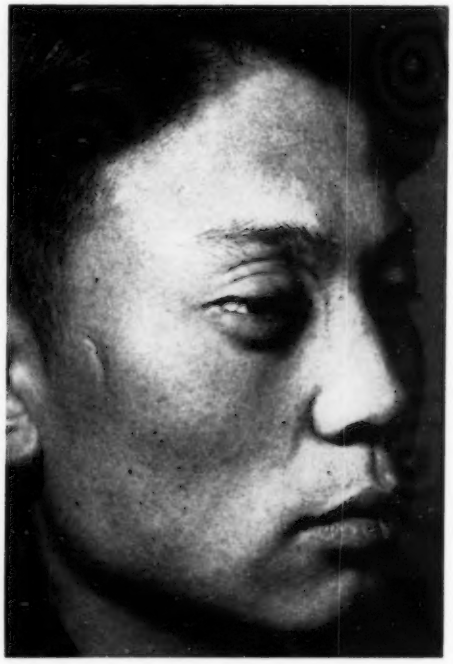


FIG. 18

FIGS. 17 and 18—Appearance of patient wearing prosthesis.

tion due to the absence of extraocular muscle function, the prosthesis created a satisfactory cosmetic result. (Figs. 17 and 18). In our opinion a comparable result could not have been achieved with any type of glass prosthesis.

SUMMARY

A technic of producing plastic ocular prostheses by impression and molding is briefly described. This method is recommended for consideration in cases of unusual and markedly irregular sockets, as exemplified by two cases which are reported.

# In Memoriam

Joseph Marie Louis Bruno ..... Brooklyn, New York
July 14, 1946

Thomas E. Carmody ..... Denver, Colorado
August 30, 1946

Robert Lewis Crockett ..... Oneida, New York
May 27, 1946

Wells Phillips Eagleton ..... Newark, New Jersey
September 11, 1946

Charles Willard Geiger ..... Kankakee, Illinois
July 19, 1946

Gaylord Crawford Hall ..... Louisville, Kentucky
June 16, 1946

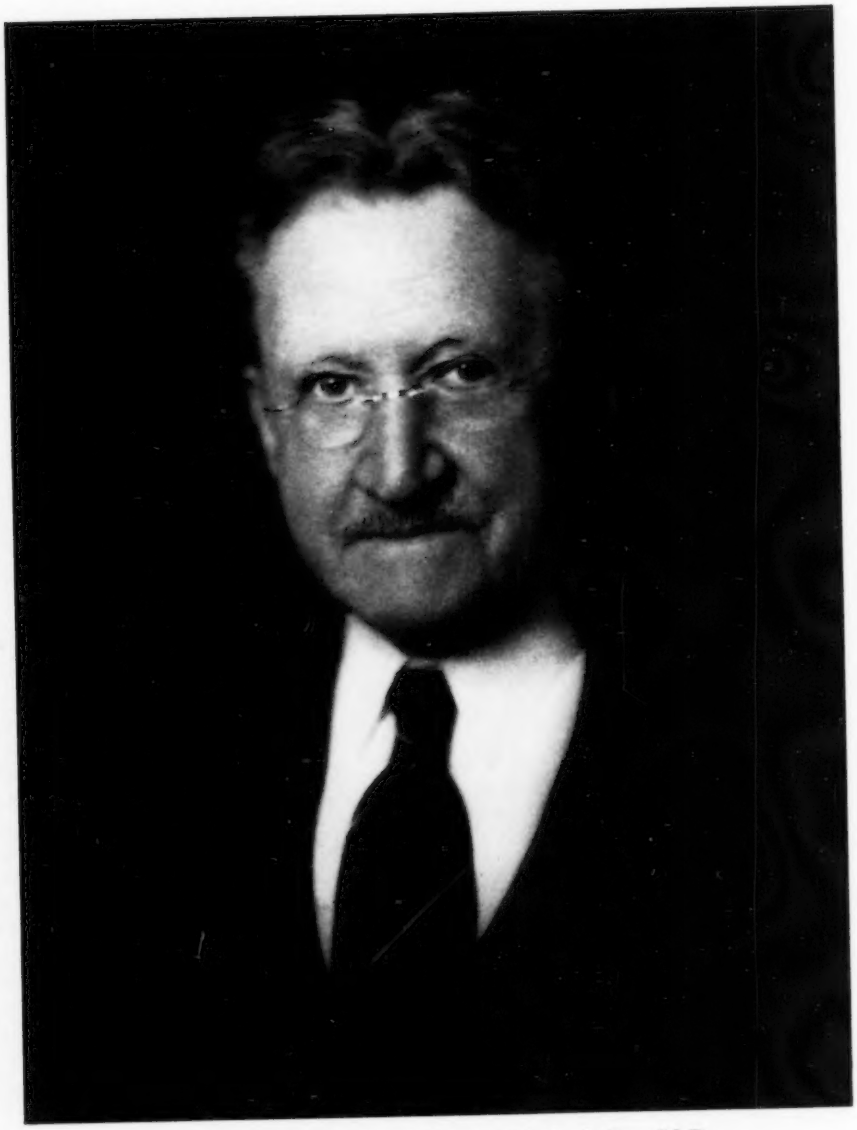
Thomas D'Arcy McGregor ..... Buffalo, New York
July 14, 1946

Howard Wilber Pierce ..... Detroit, Michigan
April 17, 1946

Theodore L. Terry ..... Boston, Massachusetts
September 28, 1946

Robert Von der Heydt ..... Chicago, Illinois
September 18, 1946

# In Memoriam



WELLS PHILLIPS EAGLETON, D.Sc., M.D.

1865 - 1946

## WELLS PHILLIPS EAGLETON, D.Sc., M.D.

### 1865 - 1946

*Dr. Eagleton was born September 18, 1865, in Brooklyn, N. Y., son of Thomas Aston Proud and Mary Emma Phillips Eagleton of American and English ancestry and a descendent of Thomas Guy, founder of Guy's Hospital, London, England.*

*He was graduated from the Polytechnic Institute, Brooklyn, and in 1888 was awarded his medical degree by the College of Physicians and Surgeons of Columbia University. After an internship of two years at the Newark City Hospital he began the practice of medicine in Newark, New Jersey, in 1890. He was awarded a doctorate of science from the University of Newark in 1942 and Rutgers University in 1943.*

*He was married May 24, 1913, to Mrs. Florence Peshine Riggs who survives him.*

*Dr. Eagleton was medical director of the Newark Eye and Ear Infirmary, secretary of the board of trustees and chief of cranial surgery. He was chief of the division of head surgery at Newark City Hospital. He served as consulting cranial surgeon at the following: St. Barnabas Hospital and Hospital for Women and Children; St. Michael's Hospital (Newark); Newark Memorial Hospital; Presbyterian Hospital (Newark); Beth Israel Hospital (Newark); Mountainside Hospital (Montclair); Muhlenberg Hospital (Plainfield); All Soul's Hospital (Morristown); Orange Memorial Hospital (Orange); Irvington General Hospital; Elizabeth General Hospital; and West Hudson Hospital of Kearny in addition to his duties as consulting ophthalmologist and otologist and cranial surgeon in numerous local and county hospitals.*

*In 1934, Dr. Eagleton was President of the American Academy of Ophthalmology and Otolaryngology. He was also a past president and member of the Essex County Medical Society (1908); Academy of Medicine of Northern New Jersey (1931); Academy of Medicine of New York; Medical Society of New Jersey (1923), (Chairman Welfare Committee (1923-24); American Otological Society (1921); New York Otological Society (1929); and the American Laryngological, Rhinological and Otological Society. He was a member of the American Medical Editors and Authors Association; A.M.A. (House of Delegates); American College of Surgeons (Fellow member Board of Governors); and associate member of the International College of Surgeons in which he was inducted in 1941. He held honorary membership in Sociedad Otorinoloargingingologica Madrilene, Spain; Academia Nacional de Medicine, Rio de Janerio, Brazil, 1936; Washington Medical and Surgical Society, Washington, D. C.; Guy's Hospital, London, England; and Kansas City Society of Ophthalmology and Otolaryngology.*

*In World War I he volunteered in the United States Army April 10, 1917, and was commissioned a Major in the Medical Corps and served as Chief of Surgery of the Head in the Base Hospital at Camp Dix, a post he held until after the signing of the Armistice. After the war he was made a Colonel in the Medical Reserve Corp, a commission he held until his death.*

*He was a member of the Essex County Country Club, Essex Club and Practitioners Club and the Browning Society of New York.*

*He contributed extensively to the medical periodical literature in his field. He was auhhor of "Brain Abscess" (Its Surgery, Pathology and Operative Treatment), 1922; "Cavernous Sinus Thrombophlebitis" 1945; (both translated into French); "Eagleton's Index—Abstracts of progress in Intercranial Lesions related to Aural and Nasal Conditions" 1941; and more than 100 monographs on cranial surgery.*

*In May, 1939, Dr. Eagleton was presented the Dr. Edward J. Ill Award by the Academy of Medicine of Northern New Jersery, the first to be given that honor. In 1940 he received the Award of Merit from the New Jersey State Medical Society, and in 1938 was awarded Gold Medal by Kings County Medical Society, N. Y.*

*Dr. Eagleton was distinguished not only in his medical career but also as a citizen of his community. He was president of the Newark Council of Social agencies and a trustee of the Welfare Federation of Newark, New Jersey, and of the Newark Museum. He was also a member of the New Jersey Commission for the Blind.*

*Dr. Eagleton's voice was frequently heard from the public platform and in the press, supporting health legislation, opposing political interference in medical matters, taking part in educational controversies and seeking to improve social welfare conditions in his city and state.*

*In 1941, Dr. and Mrs. Eagleton presented a building adjoining the headquarters of the Academy of Medicine of Northern New Jersey to the Academy to provide space for a larger library and for meetings.*

*Dr. Eagleton was deeply interested in international affairs and he and Mrs. Eagleton traveled to many parts of the world. On these journeys he always exhibited keen interest in medical and civic affairs of other nations and returned full of enthusiasm with knowledge gained while there.*

*Up to the very end of his long and brilliant career in his chosen specialty he contributed to the literature. His most recent articles were a complete review of advances in the understanding of rhinologic and otologic conditions related to the nervous system, published in the Archives of Otolaryngology for February, May, June and July, 1946. Dr. Eagleton was a man of strong convictions as to the trends and nature of medical practice, inclined usually to an intense individualism in his leadership.*

# In Memoriam

THOMAS EDWARD CARMODY, D.D.S., D.D.Sc., M.D.

1875 - 1946

## THOMAS EDWARD CARMODY, D.D.S., D.D.Sc., M.D.

### 1875 - 1946

*Dr. Thomas Edward Carmody, President of the American Academy of Ophthalmology and Otolaryngology in 1923, died August 30, 1946.*

*Dr. Carmody was born May 22, 1875, in Shiawassee county, Michigan and spent his early life in Owosso, Michigan. After receiving the degree of D.D.S. from the University of Michigan Dental School in 1897, he came to the University of Denver Dental College where he served as Professor of Bacteriology and Histology. He continued his studies and was awarded the degree of M.D. in 1903 from the Denver and Gross College of Medicine and in 1908, he received his D.D.Sc. from the University of Michigan Dental School. Dr. Carmody was Professor of Oral Surgery and Rhinology at the University of Denver Dental College until 1932. At the Medical School he served as assistant in Laryngology and Otology, and later as Chief of Otolaryngology, Child Research Council until 1936.*

*He was elected to the American College of Surgeons in 1913, American College of Dentists in 1929 and the International College of Surgeons in 1941. During World War I he served as a Major, Medical Corps, United States Army.*

*Dr. Carmody always gave freely of his time and effort to the various organizations of which he was a member. He held many offices in the local and state societies and served as first president of the organized Colorado Otolaryngological Society in 1919. He also served as president of the Colorado Society for Crippled Children, American Bronchoesophagological Society, American Laryngological, Rhinological and Otological Society, the American Laryngological Association, and was a charter member of the American Board of Otolaryngology.*

*His contributions to publications included many articles covering diseases of the mouth, nose, throat and ears and a chapter, "Diseases of the Mouth and Tongue" which appeared in a volume by Jackson and Jackson, 1945. His courses in the Instructional Program sponsored by the Academy were very popular.*

*Surviving him are his widow; two daughters, Mrs. Ruth Carmody Sumners of Denver, Mrs. Mary Alice Carmody Cobb, Minneapolis; and a son, David W. Carmody, Santa Fe, New Mexico.*

*Dr. Carmody was respected for his skill and wisdom; his pleasant companionship and leadership will be missed.*

TRANSACTIONS

American Academy of Ophthalmology and Otolaryngology

*Official Publication of the American Academy of Ophthalmology and Otolaryngology*

Published under the direction of the Editorial Board

WILLIAM L. BENEDICT, Rochester, Minnesota, *Editor*

W. HOWARD MORRISON, Omaha, Nebraska, *Associate Editor*

HENRY L. WILLIAMS, Rochester, Minnesota, *Associate Editor*

Printed by Douglas Printing Company, 108 North 18th Street, Omaha 2, Nebraska

All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, the American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

# High-Lights of the Fifty-First Annual Convention

THE American Academy of Ophthalmology and Otolaryngology held its fifty-first annual convention at the Palmer House, Chicago, October 13-18, 1946. Registration, including members, guests, exhibitors and ladies totaled 3,436.

### PRESIDENT'S RECEPTION

The president's reception, held in the Red Lacquer Room on Sunday afternoon, October 13, was attended by more than 1,000 members and guests. In the receiving line were Dr. and Mrs. Gordon B. New, Dr. and Mrs. George M. Coates and Dr. and Mrs. Alan C. Woods.

### SCIENTIFIC PROGRAMS

On Sunday afternoon at 2:30 o'clock the Committee on the Conservation of Hearing conducted a scientific program. At 8:00 o'clock Sunday evening the Committee on Industrial Ophthalmology presented a program consisting of three papers. The proceedings of these meetings will be published in future issues of the TRANSACTIONS. The Otosclerosis Study Group and the American Orthoptic Council and American Association of Orthoptic Technicians also held sessions on Sunday that were well planned and well attended.

This year the entire scientific program for the week was held in the Grand Ballroom. In spite of the fact that Instructional Courses were being conducted on the eighth floor simultaneously with the papers and motion pictures presented in the Ballroom a capacity crowd was always present. The Monday morning joint session consisted of addresses by the President and the Guest of Honor and a symposium on irradiation therapy presented by Dr. Curtis Burnam, Dr. Samuel Crowe and Dr. Charles E. Iliff of Baltimore. The papers presented in the subsequent Sections on Ophthalmology and Otolaryngology will be published in the next five issues of the TRANSACTIONS.

### ANNUAL ACADEMY BANQUET

The Academy banquet held in the Grand Ballroom, Tuesday, October 15, was attended by well over 700 persons. President Gordon B. New gave a welcome address and introduced the Guest of Honor, Dr. George M. Coates, and the President-Elect, Dr. Alan C. Woods. Presentation of honor keys was made to Dr. Frank R. Spencer of Boulder, Colorado, forty-sixth president of the Academy, retired December 31, 1945, as Senior Member of the Council; Dr. Ralph I. Lloyd, Brooklyn; Dr. Oscar V. Batson, Philadelphia; and Dr. Edmund B. Spaeth, Philadelphia. The Honorable Walter H. Judd, M.D., of the House of Representatives, Washington, D. C., delivered an address entitled "What Is Happening in Asia?" Dinner music accompanied the program.

### SCIENTIFIC EXHIBIT AWARDS

Six awards were presented by the Academy to participants in its scientific exhibit in connection with the annual session, three awards to exhibits on ophthalmology and three to exhibits on otolaryngology.

For his original investigation on "An Anatomical Study of the Oblique Muscles of the Eye" an award was made to Dr. Walter H. Fink of Minneapolis. In otolaryngology, Dr. Paul H. Holinger of the University of Illinois College of Medicine, Chicago, and co-exhibitors, Dr. Thomas Jones (by invitation) and Dr. Albert H. Andrews, Jr., received the award for original investigation on "Endoscopic Photography in Otolaryngology and Broncho-Esophagology."

To Dr. Gordon B. New of the Mayo Clinic, Rochester, and his co-exhibitors, Dr. John B. Erich, Dr. Frederick A. Figi, and Dr. Fred Z. Havens, an award was made for excellent presentation of their exhibits on "Partial and Total Loss of the Nose; Plastic Reconstruction" and "Cancer of the Larynx: Methods of Treatment and Results." In ophthalmology, the same award was made to Dr. A. D. Ruedemann of the Cleveland Clinic, Cleveland, for his exhibit "Plastic Eye Implant."

Awards for the teaching value of their exhibits went to Dr. Donald J. Lyle of Cincinnati for his display on "Neuro-Ophthalmology" and to Dr. Maurice F. Snitman of the University of Illinois College of Medicine, Chicago, and his co-exhibitors, Dr. E. V. Pellettiere, Dr. M. Skolnik (by invitation), Dr. D. J. Cronin (by invitation), and Dr. W. A. Donnelly (by invitation) for their exhibit "Surgery of the Neck."

Awards were announced by President New at the annual Academy banquet.

### DR. ALAN C. WOODS
### PRESIDENT

Dr. Alan C. Woods, Director of the Wilmer Ophthalmological Institute and Ophthalmologist-in-Chief at Johns Hopkins Hospital, Baltimore, Maryland, who has served the past two years as President-Elect, becomes President of the Academy on January 1, 1947. Dr. Woods has contributed generously to the literature of ophthalmology on the subjects of immunology and medical ophthalmology.

### DR. CARL H. MC CASKEY
### PRESIDENT-ELECT

Dr. Carl Heber McCaskey, professor of otolaryngology at Indiana University School of Medicine, was named President-Elect of the Academy at its annual business session on Wednesday, October 16. Dr. McCaskey received the degree of M.D. from Indiana University in 1908. Three years later he became a member of the American Academy whose interests and welfare he has held in mind always.

Other newly elected officers are: Dr. Arthur C. Jones of Boise, Idaho, First Vice-President; Dr. Phillips Thygeson of Menlo Park, Calif., Second Vice

President; Dr. Norton Canfield of New Haven, Conn., Third Vice-President; Dr. Lawrence R. Boies of Minneapolis, Secretary for Home Study Courses; and Dr. W. Ivan Lillie of Philadelphia, Councillor for a term of four years.

The following secretaries were re-elected: Dr. W. L. Benedict, Executive Secretary-Treasurer; Dr. Algernon B. Reese, Secretary for Ophthalmology; Dr. O. E. Van Alyea, Secretary for Otolaryngology; Dr. A. D. Ruedemann, Secretary for Instruction in Ophthalmology; Dr. Dean M. Lierle, Secretary for Instruction in Otolaryngology and Maxillofacial Surgery; and Dr. Erling W. Hansen, Secretary for Public Relations.

### PAN-AMERICAN CONGRESS

The first Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology, sponsored by the Academy through a special committee of which Dr. C. L. Jackson was the general chairman, convened on Friday, October 17, with Dr. G. B. New, President of the Academy, presiding. Scientific papers and motion pictures were presented by members of the newly formed society. English, Spanish and Portuguese were the official languages. Previously prepared abstracts of the papers in all three languages were available and interpreters were present to aid in discussions. The program occupied two days of scientific sessions. A Pan-American dinner complimenting our Latin American guests was tendered by the Academy on Thursday evening preceding the official opening of the Congress. At the University Club, the Latin guests were entertained at luncheon Friday noon and Friday evening they were entertained at a specially arranged exhibit of art in medicine during the war years by the Abbott Laboratories. Saturday afternoon, the association was formally organized at a business meeting and officers were elected. A full report of the Congress will be published in the TRANSACTIONS.

### CHICAGO — 1947

The Palmer House in Chicago has been selected by the Council as the place where the 1947 convention will be held. The dates of the meeting will be October 12-17 inclusive.

## ARMY INSTITUTE OF PATHOLOGY AND AMERICAN REGISTRY OF PATHOLOGY

WHAT is now known as the Army Institute of Pathology was established in 1863 as the Army Medical Museum. During World War II the activities of the Institute were greatly expanded, especially in the field of diagnostic pathology and research. There are now on file over 170,000 accessions. The results of research at the Institute during the past few years will be published in a volume of about fourteen hundred pages as a part of the official history of World War II. Director Colonel J. Earl Ash was succeeded on October 1 by Colonel Raymond O. Dart.

On request of Major General Norman T. Kirk, The Surgeon General of the Army, the Committee on Pathology of the National Research Council, Division of Medical Sciences, in late 1945 prepared a report on the future development of the Institute. The report has been approved by the Surgeon General and by the War Department.

The essential recommendations in this report are: 1) that a new building of adequate size be constructed; 2) that the Institute be organized in four divisions—Department of Pathology, Army Medical Illustration Service,

Army Medical Museum and American Registry of Pathology—each headed by a competent specialist; 3) that the staff of the Institute be drawn from both the commissioned ranks of the Army and from the civilian professions; 4) that a comprehensive educational and training program be undertaken; 5) that the vast store of material at the Institute be used for research and 6) that the services in pathology in the Veteran's Hospitals be centralized at the Institute.

The American Registry of Pathology founded in 1922 thus is, and will continue to be, an integral part of the Army Institute of Pathology. There were, on January 1, 1946, over 43,000 cases registered. To effectuate the new plans as they relate to the Registry, the National Research Council, Division of Medical Sciences, appointed a Committee on the American Registry of Pathology. The members of the Committee are Howard T. Karsner, Chairman, Cleveland; Colonel J. E. Ash, Washington; Brig. General R. Callender, Washington; Colonel Balduin Lucke, Philadelphia; Robert A. Moore, St. Louis; Benjamin Rones, Washington; A. R. Shands, Jr., Wilmington; and Henry A. Swanson, Washington.

At the present time there are fourteen registries as a part of the American Registry of Pathology. These include Registry of Ophthalmic Pathology, established in 1922, sponsored by the American Academy of Ophthalmology and Otolaryngology; Lymphatic Tumor Registry, established in 1925, sponsored by the American Association of Pathologists and Bacteriologists; Bladder Tumor Registry, established in 1927, Kidney Tumor Registry, established in 1940, and Prostatic Tumor Registry, established in 1943, sponsored by the American Urological Association; Registry of Dental and Oral Pathology, established in 1933, sponsored by the American Dental Association; Registry of Otolaryngological Pathology, established in 1935, sponsored by the American Academy of Ophthalmology and Otolaryngology; General Tumor Registry, established in 1937, sponsored by the American Society of Clinical Pathologists; Registry of Dermal Pathology, established in 1938, sponsored by the American Academy of Dermatology and Syphilogy; Chest Tumor Registry, established in 1942, sponsored by the American Society of Thoracic Surgeons; Registry of Neuropathology, established in 1943, sponsored by the American Association of Neuropathologists; Registry of Orthopaedic Pathology, established in 1943, sponsored by the American Academy of Orthopaedic Surgeons; Registry of Veterinary Pathology, established in 1944, sponsored by the American Veterinary Medical Association, and Registry of Gerontology, established in 1945, sponsored by the Gerontological Society, Inc.

Plans for additional registeries are under consideration. A professional scientific society wishing to sponsor a registry should communicate with the Director, Army Institute of Pathology, 7th Street and Independence Avenue, S.W., Washington, 25, D. C. The society appoints a committee to work with the Director in supervision of the activities of the Registry, and makes an annual contribution to the budget which is administered by the National Academy of Sciences.

All specimens in the Registry are available for review and research by competent investigators. Sets of slides and accompanying syllabuses on special fields are available for loan to the civilian professions and officers in the federal services. Physicians, dentists and veterinarians are urged to send unusual specimens together with an abstract of the history to the Registry. The contributor receives a report on each specimen and is asked to keep the Registry informed of the follow-up on the patient.

With the reorganization of the Army Institute of Pathology to be completed during 1946 and 1947, a full-time scientific director of the American Registry of Pathology will be appointed and sufficient clerks and technicians will be available to assure adequate use of the registries for diagnosis, research, training of young men, and education of the professions.

AMERICAN REGISTRY OF PATHOLOGY

Many diseases are so rare that any one individual or even the staff of any one hospital or clinic has a limited opportunity to study certain conditions and lesions. Conclusions in science must be based on not one, but many observations. Therefore, it is desirable to organize and support central agencies for the collection and filing of histories, specimens and follow-up studies.

For over twenty-four years the American Registry of Pathology has been maintained at the Army Institute of Pathology in Washington. It was founded in 1922 by the then curator of the Army Medical Museum, Brig. Gen. George R. Callender at the request of the American Academy of Ophthalmology and Otolaryngology. Succeeding curators, Major Paul E. McNabb, Colonel Virgil H. Cornell, Colonel Raymond O. Dart, and Colonel J. Earle Ash have given time and thought to the problems of the Registry. On January 1, 1946, material from over 43,000 cases was on file.

There are now fourteen separate registries, each sponsored by a professional scientific society. Important additions to knowledge have been made by investigations at the Registry notably on tumors and diseases of the eye, tumors of the lymphatic system, and tumors of the bladder.

With the reorganization of the Army Institute of Pathology and with plans for the erection of a new building sponsored by Major General Norman T. Kirk, The Surgeon General, the American Registry of Pathology takes on increased importance. There will be greater opportunities for the training of specialists, for research, and for educational activities.

The American Registry of Pathology deserves support from the civilian professions in terms of contributions of materials by individuals and sponsorship of registries by societies.

---

## Letter to the Editor

Philadelphia, Pennsylvania
October 11, 1946

Dear Dr. Benedict:

Besides the personal regret I have at not being able to attend the meeting of the Academy is the regret that I shall not be able to present before the Academy, as I presented before the American Ophthalmological Society and the Ophthalmic Section of the A.M.A., the terrific need for American ophthalmologists to take real concern over the facts as stated by Dutt in the December, 1937, "Archives of Ophthalmology," that, in spite of all that government and special missions do for the blind with cataract in India, there remain five hundred thousand people blind with operable cataracts whom our surgery, geared as it is, will never reach.

I should like to appeal to the Academy for these blind people and ask that should anyone have extra equipment, unused or used, instruments, from a cataract knife to a slit lamp microscope, that could be spared for the use of Indian ophthalmology such instruments might be collected and sent over for use by ophthalmologists in India. Contributions for this work would also be accept-

able, for it is my hope that a modern ophthalmic hospital with a research laboratory and scattered centers for ophthalmic surgery be established in our very needy area. In such an institution, instead of the one or two American ophthalmologists which we are now able to train in actual practice of ophthalmic surgery, we could train eight or ten or more, as we strive for our aim of doing 10,000 cataracts a year. The blind are there for us to give sight to. I appeal to the Academy for sympathetic consideration and practical interest in this weighty project.

I close, wishing the Academy the finest meeting they have ever had.

Sincerely and cordially,

(Signed) VICTOR C. RAMBO, M.D.

# INDUSTRIAL OPHTHALMOLOGY

## INTRODUCTORY STATEMENT

*It is quite evident from the ever increasing flow of letters of inquiry, dealing with industrial eye problems of every description, that interest is mounting rapidly in this newly developed field. This is especially true of ophthalmologists leaving the service and starting practice in industrial communities. This is a most heartening sign in view of the tremendous need of properly trained leadership in the field of occupational eye problems.*

*One of the phases of industrial eye work which most often baffles a medically trained person, is the proper integration of an Eye Protection (Safety) program into the general medical program of a plant. The questions that management asks relative to policy, type of protection, methods of enforcement, methods of servicing and maintaining, have in the past been altogether out of the sphere of the medico. However, the tool crib is no longer the proper place for goggles, either planos or corrected vision goggles. The latter have become a large part of an eye protection program. Hardened prescription lenses, their ordering and arrangements for dispensing, belong in the hands of professional consultants, and cannot well be separated out of a general eye protection program. Dreyer, in his excellent presentation, has shown how to "professionalize" an eye protection program. He has emphasized the most important things that are vital to a successful program, although these are all too often completely ignored. His success is demonstrated in the reduced percentages of eye injuries (in the one group he discusses) from 50 per cent to less than 30 per cent. Eyes were 15 per cent of all their lost time cases before the program was put into effect, and less than one-half of a per cent after the program was set up. This speaks for itself. The high spots of success are due to (1) individual fitting, (2) standardization of equipment, (3) follow-up service.*

*Most large industries have a house organ or company newspaper which contains material of interest to employees. Kozloski, of Western Electric in Baltimore, has written into such a sheet (The Pointer) as clear-cut and simple a statement of what their vision testing program means, as this commentator has found published anywhere for this purpose. He emphasizes quite properly that the testing of visual skills constitutes a procedure set up for matching physical capacities (eyes) to job demands. He also indicates how individuals not meeting standards are found and referred to corrective attention. His statement is included here as an illustration of this part of the overall educational approaches organized to handle industrial problems inside a plant.*

*After the publication of several confused articles on sun glasses in popular lay magazines outlining what these authors thought constituted proper protection from glare on the beach or elsewhere, Dr. Albert P. Jordan requested a statement on the healthful or beneficial effects of sun glasses from Dr. Thomas Allen. His answer is an excellent guide for all of us when comparable questions are asked us.*

*The October session of the Academy is providing excellent opportunities for conference and study of industrial eye problems from many angles. The Joint Committee appreciates the contributions of its guest lecturers on both the Sunday night program and the nine period continuous instruction course. We also note with pleasure that through the thoughtfulness of Dr. Hussey the Wayne University School of Occupational Health, in its Orientation Course scheduled for this fall, also includes the subject of Industrial Ophthalmology.*

HEDWIG S. KUHN, M.D.
*Secretary, Joint Committee*
*Industrial Ophthalmology*

---

# PROFESSIONALIZE YOUR INDUSTRIAL EYE PROTECTION PROGRAMS

CLINTON W. DREYER
Director, National Safety Council
OAKLAND, CALIF.

ALL of us in accident prevention work in considering eye protection programs long have considered the compulsory requirement on the part of management that all persons within the employment of a company should be required

to wear safety glasses as the ultimate possible to be desired. I challenge this type of thinking for several reasons. First, we have had an example of the compulsory type of program which has been very successful with an outstanding company for over twenty years, yet on a percentage basis very few large or small industries throughout the country have seen fit to follow the example set by this company in spite of the low accident rate, the increased production efficiency and the other salutary results achieved.

Frankly, I think that the compulsory eye programs are desirable but they have not had wide-spread acceptance because management and the advisory clients have not been sold on the proper methods for their installation. Further, we may not be as well informed on the subject as we would like to be, and have depended too often upon unqualified persons for a solution to this problem. Since we have not had universal acceptance under the compulsory system, we must find another approach.

What is wrong? It is my belief that we have failed to put finesse—through salesmanship, showmanship—into our eye protection program. Why not bring the professional qualities of the eye specialist into the preventive field? To better illustrate this point I will explain the development of an extensive personal protective equipment program during 1943 while I was engaged as assistant safety director at the Moore Dry Dock Company in Oakland, California.

In analyzing our first-aid cases reporting at the medical station in the yard, we learned that more than 50 per cent were eye injuries and approximately 15 per cent of our lost-time cases resulted from eye injuries that could have been prevented through the use of protective equipment.

We had our problems in getting proper goggles, hats, respirators, etc., from suppliers, just as everyone else, yet considering the quantity of equipment issued the amount of injuries indicated that it was not being used or, if used, was improperly applied. We, therefore, hired an interviewer to sit in the medical station to question all employees who had received eye injuries or other injuries that might have been prevented by protective equipment. This data was collected on a card which was analyzed daily and compiled into a monthly cumulative report.

The analysis revealed several definite conclusions, among them: lack of knowledge on the part of supervisors and employees as to the specific use and care of personal protective equipment; a general attitude that personal protective equipment interferred with production efficiency because it was either too heavy or awkward to wear.

From these conclusions we decided to embark upon an educational program which would have all of the elements of showmanship in it and yet would sell itself to supervisors and workmen alike. The medical director, the eye specialist and the hospital staff cooperated wholeheartedly in the planning of this program. They investigated hazardous conditions in various sections of the yard in company with a safety engineer so that they might have a better understanding of the preventive problem facing us.

Our first step was to build a place which we called a "Safety Dispensary" for the purpose of issuing and repairing personal protective equipment. Its interior appearance contained all of the aspects of a medical station or hospital for the psychological purpose of creating the respect of supervisors and employees for the equipment issued. An optometrist was hired to train Dispensary employees to fit and repair uncorrected eye protection equipment. This man assisted in the lay-out and designing of the Dispensary for maximum

operating efficiency and for a professional appearance. In addition he supervised the adjustment and alignment of all safety corrected glasses for the purpose of maintaining visual efficiency on the job. Dispensary personnel were trained to observe the best known practices in fitting eye protection.

Each pair of safety glasses was individually fitted as to eye size, bridge and temple length. After being fitted, the glasses were placed in a case and handed to the employee with these remarks: "These glasses have been personally fitted to you. They will be comfortable if you do not alter their adjustment. Don't let others wear them; and keep them in the case when you are not wearing them."

This same personal advice was given when all other personal protective equipment—particularly hats and respirators—was issued. Employees were asked to return to the Dispensary to have the equipment adjusted or to have worn or broken parts replaced. Strict instructions were given pertaining to the replacement of hat bands or respirator filters at specified intervals.

All equipment turned in to the Safety Dispensary for repairing or re-issue was completely dissembled, washed and sterilized. Worn parts were renewed. Much of the equipment was repainted. Because of this procedure we were able to salvage more than 90 per cent of all turned in equipment.

Renewed equipment was placed in a cellophane bag with a certificate stating that it had been sterilized according to standards approved by the California Industrial Accident Prevention Bureau and the U. S. Navy and Maritime Commission.

I wish to emphasize strongly the necessity for standardization of equipment used within any company. In order to do this a job analysis—in terms of the equipment that will be required for hazardous operations—must be made. When the equipment requirements of a company are known, types can be chosen that will give the greatest protection possible from the hazards involved.

One of the greatest problems faced by a safety director after the receipt of new equipment is to keep the employees wearing the old equipment, and to allocate new equipment on those applications for which it was originally purchased. It seems that everybody wants new equipment and they find a dozen reasons why the old equipment is unsatisfactory. Standardization will, for the most part, eliminate this problem.

For example, in our eye program, we used only three types of equipment: the spectacle, the spectacle with acetate side shields and nose guard, and the cup coverall goggle. All of our spectacle type goggles were either in 44 or 47 mm. eye size and were equipped with adjustable nose pads. We tried to keep away from the saddle bridge, leather side shield and 50 mm. types. Tinted lenses were permitted only on jobs where the analysts showed their need. We preferred the cup coverall to the regular cup goggle type because it not only afforded the same amount of protection but it did not differentiate between those who wore prescription glasses and those who did not. Further, the cup coverall type does not seem to fog as easily because of the larger air space. All of the above met or surpassed Federal specifications.

What has been said about eye protective equipment also can be said of respirators, safety hats, etc.

As a final step in our program we prepared a 20 page illustrated booklet, "Use and Care Of Personal Protective Equipment". Each type of equipment showed its use with a paragraph giving a general description of its recommended use for particular jobs as taken from our job analysis survey. The second illustration was of the equipment with a short paragraph on its care. The in-

side page of the front cover contained a message to all employees and supervisors from the Vice President of the company. One page was devoted to an explanation of how to obtain prescription safety glasses. On another page the functions of the safety department and the Safety Dispensary were defined relative to their respective personal protective equipment duties on the program.

This booklet was given to all supervisors and employees as a follow-up on our job analysis survey. It created a general acceptance for equipment through increased knowledge of its use and care.

In conclusion I want again to stress the importance of making a job analysis for personal protective equipment. The dispensary where personal eye protection equipment is issued should be of the same professional standard as that found in medical dispensaries.

After all is said and done the supervisory personnel and employees look upon personal protective equipment as the tangible tools of the safety department. If it is not properly dispensed with salesmanship and showmanship, the entire safety program may suffer.

After the above program had been in effect for a year, eye injuries had declined to less than 30 per cent and lost-time eye cases to less than one-half of one per cent.

---

## VISION TESTING AT WESTERN ELECTRIC, BALTIMORE PLANT[1]

H. R. KOZLOSKI
BALTIMORE, MD.

AN industrial vision testing program is being undertaken by the Company to determine the visual requirements of specific jobs. The program is in effect right now, and the purpose of it is to aid those employees whose visual skills are found to be below determined standards.

This measurement of visual skills will be performed through the use of an apparatus called an Ortho-Rater, a remarkable precision instrument.

In taking the test, the person being tested faces the instrument in the same manner as he faces his job. If he wears glasses to see the movies and baseball games and does not require them for close work, he is given the Far Test with his glasses on and the Near Test without them; if he wears his glasses only for reading and close work and does not wear them for distance seeing, he is given the Near Test with and the Far Test without his glasses; and if he wears his glasses for both distance and near seeing, he is tested with his glasses on for both tests.

Here is how the Ortho-Rater works. By means of a series of 12 tests, the instrument measures the balances of the eye muscles, the ability to distinguish depth and color, and the ability to perceive objects with both eyes and separately with each eye at two different distances, 13 inches and 26 feet—referred to as Near Tests and Far Tests, respectively. These distances are created optically within the dimensions of the instrument.

No tests have been included arbitrarily—all measure some aspect of vision which has been shown to relate to success and safety on the job.

Inasmuch as the Ortho-Rater was designed specifically for industrial use, no attempt will be made to diagnose eye difficulties or to write prescriptions for glasses. Each person being tested will be asked direct questions on each test as it is presented and the answers will be scored to record a performance. If the scoring falls below normal on any persons, they will be notified by the Hospital Department and referred for appropriate attention.

1. Published in "The Pointer", V. 15, p. 2, July 1946, Western Electric Co., Baltimore, Md.

## "IN REPLY TO SUN GLASS INQUIRIES"

August 9, 1946

Dr. Edwin P. Jordan
Journal of the American
Medical Association

Dear Dr. Jordan:

"I am in receipt of your letter, enclosing a question about the harmful or beneficial effects to the eyes of the sun glasses, sold in drug stores and dime stores throughout the country.

Many poorly fitting and cheap glasses cause ocular distress and headaches; a person who has such distress when he wears glasses naturally takes them off, just as a man takes off a pair of shoes that do not fit him comfortably.

The light from which we protect our eyes by "sun glasses' may be considered in four ways:

(a) Sunlight:

Very bright sunlight is distressing in varying degrees to all eyes. This is the reason why eyelids are kept closed, or nearly closed, when facing such a light.

(b) Ultraviolet:

Ultraviolet light, of the wave length of approximately 300 mu., such as is present in rarified atmosphere on high mountains or in dense snow fields, or such as comes from arc lights or welding machinery, etc., causes irritation and temporary inflammation of the superficial cornea and conjunctiva, coming on 8 to 24 hours after exposure, and lasting a day or more. Welders, for instance, are protected by special goggles.

(c) Infra-red:

Eclipse blindness is a burn of the macular area, a result of the infra-red rays, and comes on immediately and is permanent. Smoked glasses are used to prevent this in those who wish to look at an eclipse.

(d) Glare:

Almost all of us are made uncomfortable by "glare". We have yet to find, however, any pathologic lesion produced by "glare".

The average sun glasses, obtained in drug and dime stores, if of sufficiently dark color, are, as a rule, comforting to the eyes of the average wearer on an average bright day, on the water or on the beach where they are usually used. They reduce the total amount of light entering the eyes. The idea that certain makes of glass do not transmit the "harmful" ultraviolet light is mostly sales talk, as the ordinary white glass allows very little ultraviolet light to pass through. In fact, in order to obtain passage for ultraviolet through a window-pane, it has to be specially made for that purpose. A certain amount of ultraviolet light is beneficial to the eyes; it is for this reason we take vacations at the seashore and in the mountains, and in the winter send our patients to sunny climes.

A very interesting and thoroughly scientific investigation of the effect of various wave lengths of light was made in 1905 by Verhoeff and Bell, and published in the Proceedings of the American Academy of Arts and Sciences—51: No. 13, July, 1906.

In a recent book on Industrial Ophthalmology, by Kuhn, there are several pages devoted to the subject of protecting the eyes against various types of illumination.

This subject was brought up at the business meeting of the Section on Ophthalmology at the American Medical Association recently, in connection with an article that was published in Hygeia and reviewed in the Readers' Digest for July, and which article appeared in the Readers' Digest some 10 days before it appeared in Hygeia. A resolution was passed by the Section, instructing the delegate of the Section to investigate

the publication of such an unscientific article and take steps to prevent a recurrence.

In regard to the "serious consequences of wearing cheap sunglasses", a poll was recently taken at the American Medical Association convention. It was the opinion of the several members and former members of the American Board of Ophthalmology polled, that they "had never seen any serious disorder of the eyes produced by any glasses, no matter how poor they were, or how ill-fitting." One of the group thought that it possibly might occur, but in his 40 years in practice in a mountainous region he had not seen it.

Sincerely yours,
THOMAS D. ALLEN
Chicago, Ill.

---

**CONTINUING BIBLIOGRAPHY ON INDUSTRIAL EYE PROBLEMS**


American Medical Council on Physical Medicine. *Eye Discomfort Caused by Improperly Shielded Black Light Ultra Violet Lamps.* J.A.M.A., Vol. 131, p. 287, May 25, 1946.

Anon. *Conservation of Vision and Prevention of Blindness.* J. Indiana M.A., Vol. 39, No. 5, p. 232, May, 1946.

Anon. *Illinois Society's Industrial Program.* Sight Saving Review, pp. 111-113, Summer, 1945.

Anon. *Eye Injury: Occurrence of Accident within meaning of act; Calculation of award. Consolidated Coal Co. vs. Dill.* Alabama Supreme Court, May 9, 1946. Industrial Hygiene Digest, Vol. 10, p. 5, June, 1946.

Burch, Norval J. *Safe at Servel.* National Safety News, pp. 20-22, 100, 102, 104, January, 1946.

California Industrial Vision Courses. The Optometric Weekly, Vol. 35, No. 45, pp. 1247-1250, Dec. 1, 1944.

Carson, L. D. *Ocular Effects of Altitude Flying and Deep Sea Diving.* Arch. Ophthal., Vol. 33, pp. 173-176, March, 1945.

Dickson, R. M. *First Aid Treatment of Industrial Eye Injuries.* Brit. J. Ophth. Vol. 27, pp. 544-547, Dec. 1943.

Duke-Elder, Phyllis. *One Eye as Good as Two.* Hygeia, Dec., 1945.

Ficklin, J. B. *Safety in Chemical Laboratories.* Safety Engineering, Vol. 90, pp. 52-57, Nov., 1945.

Fisher, Jas. A. *Severe Laceration of Only Eye With Recovery of Useful Sight.* Am. J. Ophth., Vol. 28, pp. 101-105, Sept., 1945.

Freeman, E. *A Basis for Testing and Improving Vision in Industry.* J. Consulting Psychology, Vol. 9, pp. 35-444, Jan.-Feb., 1945.

General Motors. *Doctor In Industry, General Motors Dramatizes Industrial Medicine Through the Life Story of an Industrial Physician.* Indust. Med., Vol. 15, pp. 264-267, April, 1946.

Hardy, LeGrand H. and Rand, Gertrude. *Elementary Illumination for the Ophthalmologist.* Sight Saving Review, Vol. 5, No. 15, p. 30, Spring, 1945.

Hollaender, Alexander. *Effects of Ultraviolet Radiation.* Annual Review of Physiology. Vol. 8, pp. 1-16, May, 1946.

Lyle, Donald J. *Eye Manifestations of Head Injuries.* J. Indust. Hyg. & Toxicol., Vol. 26, No. 10, p. 37, Dec., 1944.

MacMillan, J. A. *Problems in X-Ray Localization of Foreign Bodies in and About the Eyes.* Canad. M.A.J., Vol. 52, pp. 173-175, Feb., 1945.

McCoy, David A. *Suggested Standing Orders and Procedures for Industrial First Aiders.* Ind. Med., Vol. 13, No. 9, pp. 689-692, Sept., 1944.

McGregor, Ian Stewart. *A Study of the Histopathological Changes in the Retina and Late Changes in the Visual Field in Acute Methyl Alcohol Poisoning.* Brit. J. Ophth., Vol. 27, pp. 523-543, Dec., 1943.

MASON, B. R. *Color Discrimination in Industry.* The Optometric Weekly, Vol. 35, No. 34, pp. 933-940, Sept. 28, 1944.

MERRILL, ELEANOR BROWN. *Prevention of Blindness on the Home Front.* The Sight Saving Review, Vol. 14, No. 1, pp. 3-10, Summer, 1944. N.S.P.B. Publ. 411.

NATIONAL SAFETY COUNCIL. *A Look at Eye Injuries.* National Safety Bulletin, Nov., 1944.

NATIONAL SAFETY COUNCIL. *Visual Aids in Safety.* National Safety News, Vol. 51, No. 3, p. 195, March, 1945.

PIERSE, DERMOT. *A Case of Removal of Nonmagnetic Foreign Body from the Ciliary Region.* Brit. J. Ophth., Vol. 27, pp. 550-551, Dec., 1943.

ROCCO, ALFREDO. *Intraocular Foreign Body.* J. Indust. Hyg. & Toxicol., Vol. 26, No. 10, p. 146, Dec., 1944.

SPOLYAR, L. W. *The Effect of Fluorescent Light on Vision.* Monthly Bulletin, Indiana State Board of Health, pp. 289-294, Dec., 1945.

SMYTHE, HENRY F., JR., AND CHARLES CARPENTER. *The Place of the Range Finding Test in the Industrial Toxicology Laboratory.* J. Indust. Hyg. & Toxicol., Vol. 26, No. 10, p. 339, Dec., 1944.

# News - - - Notes

### NATIONAL

At the 1946 annual meeting of the American Broncho-Esophagological Association, new officers were elected as follows: President, Robert M. Lukens, Philadelphia; Vice President Dr. Murdock Equen, Atlanta; Treasurer, Dr. Clyde A. Heatly, Rochester, N. Y.; Secretary, Dr. Paul H. Holinger, Chicago; Editor, Dr. Louis H. Clerf, Philadelphia. The Council consists of Dr. Carlos E. Pitkin, Dr. Robert L. Moorhead, Dr. C. L. Jackson, and Dr. Millard F. Arbuckle.

### ILLINOIS

The Chicago Ophthalmological Society held an opening meeting Thursday, October 17, at the Palmer House. The clinical part of the program was given by the Department of Ophthalmology of the University of Chicago. Following the business meeting, Dr. R. T. Paton (by invitation) of New York gave a movie demonstration on "Keratoplasty" and Dr. S. V. Abraham (by invitation) of Los Angeles spoke on "A New Classification of Strabismus."

The Seventeenth Semi-Annual Postgraduate Conference in Neuromuscular Anomalies of the Eyes will be held at The Children's Memorial Hospital, Chicago, Illinois, November 3-8, 1946, by Dr. George P. Guibor. Some of the topics for discussion are as follows: "Anatomy of the Extraocular Muscles—Practical Applications", "Examination Routine for Strabismus in Relation to Physiology—Supranuclear and Infranuclear Lesions", "Theories of Fusion", "Prisms and Other Modifications in Prescriptions for Glasses", "The Treatment of Neuromuscular Anomalies by Refraction, Bifocals and Prisms", and "Surgical Treatment of Strabismus."

### MASSACHUSETTS

The postgraduate teaching program at Harvard Medical School Department of Ophthalmology started October 1, 1946. Three courses are being offered: (1) a three month basic science course, (2) a one month course in clinical-pathological ophthalmology, (3) a one month course in the fundamentals of refraction.

### MONTANA

The forty-seventh semi-annual meeting of the Montana Academy of Oto-Ophthalmology was held in Great Falls just previous to the State Medical Meeting on July 17 and 18.

Dr. Kenneth Swan, Professor of Ophthalmology at the University of Oregon Medical School presented two papers with colored slides: "Infections of the Eye" and "Tumors of the Eye and Adnexa."

The minimum requirements for membership was made the same as those for the American Boards. The next meeting will be held in Lewistown in February, 1947.

### NEW YORK

The American Academy of Allergy will hold its annual convention at Hotel Pennsylvania, New York City, November 25-27 inclusive. All physicians interested in allergic problems are cordially invited to attend the sessions as guests of the Academy without payment of registration fee. The program has been arranged to cover a wide variety of conditions where allergic factors may be important. Papers will be presented dealing with the latest methods of diagnosis and treatment as well as the results of investigation and research. Advance copies of the program may be obtained by writing to the Chairman on Arrangements, Dr. Horace S. Baldwin, 136 East 64th Street, New York City, prior to November 10th.

The Section of Ophthalmology of the New York Academy of Medicine met Monday evening, October 21. The program consisted of an instruction hour during which Dr. Ludwig vonSallmann (by invitation) discussed "Controversial Points in Ocular Penicillin Therapy"; case reports, "Malignant Teratoma of the Orbit, 6½ Years Observation" by Dr. Albert V. Saradarian (by invitation), and "Lipin Dystrophy of the Cornea" by Dr. Samuel Frankel (by invitation); and a paper "Some Clinical Disturbances of the Ocular Motor System" presented by Dr. David G. Cogan (by invitation) of Boston. Discussion was by Dr. Harold W. Brown (by invitation).

# TRANSACTIONS

## AMERICAN ACADEMY
*of*
## OPHTHALMOLOGY
*and*
## OTOLARYNGOLOGY

Vol 51 No 2

**NOVEMBER - DECEMBER**
*1946*

Published bimonthly, February, April, June, August, October, and December, by the Douglas Printing Company, 109 North 18th Street, Omaha 2, Nebraska, for the American Academy of Ophthalmology and Otolaryngology. Editorial and business office, 100 First Avenue Building, Rochester, Minnesota. Subscription price $3.00 yearly to members, $6.00 yearly to non-members. Entered as Second-Class Matter November 28, 1938, at the Post Office at Omaha, Neb., under Act of March 8, 1879.

**ANNUAL MEETING OCTOBER 12-17, 1947, CHICAGO**

*Jackson Memorial Lecture*

# THE INFLUENCE OF HYPERSENSITIVITY ON ENDOGENOUS UVEAL DISEASE

ALAN C. WOODS, M.D.
BALTIMORE, MD.

IT is my privilege tonight to deliver the Jackson Memorial Lecture. I am deeply appreciative of the high honor. I knew Dr. Jackson only as one of the great stalwarts of ophthalmology He was one of the leaders of his day. To my mind he achieved his high position through the happy combination of many sterling qualities: a searching and analytical mind, a tireless and meticulous attention to detail,—but details were never allowed to obscure his prime objective,—a rugged honesty and above all a kindly love for his fellow man. Although it was never my good fortune to serve under him, or be trained directly by him, no man who received his training in the period I did could help but be influenced for the better by Dr. Jackson's example and teachings. I freely acknowledge the debt.

The subject I have chosen for this lecture may at first glance appear somewhat foreign to the subjects in which Dr. Jackson was preeminently interested. Yet I excuse myself this seeming digression by recollecting a remark that Dr. Jackson once made to me. It was some years ago at a medical convention in Texas, when I had delivered a lengthy and probably quite dull lecture on the relation of immunity in syphilis to syphilitic disease of the eye. After a perfunctory discussion had come to a close and the business had moved on to the next paper, Dr. Jackson sought me out and went out of his way to thank me for the paper. When I told him I was afraid it was a little foreign to the general interest of the audience, he told me frankly that he was well aware of that, but he believed it was a healthy step to present such subjects to ophthalmologists, and that he for one, trusted I would continue to do so. I have never forgotten his kindliness or the sincerity of his advice. So with this pleasant memory, I have less temerity than I would otherwise have in discussing the subject of the paper—The Influence of Hypersensitivity on Endogenous Ocular Disease.

Primarily there is an enormous confusion in the terminology of the hypersensitive reaction. This applies especially to the word "allergy". As used originally by von Pirquet it was defined as an altered reaction capacity of the organism. Its meaning was gradually extended by von Pirquet to include the general bodily changes that occur with advancing years—even those which might favor the development of malignant tumors. While in the minds of most medical men the term allergy is synonymous with acquired hypersensitiveness, it is used with a wide variety of meanings. Some authors limit its application to certain specific types of hypersensitivity, others use it to include all types of hypersensitiveness to foreign antigens, both bacterial or nonbacterial, and still others include in its

From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University.
Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.
Sponsored by the Ophthalmic Publishing Co., publishers of the American Journal of Ophthalmology.

meaning all forms of altered reaction capacity, including immunity. Rich has frankly stated that "the term 'allergy' has become so debauched by indiscriminate usage that it would be fortunate indeed if it could be dropped completely from the vocabulary of science." This confusion of terminology is reflected in the ophthalmological literature to such an extent that the terms hypersensitivity and allergy are used almost as scrap-baskets or cover-alls, being invoked to explain a variety of mysterious and often totally unrelated conditions for which there is no obvious explanation. Thus we reach the final absurdity where we find a uveitis of unknown etiology attributed to an assumed allergy to an undiscovered virus! Scylla piled upon Charybdis!

Yet despite this indiscriminate and unjustified use of specific terms, and the definite gaps which exist in our knowledge, there is nevertheless a great fund of information on the hypersensitivity reaction and a generally accepted terminology. This knowledge and the terminology has recently been set forth in an orderly manner by Rich,[7] and since I know of no clearer exposition, for our purposes here, we may follow his presentation.

### THE TYPES OF HYPERSENSITIVITY

Two definite forms of hypersensitivity are recognized—the anaphylactic state, and the hypersensitivity of infection. While there are many superficial resemblances between these two types, nevertheless there are differences so fundamental that they can be clearly differentiated one from the other.

### I. ANAPHYLAXIS

a) *The anaphylactic state* is a form of hypersensitivity which results when foreign protein enters the tissues. When this hypersensitivity is once established, if small amounts of the same foreign protein are brought into contact with the sensitized tissues, either by intracutaneous injection or a patch test, a localized urticarial weal develops. If the antigen enters the circulation in sufficient quantities, various anaphylactic manifestations occur which include urticaria, "serum sickness", complete collapse, and even death. These symptoms come on promptly after the second exposure to the foreign protein, and are caused by a spasmodic contraction of the involuntary smooth muscle and an increase in capillary permeability.

In addition to the local and general reactions, a focal reaction can occur in the anaphylactic type of hypersensitivity. Thus if the initial or sensitizing injection of the foreign protein is given directly in the eye or in the cornea, and the second "intoxicating" injection is given intravenously, a sudden sharp inflammatory reaction may take place in the injected eye. This is a form of the "ophthalmic reaction" produced by protein antigens. It is manifestly due to a local increased reaction capacity of the eye, although it is not fully established to what degree this is due to a local vascular reaction dependent on the increased capillarity induced by the primary injection.

The anaphylactic type of hypersensitivity is produced probably exclusively by proteins. When it is apparently dependent on such nonprotein substances as inorganic chemicals, drugs, carbohydrates and the like, the probable mechanism is a combination of the foreign nonprotein material with the native protein of the body to form a new protein compound which is foreign to the host. The anaphylactic manifestations result from an antigen-antibody reaction affecting primarily the fixed cells of the involuntary smooth muscle, the endothelium of the capillaries, and collagenous fibers. While the anaphylactic reaction is characterized by edema, increased capillary permeability, congestion, hemorrhage, and spasm of the smooth mus-

cle, it does not produce *per se* necrosis or death of the fixed cells. The anaphylactic type of hypersensitivity can be passively transferred, and is usually accompanied by the presence of specific precipitins in the body fluids.

b) *The Arthus phenomenon* is essentially an exaggerated local type of anaphylactic reaction. If foreign proteins, including the soluble portions of bacterial proteins, are introduced repeatedly and in small amounts into the tissues at intervals of several days instead of only once, an anaphylactic state will be produced. After sensitivity appears the injections of the protein at first call forth the usual urticarial weal which vanishes in a few hours. But as these injections are continued, finally the introduction of a small amount of the foreign protein will produce an enduring local reaction characterized by hemorrhage, necrosis and finally sloughing of the skin at the site of the reaction. In the case of bacterial protein, the Arthus type of hypersensitivity can be induced only by the injection of the soluble portions of the bacterial proteins, and when thus established, the phenomenon is elicited by the injection of the soluble bacterial proteins. While the Arthus phenomenon superficially resembles the reaction of the bacterial type of hypersensitivity, in that it may produce local necrosis, the fundamental mechanism is quite different. The local necrosis and death of the fixed cells in the Arthus phenomenon is not caused by a direct action on the extravascular tissue cells but is due to an interference of their blood supply by thrombosis and rupture of the nutrient vessels.

c) *The "Pollen" type* of hypersensitivity, which is produced not only by pollens but also by other protein agents, is quite similar to the usual anaphylactic type. It differs from it chiefly in its tendency especially to involve certain tissues, the bronchioles, nasal and conjunctival mucous membranes, and in the fact that although the antibody is demonstrated in the serum by passive transfer its specific precipitins are ordinarily not demonstrable in the serum. In other respects—the immediate reaction, urticaria, erythema, smooth muscle spasms, etc., it is indistinguishable from the anaphylactic reaction. Since the differences are not fundamental it may be regarded as a form of anaphylactic hypersensitivity.

## II. THE HYPERSENSITIVITY OF INFECTION—BACTERIAL HYPERSENSITIVITY—THE TUBERCULIN TYPE OF HYPERSENSITIVITY

These terms are all synonomous, and are used to describe a type of hypersensitivity quite different from the anaphylactic type in its various characteristics. This type of hypersensitivity can be readily induced by parenteral contact of the tissues with the living or dead bacteria, fungi or filterable viruses, but not by any soluble antigens so far extracted from micro-organisms. However, once it is established by contact with the bacterial bodies, reactions may be elicited by injection of the soluble bacterial proteins. The bacterial type of hypersensitivity is not characterized by the appearance of precipitins in the blood serum and it cannot be passively transferred. The reactions elicited in the hypersensitive patient or experimental animal are (a) a local reaction, and (b) if the specific antigen is introduced in sufficient quantity, a systemic and (c) a focal reaction.

a) *The local reaction.* When the bacteria causing this hypersensitive state, or their soluble products, are introduced in the hypersensitive tissues there is no immediate reaction, but a delayed reaction which comes on after some hours and reaches its maximum in 24-48 hours. The local reaction is characterized by erythema and edema, and in its extreme form by local tissue necrosis

and sloughing. The cells of the body with the bacterial type of reaction are damaged by the contact of the antigen with the cell body, both *in vitro* and *in vivo*, and if the antigen-antibody reaction is sufficiently intense, the cells may be actually killed.

b) *The systemic reaction.* If the antigen is introduced in sufficient quantity, there may occur a severe systemic reaction, but unlike the anaphylactic reaction, this reaction is delayed and comes on gradually after some hours. In its mild form the systemic reaction is characterized by malaise, joint pains, fever, etc. In its severe form death may ensue. The mechanism of this delayed reaction is not understood. It is variously believed that the antigen-antibody reaction may produce a toxin which damages the cells or that the cells undergo some bodily alteration which makes them capable of exerting an enzyme-like activity on the antigen with the liberation of an irritant substance.

c) *The focal reaction.* In addition to the local and systemic reactions, a "focal" reaction may be induced if sufficient quantities of antigen are introduced. This focal reaction consists in the lighting up of any remote focus of infection caused by the the specific infecting agent. Again it is a delayed reaction, coming on only after some hours.

The bacterial type of hypersensitivity differs from the anaphylactic type, therefore, in the fact that it can be produced only by introduction of the whole bacteria, and not by soluble protein, and therefore usually follows infection and vaccination with the bacterial body. The reaction to later introduction of the antigen is delayed and is accompanied by local inflammation, and often necrosis. There may occur also systemic and focal reactions. The site of the reaction is not limited to the involuntary smooth muscle, capillary endothelium and collagenous fibers, but is a widespread reaction in the sensitized cells of the body, and is associated with no demonstrable excess of free antibody in the circulation. The antigen-antibody reaction acts directly on the cells, causes cellular damage, and may cause death of the cells.

While anaphylactic and bacterial hypersensitivity are fundamentally different and in the main tend to be mutually exclusive, they can, nevertheless, occur together in the same individual or animal. Thus after bacterial infection the infected body may develop not only the usual bacterial type of hypersensitivity from the protein of the bacterial body, but may also develop the anaphylactic type of sensitivity to the soluble products of the bacteria. For example, in pneumococcal infections the bacterial type of hypersensitivity to the pneumococcal protein may develop, and also an anaphylactic type of hypersensitivity to the specific polysaccharide of the pneumococcus. Likewise in tuberculosis, while the injection of tuberculin or tuberculoprotein will not induce the tuberculin type of hypersensitivity, under certain conditions an anaphylactic type of sensitivity to tuberculin may develop.

## III. IMMUNITY

There remains one last term to define —"immunity". In general, immunity may be defined as an increased resistance to infection. This increased resistence may be either native to the organism or acquired by infection or inoculation. There are two main recognized phases to the phenomenon of immunity. The first is a humoral factor, due to the antibodies circulating in the blood stream and tissue fluids, which tend to fix, immobilize and opsonize the bacteria, and in some infections may provoke extracellular destruction. The second is a cellular phase, centered chiefly in the mononuclear and phagocytic cells, which engulf the opsonized bacteria and either inhibit their prolif-

eration or destroy them. In most infections the important role of antibodies and phagocytes is readily demonstrable. In tuberculosis the role of the circulating antibody is not as well established as in various other infections, although it probably plays an important part.

The essential basic manifestations of acquired resistance are inhibition of the spread of bacteria and the suppression of their proliferation. In acute infections this is accompanied by an actual destruction of the bacteria. In tuberculosis, however while many bacilli are destroyed by the forces of acquired resistance, bacteriostasis with fixation and immobilization of the bacteria is prominent. Thus the bacilli may long remain viable, ready to proliferate and spread when the forces of resistance which hold them in check diminish and wane.

There has been much confusion on the relationship of allergy and immunity. For example, in tuberculosis, and indeed in many other infections, for years it was generally accepted that allergy, (i.e. a hypersensitive state) was responsible for the immunity (i.e. acquired resistance)—that the hypersensitive reaction inhibited the spread of the invading specific bacteria and suppressed their proliferation. This idea was challenged by Rich and his coworkers in 1929.[8] In a long series of experiments by these and other investigators, it was clearly demonstrated that acquired resistance is in no way dependent on hypersensitivity. There is no actual correlation between the two. Hypersensitive inflammation itself is incapable of preventing the spread of invading bacteria in the absence of acquired resistance. Effective resistance can be established in the complete absence of any hypersensitive reaction.

Allergy (acquired hypersensitivity and immunity (acquired resistance) should therefore be regarded as distinct phenomena. It should, however, be born in mind that as a result of infection or exposure to specific bacteria, both allergy and immunity tend to develop in the infected body. For example, both in clinical and experimental tuberculosis, the infected body usually develops a well marked hypersensitivity to tuberculoprotein, while at the same time it shows a marked resistance to reinfection. This same holds true in other infections. The two phenomena, hypersensitivity and resistance, are however, independent one of the other.

So much then for a brief summary of the clinical characteristics and fundamental differences of the two recognized types of hypersensitivity, a definition of acquired resistance or immunity, a word as to its mechanism, and its independence of any hypersensitive reaction. The main question before us is in what way the different types of hypersensitivity, both of which may follow infection, may influence endogenous ocular disease, how such influences may be suspected and detected, and how they may direct our therapeutic attack on the ocular disease.

## TYPES OF ENDOGENOUS UVEITIS

Endogenous uveitis may conveniently be divided into three different groups: (1) sympathetic ophthalmia and the so-called endophthalmitis phaco-anaphylactica; (2) the various metastatic purulent infections of the eye which arise from direct infection by blood-born bacteria; (3) the usual nonpurulant uveitis, iritis, or choroiditis, which so often is recurrent, and comprises the greater percentage of all uveal disease.

While there is considerable evidence that a hypersensitive reaction is of paramount importance in both sympathetic ophthalmia and endophthalmitis phaco-anaphylactica, the hypersensitivity in these diseases is dependent on the organ-specific proteins of the eye itself. This is a separate problem which has been amply discussed elsewhere, and is

not germane to the present subject. Likewise, the various forms of metastatic ophthalmia, endophthalmitis, choroiditis septica, and panophthalmitis, which result from the direct infection of the eye by bacteria carried by the blood stream do not concern us in this discussion. In such direct infection with the usual pathogenic organisms the process either becomes localized and walled-off with varying amounts of damage to the eye, or the infection spreads through the eye with the picture of a purulent ophthalmia. We are here concerned with the acute, the recurrent, and the chronic uveitis which constitute the usual clinical picture.

The older writers divided disease of the anterior uvea into the "serous" and "plastic" iritis. To my mind a much better terminology is "nongranulomatous" for these terms give an indication of the underlying pathologic process. In some instances it may be difficult or even impossible to make this differentiation, but as a rule the clinical pictures as well as the underlying pathology of these two forms of inflammation are quite different. The salient characteristics of these forms of uveitis are as follows:

*Nongranulomatous uveitis.* In the anterior uvea the onset is usually acute, rather than insidious, and ciliary congestion is marked with pronounced photophobia and lacrimation. The inflammatory reaction in the iris is usually slight and is limited to loss of luster, blurring of the iris pattern and dilatation of the capillaries. There are no nodules and but little tendency to the formation of posterior synechia unless there are repeated recurrences. The aqueous ray is usually intense due to the outpouring of serum and there may even be a heavy gelatinous or fibrinous exudate in the anterior chamber. The deposits on the posterior surface of the cornea are small and pin-point, and are composed chiefly of lymphocytes. Heavy, greasy exudates do not occur, and there is little or no tendency to capsular clouding of the lens. Koeppe nodules are never observed. The course of a nongranulomatous iritis is usually short and the eyes usually recover with amazingly few residua. Only after repeated attacks is organic damage done to the eye.

A nongranulomatous inflammation in the posterior uvea is not so typical and the diagnosis may be difficult or impossible. The general characteristics of a nongranulomatous choroiditis are slight clouding in the vitreous, fine muscae, wide spread and intense subretinal edema, blurring of the neuroretinal outlines, obscuration of the physiological cup, overfilling of the retinal venous bed, and the absence of manifest or well-defined choroidal exudates. There is little tendency to actual tissue destruction and secondary gliosis.

*Granulomatous uveitis.* In the anterior uvea, the onset of the granulomatous iritis is usually insidious. The cellular reaction in the tissues is greater than the vascular reaction, and the ciliary congestion is not usually severe. Organic changes take place in the iris with thickening of the stroma from cellular infiltration with blurring of the iris pattern and loss of the normal luster. Well defined nodules or tubercles on the surface of the iris, or diffuse localized thickenings suggesting nodules deep in the iris stroma, are sometimes present. There is a marked tendency to the formation of posterior synechiae, with greasy exudates on the anterior capsule of the lens and capsular clouding. The keratitic precipitates are of the so-called "mutton-fat" variety, and histologically consist chiefly of epithelioid cells. Koeppe nodules, or accumulations of epithelioid cells at the pupillary border of the iris, are not uncommon. The aqueous ray may be intense, but more often is rather mild.

In the posterior uvea, the granulomatous type of inflammation is usually marked by heavy veils and exudates in the vitreous. Manifest visible exudates in the choroid are the rule and they quickly involve the overlying retina. The subretinal edema may be diffuse, but it is usually localized and limited to the portion of the fundus adjacent to the exudates. The exudates are at first ill-defined. In some cases they spread and tend to involve the entire fundus with necrosis and destruction of the choroid and retina with later secondary gliosis. In other instances, they become quickly circumscribed and limited. In either case, the essential underlying pathologic process is tissue destruction with secondary overgrowth of glial tissue. When the process is localized, as in the well known "circumscribed plastic choroiditis", recurrences are common, and usually take place contiguous or at the site of the earlier lesion.

In the main nongranulomatous and granulomatous uveitis tend to be mutually exclusive, the clinical picture usually following one or the other pattern. A few cases are encountered in which differentiation is difficult, characteristics of both types appear present in the same eye—acute inflammation followed by granulamatous changes. In these cases one has the impression that both processes are present together.

### ETIOLOGY OF NONGRANULOMATOUS AND GRANULOMATOUS UVEITIS

There is a definite conflict of opinion on the etiology of endogenous uveitis. This is not remarkable since there are no fluids readily available for culture or animal inoculation, and no tissue for biopsy. The etiologic diagnosis is made on clinical observation, correlation of the clinical picture with the accumulated material from similar eyes, and the general study of the patient. As a result, the literature reflects the individual clinical acumen and knowledge of clinical-histologic correlation of the various reporters and their resources for medical study of the patient. While most authors agree the various forms of uveal inflammation are infective in nature, they admit the clinical picture is quite different from the purulent reactions usually induced by bacterial metastasis. It is assumed that in some unknown way, either by passage through the body or by the local environment in the eye, the usual action of the infecting bacteria has become modified and an atypical picture results.

A great number of ophthalmologists believe that remote foci of infection are the most important cause of uveal disease, the disease in the eye resulting either from direct bacterial metastasis, or by toxins liberated from the focus of infection. While syphilis, tuberculosis, and other systemic diseases are recognized as causes of endogenous uveitis, they are considered less important. Another group of ophthalmologists place chief emphasis on tuberculosis and the other so-called infectious granulomata as etiologic factors. Still others express a middle-of-the-road opinion. There is little differentiation in the literature between the etiology of nongranulomatous and granulomatous uveitis. In fact, these terms are not in common usage. In general however, the type of uveitis described here as nongranulomatous, is usually believed to be infective in nature, caused in one way or another by the usual common pathogenic bacteria. The usual recognized causes for granulomatous disease are syphilis, tuberculosis, brucellosis, sarcoid, lymphogranuloma venereum and certain fungus infections. It is quite probable other etiologic agents, viruses and unrecognized bacterial infections, may produce a similar pathologic picture—for in an appreciable percentage of granulomatous uveitis, the most thorough medical survey reveals no recognizable cause.

In recent years it has become fashionable to speak continually of allergy as influencing, and actually causing endogenous uveitis, although rarely if ever does there appear to be any clear concept of the type of hypersensitive reaction involved, or the mechanism thereof. However, in many cases there appears to be a close correlation between the anaphylactic state, the hypersensitivity of bacterial infection, and the various manifestations of uveitis. It is not my intention to repeat here any of the statistical or experimental studies already published on the etiology of uveitis. Rather I would present to you my overall impression on the influence of the different types of the hypersensitivity on endogenous uveitis, the methods used for the detection of the hypersensitive state, and what therapeutic measures may be used to combat it.

## I. NONGRANULOMATOUS UVEITIS

The clearest-cut examples of nongranulomatous uveitis are the recurrent iris inflammations found in association with rheumatoid arthritis, and old gonococcal infections. A similar acute iritis is sometimes found following acute systemic infections, and is occasionally found in association with remote foci of infection, no other obvious cause being apparent. The ocular picture in these conditions follows the same general pattern of nongranulomatous iritis already described. The course is sometimes so stormy that an inexperienced ophthalmologist might almost despair of saving the eye, but the disease finally subsides in a relatively short time with practically no organic damage to the eye. This type of iritis is remarkably resistant to ordinary forms of treatment. Certainly in my experience neither the sulfa drugs nor penicillin appears to influence the course of the disease, although some form of heat therapy, either nonspecific protein, induced hyperprexia, or prolonged local diathermy, appears of value.

Is this type of iritis due to the actual presence of living bacteria in the tissues of the eye? My contention is that in the recurrent nongranulomatous iritis associated with rheumatoid arthritis, old gonococcal infection, and sometimes with focal infection, there is no evidence whatsoever of living bacteria in the eyes. All the evidence, both clinical and experimental is against such a supposition, and points strongly to a hypersensitive reaction being the basic cause.

### THE INFLUENCE OF HYPERSENSITIVITY IN NONGRANULOMATOUS UVEITIS

Various authors, (Kolmer,[4] Berens, Rothbard and Angevine and others[1]) have commented on the routinely negative blood cultures obtained in patients with acute iritis. It is true, however, that these cultures have been made after the outbreak of the iritis and it is still conceivable that cultures immediately prior to the onset of the iritis might have been positive. However, such specimens of the aqueous as have been examined are sterile on culture and bacteriologic examination. In my experience, not only have the blood and aqueous cultures been sterile, but animal inoculations of the aqueous, either intraperitoneal in guinea pigs, or in the anterior chamber of a rabbit's eye, have all been negative.

Experimental investigations also argue against the actual presence of living bacteria in the eye and are in favor of a hypersensitive reaction being responsible for nongranulomatous iritis. Inoculation of the eye, either by direct injection in the aqueous or by intracarotid injection of various strains of streptococci, pneumococci, and such organisms, almost invariably results in a purulent ophthalmia which may progress up to destruction of the eye, accord-

ing to the virulence of the invading organism. However, if the animals are sensitized to streptococci by repeated intracutaneous injections of living organisms, or by an infected agar implant, and the organisms are later brought into contact with the eye, a nonpurulent inflammatory reaction is produced. This was first described by Derick and Swift in 1929[3] as "the ophthalmic reaction". These authors produced the ocular inflammation in sensitized animals by corneal scarification, and instillation of the organisms in the conjunctival sac. This procedure has been amplified and modified by others. It has been clearly shown that a nonpurulent iritis may be produced in animals by various forms of systemic or local sensitization and intoxication by intravenous injection of the antigen. Specific attention has been called to the fact that the iritis so produced corresponds clinically to that observed in humans. The picture is ciliary congestion, dilatation of the vessels of the iris, contraction of the pupil, and even small exudates in the anterior chamber. The course is short and acute. The inflammatory process lasts several days only and then subsides without organic residua. Histologic examination of such eyes shows cellular infiltration of the iris, chiefly by lymphocytes and plasma cells (Brown[2]). If foreign proteins, such as heterologous serums are used as antigens the ocular reaction is prompt. If bacterial antigens are used, the ocular reaction is usually delayed.

The most frequent positive findings in patients with nongranulomatous uveitis are evidences of rheumatoid arthritis, an old gonococcal infection with a positive gonococcus complement-fixation reaction, a small active focus of infection, or a history of a recent acute infection of some type. Syphilis, tuberculosis, or other infectious granulomata are conspicuously absent. Positive reactions for bacterial hypersensitivity are the rule in these patients. The usual diagnostic procedures to detect such bacterial hypersensitivity are the intracutaneous injections of a 1:100 dilution of a killed 24-48 hour culture of various common organisms: alpha streptococci, beta streptococci, nonhaemolytic streptococci, H. influenza, gonococci and finally a 1:100 dilution of mixed streptococci. The tests are read at the end of 15 minutes for an immediate anaphylactic type reaction to the soluble bacterial products, and again at 48 hours for the delayed tuberculin-like reactions to the protein of the bacterial body. The delayed tuberculin-like reaction is almost constantly present, although frequently, especially in the case with an acute fibrinous or gelatinous exudate in the anterior chamber, an immediate reaction to the soluble bacterial products is also present.

A word of caution must be given in the interpretation of the cutaneous reactions to bacterial antigens. The bacteria concerned are all common organisms to which the average individual has been constantly exposed for years, and reactivity to the intracutaneous inoculation of these organisms may occur in normal individuals and may be especially marked after any acute infection. Before any diagnostic significance is attached to positive reactions it should be established *first*, that no other obvious cause can be found to which the uveitis is attributed; *second*, that the patient shows an especial and unexplained hypersensitivity to specific organisms, and not to all the bacteria tested; and *third*, that the specific reactions shown are greater than are found in normal control individuals. With these reservations, in my experience, positive cutaneous reactions to such bacterial antigens are of diagnostic significance in nongranulomatous uveitis.

If the clinical nongranulomatous iritis under discussion is due to bacterial hypersensitivity, immediately it may be asked what is the mechanism whereby

the hypersensitive state is produced in the eyes and how does intoxication occur? One can only surmise the mechanism through experimental analogy. Primarily, since the reaction is usually limited to the eye and only rarely accompanied by an urticaria or any pronounced general symptoms, one must assume an increased reaction capacity of the uveal tract. It has been clearly shown that such a local increased reaction capacity can be evoked experimentally by direct innoculation of the eye with the antigen. Therefore, to explain the clinical picture, it may be assumed that at some time the sensitizing bacteria have directly reached the eye. This may occur during the course of any transient acute general infection, from a remote infected focus or by absorption from an infected or even a normal mucous membrane or cutaneous surface. The infecting dose of organism must be so small or of such low grade virulence that the invading organisms are readily killed off by the bacteriocidal action of the ocular fluids, otherwise a purulent ophthalmia would result. If there is a repetition of such a bacteremia or later absorption either of the soluble products or of the bacterial body, when such antigens reach the eye they encounter sensitized tissue and produce a hypersensitive reaction. This is the mechanism in the experimental animal, and from what is known of the frequency of transient bacteremias and infected mucous membrane and cutaneous surfaces in man, it is a plausible explanation for the clinical phenomenon. Occasionally the intoxicating bacterial protein may reach the eye from an exogenous source in such concentration that it may evoke symptoms in an eye not especially hypersensitive, but only participating in the general tissue hypersensitivity. This is the case in the experimental ophthalmic reaction when the eyes are sensitized as part of the general tissue reaction, and intoxication is produced by local trauma of the eye and contact with the specific antigen. An analogous picture may occasionally occur clinically. Such a clinical phenomenon appeared to be the case in a physician whose eye was accidentally exogenously infected with streptococci. Two days later he developed an acute nongranulomatous iritis. He was found to have a high degree of hypersensitivity to the specific invading streptococcus, and no other cause for the iritis could be found.

Is the hypersensitivity responsible for such a nongranulomatous iritis of the immediate anaphylactic type or of the delayed tuberculin type? In humans there is obviously no way of determining the time relation of the antigen entering the eye to the beginning of symptoms. In experimental animals the evidence indicates the reaction is usually of the tuberculin type if bacterial antigens are employed. Thus Derick and Swift clearly showed that the ocular and general sensitivity produced by streptococcus antigens was of the delayed type, and the ophthalmic reaction came on 48 hours after the corneal scarification. Brown reported that in his sensitized animals the first signs of iritis produced by intravenous injection of bacterial antigens were observed only after 5 hours. In MacLean's experiments, where the iritis was produced by intravenous intoxication, the reaction came on from 24 to 48 hours after the later intravenous injection. However, if foreign serums are used as the sensitizing antigens, the anaphylactic type of hypersensitivity results. This was first demonstrated by Kümmel in 1910[6] who found iridocyclitis with exudation in the anterior chamber 6 hours after intravenous injection of the foreign serum in animals previously sensitized by intraocular injection. However, this iritis was so marked at the end of six hours that it is obvious the first symptoms must have occurred much earlier.

Such immediate reactions have been found by various other investigators when foreign serums were used. (Wessely,[11] Krusius,[5] etc.) An anaphylactic type reaction in the eye was also shown by an experiment I did in 1916[12]. In this experiment the eyes of dogs sensitized by systemic injection of horse serum were later perfused with defibrinated blood. When horse serum was added to the perfusion fluid, an immediate contraction of the pupil occurred together with conjunctival and pericorneal congestion, and the occurrence of petechial hemorrhages throughout the fundus. This appeared to be an example of the spasmodic contraction of sensitized smooth muscle and increased capillary permeability produced in eyes with the anaphylactic type of hypersensitivity on contact with the specific antigen. It is apparent, therefore, that in experimental animals either the delayed bacterial type or the immediate anaphylactic type of hypersensitivity may be produced in the eye according to the type of sensitizing antigen employed. When patients are tested with bacterial antigens the delayed reaction is usually found, although frequently, especially in patients with acute exudation in the anterior chamber, there may also be an immediate reaction to the soluble bacterial products. It is a plausible hypothesis that such hypersensitivity to the soluble bacterial products may be responsible for the acute edematous reaction. It has already been noted that the initial attacks of nongranulomatous iritis rarely produce much appreciable damage to the eye, but after repeated attacks, damage and tissue destruction often occur. These later organic changes are probably the result of repeated minor insults caused by an antigen-antibody reaction on fixed cells with the bacterial type of hypersensitivity. However, it is an interesting speculation that in patients who also show an associated anaphylactic type of hypersensitivity the final damage done by repeated attacks may in part be a true Arthus phenomenon in the eye, i.e., tissue destruction and secondary connective tissue changes due to interference with the blood supply by thrombosis or rupture of the nutrient vessels.

This concept of nongranulomatous uveitis as a bacterial hypersensitivity phenomenon obviously suggests desensitization as a therapeutic procedure. Such desensitization should be as specific as possible. In the case of streptococci there are now some forty odd antigenically different strains known. The specific strains responsible for the hypersensitive state should be determined, if possible. This is done by individual cutaneous testing with all available strains. Many laboratories, especially those interested in vaccine therapy for rheumatoid arthritis, have a large number, although usually not all, of the known strains on hand, which may be used for the determination of specific sensitivity. After the demonstration of cutaneous hypersensitivity to stock or mixed strains of streptococci, the patient should be tested against all individual strains obtainable, the final vaccine should be prepared from the specific strain or strains to which the patient shows the most pronounced sensitivity, rather than use a stock or mixed strain which may, however, be antigenically related.

The route of administration of streptococcus vaccine appears of some importance. It has been shown that the subcutaneous administration of streptococcus vaccine tends to increase and maintain the sensitivity rather than diminish it, while the converse is true when the vaccine is administered intravenously. Wainwright[9,10] has utilized this principle in the treatment of rheumatoid arthritis with streptococcus vaccine, giving the vaccine intravenously. This idea has been followed in the streptococcus vaccine therapy of the recur-

rent iritis associated with rheumatoid arthritis. The initial dose of the vaccine must be small and cautiously given, otherwise focal reactions will be produced in the eye. The results of such treatments have been gratifying. There are now in records of the Wilmer Institute a number of patients, all with the history of recurrent nongranulomatous iritis associated with rheumatoid arthritis, who have been under intravenous streptococcus vaccine therapy for periods up to seven years. The general result is the same—diminishing frequency and then freedom from attacks during the period of treatment. When treatment is stopped, however, recurrences in two to four months are not infrequent. Synchronous with the iridorelapse there is usually a recurrence of the cutaneous sensitivity which had previously diminished or disappeared under the vaccine therapy. In several of these patients continual weekly injections of the streptococcus vaccine appears to be the price of freedom from the attacks of iritis.

My only other experience with vaccine therapy for nongranulomatous iritis is with recurrent gonococcal iritis accompanied with a positive history of infection, a positive complement fixation test with gonococcal antigens, and a positive skin test to gonococcus vaccine. Here the vaccine has usually been given subcutaneously. The majority of patients so treated appear to be benefited in the sense that the attacks occur at much longer intervals and are much less severe. The results, however, have not been as striking as those obtained with the streptococcus vaccine in iritis associated with rheumatoid arthritis.

*Choroiditis.* By far the great majority of cases of choroiditis belong to the granulomatous type, characterized by exudation, tissue destruction and secondary gliosis. While granulomatous disease appears equally frequent in the anterior and posterior uvea, nongranulomatous disease has an undoubted predilection for the anterior uvea. Why this should be so is not clear. Occasionally cases of choroiditis are encountered when the salient symptoms are marked subretinal edema, and an absence of visible exudates and tissue destruction. These cases usually run a short course and are not followed by secondary gliosis. I have seen several such cases which were clearly focal reactions following the diagnostic injection of injudicious amounts of tuberculin. I have not seen such cases in which a bacterial hypersensitivity to other organisms was demonstrated. If such cases occur, they are certainly rare. All that can be said, following the arguments presented for a hypersensitive nongranulomatous iritis, is that such a nongranulomatous choroiditis from bacterial hypersensitivity is a possible clinical entity, and careful observation may establish such a mechanism.

### II. GRANULOMATOUS UVEITIS

Granulomatous disease affects both the iris, choroid and often the entire uveal tract. It may therefore be discussed as a single entity.

The evidence indicates that in granulomatous uveitis the various recognized specific etiologic agents are present in living form in the uveal tract. In syphilis this is certainly true. In tuberculosis it is likewise almost certainly true. In experimental ocular tuberculosis, practically all the various clinical manifestations can be produced by inoculation of the eyes of properly prepared animals, and living bacilli can usually be demonstrated in such eyes long after all symptoms have disappeared. In the occasional spontaneous uveitis that occurs in animals systemically infected with tuberculosis, the bacilli can be demonstrated in the eye either by bacterial stains or by animal inoculation of the diseased eye. In some forms of clinical ocular tuberculosis, the ba-

cilli can frequently be demonstrated by bacterial stains of sectioned eyes, and the inability sometimes to find them can probably be explained either on technical grounds or on the chronicity of the disease with final healing and consequent destruction of the bacteria. In lymphogranuloma venereum the living virus is almost undoubtedly present in the ocular lesions, for in the characteristic oculoglandular conjunctivitis that frequently complicates the general infection, the virus has been recovered from the conjunctival scrapings. In brucellosis, the bacteriologic evidence at hand indicates the living organisms are probably present in the ocular lesions, although their presence has not yet been conclusively demonstrated beyond shadow of doubt. Nothing can be said of sarcoid, for the specific causative agent, whatever it may be, has never been isolated and the etiology of the disease is a complete mystery. Nothing can be said of other possible etiological agents until they are identified.

### INFLUENCE OF HYPERSENSITIVITY IN GRANULOMATOUS UVEITIS

In syphilis a great deal is known of the influence of specific immunity on the occurrence of syphilitic lesions. The effect of such immunity on syphilitic lesions of the eye has been discussed elsewhere, and need not be repeated here. Little or nothing is actually known of the effect of specific hypersensitivity on syphilitic lesions. The reason for this is that no antigen or syphilitic material has ever been discovered with which specific hypersensitivity can be effectively demonstrated. Any hypotheses on the subject are therefore only by analogy and are little more than idle surmise. In brucellosis, the general experimental study of the disease, and of ocular brucellosis in particular, has not yet been sufficiently extended to warrant any conclusions on the influence of general or local hypersensitivity on the course of the lesions. What evidence there is indicates both immunity and hypersensitivity may profoundly affect the course of the general and local disease. There is little or no clinical or experimental knowledge of the effect of hypersensitivity and immunity in the uveitis produced by viruses and fungi. What we do understand, with reasonable clarity and certainty, is the influence of both hypersensitivity and immunity on the lesions caused by the tubercle bacillus. In this regard, both the general lesions of the disease, and the lesions of ocular tuberculosis follow the same pattern. We may therefore use tuberculosis as an example to illustrate the effect of hypersensitivity and immunity in granulomatous uveitis.

The instillation of tuberculin in an already tuberculous eye may at times evoke a crop of phylectenules, and the systemic injection of excessive amounts of tuberculin may likewise cause a focal inflammatory reaction in a tuberculous eye. These are true hypersensitive reactions, they are dependent on the previous sensitization of the eye by invasion of the eye by the bacilli, and are an example of the delayed reaction produced by the soluble portion of the tubercle bacilli when the eye has been previously sensitized by the protein of the bacterial body. Tuberculous lesions may also be produced by the injection of killed tubercle bacilli, and even by their fractions, tuberculolipides and especially tuberculophosphatide. But these lesions are nonprogressive, even in the nonimmune animal, and their extent is directly proportional to the number of bacilli or the quantity of lipides injected. Such lesions do not concern us clinically. The point to be emphasized is that true progressive tuberculous lesions with cellular infiltration of lymphocytes and epitheloid cells, tubercle formation, and caseation or encapsulation are produced only by the actual invasion of the eye

with tubercle bacilli and the presence of the bacilli in the eye in living form. The course of the lesions resulting from such bacterial invasion is profoundly modified by the factors of immunity and tissue hypersensitivity, but hypersensitivity to tuberculoprotein *per se* does not produce characteristic tuberculous lesions, notwithstanding the innumerable statements to the contrary with which the ophthalmologic literature fairly bristles. It is therefore incorrect to speak of "allergic tuberculous lesions", or to say certain specific ocular lesions (excluding phylectenules and focal reactions) are "caused by allergy". The lesions are caused by invasion of the ocular tissues by the tubercle bacilli. The course and character of the lesions is modified and influenced by the factors of immunity and tissue hypersensitivity.

In what manner do hypersensitivity and immunity modify and influence the course of tuberculous lesions? This can best be illustrated by following the course of ocular tuberculosis in experimental animals, for here the picture simulates the clinical disease with amazing faithfulness.

If a normal nontuberculous rabbit is innoculated in the anterior chamber with virulent human tubercle bacilli, there is practically no immediate reaction. With the usual dose of organisms, in about two weeks small hard tubercles develop on the iris, in the cornea, and in the ciliary body. There is only a minimum inflammatory reaction. As the bacilli propagate and the tuberculous lesions multiply, the picture gradually changes. In about four to six weeks after inoculation the inflammatory reaction increases, evidences of caseation appear with necrosis of the involved cornea and sclera, and somewhere about the twelfth week the eyes usually perforate. If sample rabbits from such an experiment are tested from time to time for sensitivity of the eye to tuberculoprotein, it is found that the development of acute inflammation, caseation and necrosis parallels the development of the hypersensitivity to the tuberculoprotein.

When the eyes of rabbits which have had a previous systemic tuberculosis (immune-allergic animals) are similarly inoculated, the picture is quite different. As a result of the old systemic infection such rabbits have developed a generalized hypersensitivity of all the body tissues, including the eyes, to tuberculin. At the same time they have developed a greatly increased resistance to reinfection, although there is no relation between the hypersensitivity and the immunity. This resistance to reinfection may be so great that it requires many times the dose given normal rabbits to produce ocular lesions. When the necessary dose of bacilli is injected in the anterior chambers of these rabbits, an acute inflammatory reaction develops in 24-48 hours which lasts for several days. This is the local reaction to the tuberculin in the inoculum and is dependent on the ocular hypersensitivity which has occurred as part of the general sensitization. This reaction subsides within a week and thereafter there is a latent period of several weeks without symptoms. Hard tubercles then slowly develop in the iris, cornea and ciliary body. However, these lesions do not increase in size or progress to the extent they do in the normal rabbit. The inflammatory reaction is usually of low degree, there is very little caseation and necrosis, and these eyes rarely if ever perforate. The entire course of the local disease is restrained, and after three to four months the process burns out with moderate scarring and damage to the eye. If the eyes of sample rabbits from a series of such animals are examined for local reactivity to tuberculoprotein, it will be found that there is only a very moderate increase in the local reactivity and the degree of inflam-

matory reaction and tissue destruction in the eyes parallels the degree of local hypersensitivity.

This fundamental principle, that inflammation, caseation and necrosis parallel the degree of local tissue reactivity to tuberculoprotein, can be illustrated in various other experiments. For example, rabbits develop varying degrees of hypersensitivity to tuberculoprotein after systemic infection. Thus when the eyes of animals with a low sensitivity are inoculated with tubercle bacilli only minimal inflammatory lesions result, while if the eyes of animals with high sensitivity are selected much more pronounced inflammatory symptoms result. Similarly, if hypersensitive rabbits are desensitized with tuberculin prior to inoculation of the eyes, there is almost no inflammatory reaction to the tuberculin in the inoculum and any later inflammation or caseation is proportional to the degree of local hypersensitivity the eyes later again develop as a result of the local infection.

Just as different rabbits develop different degrees of tissue hypersensitivity after systemic infection, they likewise develop different degrees of immunity. When a series of rabbits with systemic tuberculosis are inoculated in the eyes, some develop no symptoms whatsoever, while others develop ocular lesions after widely varying inoculation periods. Some require a huge dose of bacilli to evoke ocular lesions, while others develop lesions after a relatively small dose of bacilli. In rabbits with low immunity the tuberculous lesions usually spread throughout the eye, and with the spread of the lesions increased local hypersensitivity develops, and as a consequence an increased inflammatory reaction with greater tendency to caseation and necrosis. In rabbits with high immunity, the lesions are much smaller, show little tendency to spread, run a shorter course, and heal comparatively quickly.

Thus by studying large numbers of systemically infected rabbits, which have later been inoculated in the eye, the factors which modify the course and character of ocular lesions become apparent. If the immunity is high and the tissue hypersensitivity is low, minimal lesions result, with little inflammatory reaction, and such lesions usually become quickly encapsulated. On the other hand if the immunity is low and the tissue hypersensitivity is high, then spreading inflammatory lesions result with caseation and necrosis. Various fluctuations between these two extremes produce the intermediate chronic type of reaction.

I have spoken of "local" tissue hypersensitivity, meaning the hypersensitivity of the tissues of the eye and not the general cutaneous or systemic hypersensitivity. In experimental animals the local ocular sensitivity can be readily determined by injecting measured amounts of tuberculin or purified protein derivitive into the eye, estimating the ensuing inflammatory reaction, then enucleating the eye and evaluating the histologic reaction. Unfortunately in patients, there is no safe way of estimating this ocular sensitivity. From experimental studies, it has been closely demonstrated that in a tuberculous eye, there may be no parallelism between the local ocular reactivity to tuberculin and the cutaneous reactivity. The local disease in the eye may produce a high degree of local ocular reactivity, while the cutaneous sensitivity remains low. The tuberculous process in the scleral envelope is insufficient to affect profoundly the cutaneous sensitivity. However, if the cutaneous sensitivity is high, the ocular sensitivity is likewise high, for the eye participates in the general tissue reaction.

In the study of clinical ocular tuberculosis, we are handicapped by this in-

ability to determine the degree of local ocular hypersensitivity. All that can be determined is the degree of cutaneous sensitivity. If this is high, then the ocular sensitivity can safely be assumed to be high. But if the cutaneous sensitivity is low, the ocular sensitivity may be much higher, influenced by the local disease. The degree of ocular sensitivity in such cases can be estimated only by studying the degree of inflammatory reaction, searching for evidences of caseation and necrosis, which are the concomitants of local tissue sensitivity. Yet, despite this handicap, when cases of clinical ocular tuberculosis are studied over a period of years, they appear to follow the same general pattern of experimental lesions.

Thus in infants and children ocular tuberculosis is characterized usually by a high degree of hypersensitivity and a low immunity. A tuberculous choroiditis in a child or in the Negro (where tuberculous process usually is of the infantile type) usually spreads rapidly and tends to involve the entire fundus with extensive caseation and tissue destruction. Tuberculosis of the anterior uvea usually is of the same severe type. In children with a miliary tuberculosis, tubercles on the iris and choroid are not uncommon. These children may be so ill that they are unable to develop any tissue response to the invading bacilli and there may be little inflammatory reaction around the tubercles. But if these children are hypersensitive or if they do not die from the generalized infection and bacillemia and later develop hypersensitivity, then the iris tubercles undergo an inflammatory reaction, increase in size, coalesce, and there is caseation, tissue necrosis and often actual perforation of the eye.

In young adults or older patients when a general systemic immunity of varying degree has developed, but where tissue hypersensitivity may still be high, tuberculous lesions such as sclerokeratitis run a typical course of attacks of acute inflammation, but tend to be walled-off, self limited, only to recur again and again as the resistance fluctuates. Tuberculous periphlebitis with recurrent vitreous hemorrhages is commonly a disease of early adolescent life, and is usually accompanied by a high degree of sensitivity to tuberculin. What histologic evidence there is indicates that the so-called "circumscribed plastic choroiditis", is a localized tuberculous lesion. It occurs as a rule in adults and is characterized by single exudates near the posterior pole of the eye which quickly become circumscribed and walled-off. In such patients the cutaneous sensitivity to tuberculin is usually moderate or low. The course of the choroidal lesion, with an absence of surrounding inflammatory reaction, and rapid walling-off and gliosis, suggests the local tissue sensitivity is also low, while the immunity of the patient is high. In short, in individuals with high sensitivity and low immunity, the lesions of ocular tuberculosis are characterized by high inflammation, invasive tendency and tissue destruction, while in individuals with low sensitivity and a well developed immunity the lesions tend to be minimal, attended with little inflammatory reaction, and are rapidly encapsulated. The long drawn-out, chronic fluctuating lesions are indicative of both moderate resistance and hypersensitivity.

If we grasp the underlying principles which determine the character and course of tuberculous lesions of the eye, the therapeutic indications are clear—to enhance the resistance of the patient to the disease, and to abolish the tissue hypersensitivity. Unfortunately there is no direct way to increase the immunity, artificial immunization is still much of a sought-for dream. General hygienic measures are still our main resource. Desensitization with tuberculin is a long and laborious process, often difficult and

sometime impossible to accomplish, and when once accomplished can usually be maintained only by continued tuberculin treatment over years. Yet unsatisfactory as these procedures may be, they are still the cornerstones of the treatment of ocular tuberculosis.

I have endeavored to point out the clinical differences between two types of uveal disease, nongranulomatous and granulomatous. Both types are dependent on invasion of the eye, at one time or another, by living bacteria, but the pathogenesis and character of the lesions in these two types of uveal disease, appear to be quite different. In the nongranulomatous type the primary invasion of the eye is probably by organisms either of low virulence or in small numbers. The result is that they are destroyed by the normal bacteriocidal action of the ocular fluids. This primary invasion produces either insignificant lesions or no lesions at all, but it does produce a local hypersensitivity of the ocular tissues either to the bacterial protein or to the soluble bacterial products. This hypersensitivity may be of the bacterial type, or of both the anaphylactic and bacterial type. When the bacterial antigens again reach the eye, either through reinfection or absorption from an infected focus or even a normal cutaneous or mucous membrane surface, there results a hypersensitive reaction in the eye. This reaction is produced either by organisms which do not proliferate, or by their bacterial products. In either event, the reaction is essentially an evanescent one and is characterized by an intense vascular congestion and a minimum of tissue damage—the picture of a nongranulomatous uveitis. In the granulomatous type of uveitis the mechanism is quite different. The organisms reaching the eye are not destroyed, but remain viable in the ocular tissues, and by their presence, proliferation, and inherent toxicity, produce local lesions in the eye. The character of the resulting lesion is profoundly modified and influenced by the factors of local hypersensitivity and general immunity. The proliferation of the organisms in the eye produces a local hypersensitivity to the bacterial products. This hypersensitivity is of the bacterial type, and a reaction between the hypersensitive tissue and the bacterial products may thereafter result. If the proliferation of the bacteria is not restrained by the forces of immunity, this hypersensitive reaction will be progressive, characterized by inflammation, caseation, necrosis, tissue destruction and often a compensatory overgrowth of granulomatous tissue. If however, there is present either a natural or an acquired resistance to the infection, the proliferation of the bacteria is restrained and the reaction is minimal.

I have used tuberculosis as an example to illustrate the influence of hypersensitivity and immunity in granulomatous uveitis. I realize fully that there are many peculiarities of the tubercle bacillus which differentiate it sharply from non-acid-fast pathogenic organisms and open the door for the criticism that the general laws which influence tuberculous lesions may not be applicable to disease caused by other bacteria. Yet I do not believe this to be true. I believe the same general principles of resistance to infection and tissue hypersensitivity which have been so extensively studied in tuberculosis, will likewise apply, with minor variations, to the lesions found in other infectious granulomatous diseases. This can be determined only through further animal experimentation and clinical observation. Our knowledge of resistance and hypersensitivity and their influence on local and systemic disease has broadened greatly in the last few decades, but there is still much to be learned. If we approach the problem of the etiology of uveitis with an orderly concept of the underlying principles of infection, resistance, and hypersensitivity,

as they are now known and may later be amplified, I believe the influence of these fundamental tissue reactions will be demonstrated, not only for the present known causes of uveal disease, but also for other etiological factors which yet remain to be discovered.

## REFERENCES


1. Berens, C., Angevine, D.M., Loren G., and Rothbard, S.: Eye lesions in experimental infections. Special reference to arthritis, Am. J. Ophth., 21:1315-1329, 1938.
2. Brown, A. L.: Considerations underlying the experimental production of uveitis, Am. J. Ophth., 15:19-23, (Jan.) 1932.
3. Derick, C. L., and Swift, H. F: Reactions of rabbits to non-hemolytic streptococci; General tuberculin-like hypersensitiveness, allergy or hyperegy following secondary reaction, J. Exper. Med., 49: 615-636, (April) 1929.
4. Kolmer, J. A.: Bacteriological and immunological aspects of iritis with special reference to its relationship to bacterial allergy, Am. J. Ophth., 14:217-222, (March) 1931.
5. Krusius, Franz F.: Ueberempfindlichkeitsversuche vom auge aus, Arch. f. Augenh., 67:6-35, 1910.
6. Kümmel, R.: Ueber anaphylaktische erscheinungen am auge, Arch. f. Ophth., 77:393-408, 1910.
7. Rich, A. R.: The pathogenesis of tuberculosis, Charles C. Thomas, Springfield, Illinois, 1944.
8. Rich, A. R.; and McCordock, H. A.: Enquiry concerning role of allergy, immunity and other factors of importance in pathogenesis of human tuberculosis, Bull. Johns Hopkins Hosp., 44:273-422, (May) 1929.
9. Wainwright, C. W.: Treatment of chronic rheumatoid arthritis with streptococcus vaccine on basis of skin sensitivity, J.A.M.A., 103:1357-1361, (Nov.) 1934.
10. Wainwright, C. W.: Treatment of chronic rheumatoid arthritis; Further observation on the use of streptococcus vaccine, Ann. Int. Med., 9:245-251, (Sept.) 1935.
11. Wessely, F.: Ueber anaphylaktische erscheinungen an der hornhaut, Munchen. Med. Wchnschr., 58:1713-1714, 1911.
12. Woods, Alan C.: Ocular anaphylaxis. I. The reaction to perfusion with specific antigen, Arch. Ophth., 45:557-573, 1916.

# THE TREATMENT OF TUBERCULOSIS OF THE LARYNX WITH STREPTOMYCIN

FREDERICK A. FIGI, M.D.
*and*
H. CORWIN HINSHAW, M.D.
BY INVITATION
ROCHESTER, MINNESOTA

TUBERCULOSIS of the pharynx or larynx complicating pulmonary phthisis is extremely serious since it indicates lessened resistance on the part of the patient or increased virulence of the infecting organism or both. Moreover the severe pain associated with tuberculosis of the larynx or pharynx tends to lower the patient's morale, interferes seriously with ingestion of fluids and nourishment, and thereby adversely affects the ultimate prognosis. The deleterious effect of secondary invasion of the throat is especially serious in the presence of incipient or early involvement of the lungs as is shown by the studies made by St. Clair Thomson[5] in this connection. He observed that in cases of early pulmonary tuberculosis the development of laryngeal involvement reduced the likelihood of cure by approximately a half. In cases of moderately extensive pulmonary disease, invasion of the larynx lessened the chance of cure only about a third, while in cases of advanced disease of the lungs the superimposition of laryngeal involvement rendered the outlook only slightly worse because the prognosis was already poor.

In the past the treatment of tuberculosis of the pharynx and larynx has been protracted, tedious and in many instances ineffective. As Spencer[4] has stated, nearly everything has been recommended and used for the treatment of this condition. Among the therapeutic measures employed were chemicals, such as lactic acid, formalin, trichloracetic acid and chaulmoogra oil, also tuberculin and physical agents including the sun's rays or quartz light reflected directly onto the involved region, radium and roentgen rays. Most of these accomplished little or afforded only temporary palliation. The most effective agents in dealing with this disease have been the actual cautery and electrocoagulation. These are particularly useful in dealing with chronic lesions and especially localized tuberculomas. By use of the cautery combined with sanatorium regime and resting the voice, Thomson[6] in 1924 secured what were probably the most favorable results in the treatment of cases of tuberculous laryngitis recorded up to that time. He presented 119 cures in a group of 477 patients. The therapeutic armamentarium has changed little since the publication of his report. Mildly encouraging results have been reported recently from the topical use of a solution of promin in the larynx. However, public health measures, educational programs and better living conditions have decreased the incidence of pulmonary tuberculosis while collapse of the lung and other more radical operative measures have tended to lessen further the number of patients who have involvement of the throat.

Four patients who had severe involvement of the throat have been treated

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.
Section on Laryngology, Oral and Plastic Surgery and Division of Medicine, Mayo Clinic, Rochester, Minn.

with streptomycin at the Mayo Clinic in the year and a half prior to September, 1946.

In the treatment of the cases of tuberculosis of the pharynx and larynx, streptomycin was given both by parenteral injection and by inhalation of nebulized aerosol. The dose which was administered parenterally varied from 0.5 to 2 gm. (500,000 to 2,000,000 units) of streptomycin hydrochloride per twenty-four hours. This quantity was divided into eight equal doses and one of these was injected subcutaneously or intramuscularly every three hours. For nebulization 0.5 gm. of streptomycin hydrochloride was dissolved in 20 or 30 c.c. of physiologic saline solution and 2 or 3 c.c. of this was administered at the beginning of each hour for ten hours each day. In this treatment the technic described by Olsen[3] was utilized. The length of time that the treatment was continued and the total amount of streptomycin given varied greatly in the different cases. These were determined by the response of the patient to the treatment and the availability of the drug. The periods of treatment in the four cases were twenty-six days in case 1, forty-six in case 2, seventy in case 3 and 104 in case 4. The total combined dose administered parenterally and by nebulization to each of the four patients was 37 gm., 56 gm., 96.5 gm. and 178 gm. respectively.

Toxic phenomena attributed to impurities rendered it necessary to discontinue treatment for a day in one or two cases but subsequent administration of a different lot of streptomycin usually was well tolerated. The only other evidence of toxicity to the drug shown by these patients was that affecting the vestibular labyrinth. This complication has been described recently by Brown and Hinshaw.[1] It consists of vertigo and tinnitus of varying duration and occurred in three of the four patients which form the basis of this report.

The response of the pharyngeal and laryngeal lesions in these patients to streptomycin was striking.

## REPORT OF CASES

*Case* 1.—The patient, a woman thirty-six years of age who had multiple, superficial, painful tuberculous ulcers of the hypopharynx, came for consideration because of acute pain on swallowing. The ulcers had been present continuously for two years and during the two weeks prior to her registration at the clinic she had lost 6 pounds (2.7 kg.) chiefly because of her inability to take ample nourishment. On examination of the sputum tubercle bacilli were found and roentgenograms of the thorax revealed extensive active tuberculosis involving the upper lobes of both lungs. Tissue removed from one of the ulcerated regions in the hypopharynx proved to be tuberculous on microscopic study (fig. 1).

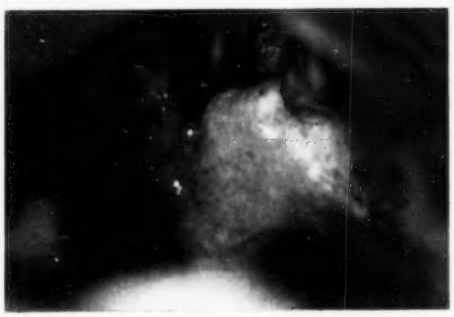


FIG. 1. (case 1). Tuberculous laryngitis and pharyngitis with marked edema of the right arytenoid and ulceration of the larynx and hypopharynx. These ulcers were completely healed seventeen days after treatment with streptomycin was instituted.

Streptomycin was given both intramuscularly and by nebulization. Within two weeks of the time this treatment was started, the cough had decreased noticeably, there was less sputum and the patient was able to swallow and yawn without pain. Examination of the throat on the seventeenth day of treatment disclosed that the ulcers of the throat were healed completely. The treatment was discontinued on the twenty-sixth day as it did not appear likely that sufficient streptomycin could be obtained to permit our keeping the patient until her pulmonary lesion could be stabilized.

After leaving the clinic, she entered a sanatorium where routine treatment was carried out. Her physicians have recently reported that the ulcers in her throat have not recurred, that her general health has improved greatly and that healing of the lesion of her left lung has taken place. She is still receiving pneumothorax for the involvement of the right lung.

*Case 2.*—The patient, a man sixty-one years of age, who had extensive, bilateral fibrotic pulmonary tuberculosis, came for consideration because of excruciating pain in his throat and ears especially on swallowing. This pain had been so severe that a month previously he had been unable to eat for several days. He had lost 28 pounds (12.7 kg.) during the two months prior to registration in the clinic and on admission was coughing badly and expectorating large amounts of sputum. In addition to the extensive active pulmonary disease a large excavated ulcer covered most of the anterior and posterior aspect of the epiglottis and involved the right lateral epiglottic fold. This had destroyed the upper portion of the epiglottis. Ulceration was also present on the right arytenoid and both vocal cords were slightly edematous and were covered with superficial ulcers. Five days after treatment with streptomycin was started by intramuscular injection and nebulization, the patient stated that his throat felt better and that he no longer experienced pain on swallowing. Whereas he had previously drunk water only at mealtime he now took fluids freely ad libitum. The margins of the excavated ulcer of the epiglottis had already smoothed off considerably. Prior to this time we had not felt justified in administering pentothal sodium anesthesia and performing suspension laryngoscopy in order to remove a specimen for biopsy and to secure photographs of the laryngeal and pharyngeal lesions.

By the twenty-fourth day the patient's general appearance was much improved. He was eating well and gaining weight. On examination at that time the ulcers of the epiglottis and of the remainder of the larynx were found to be healed (figs. 2*a* and *b* and 3). However, marked reddening and thickening of the entire laryngeal introitus still persisted. When the patient left the clinic after three and a half months of treatment his voice was normal, his larynx had been healed for more than two months and he had been eating without discomfort for more than three months. His weight had increased from 100 to 124 pounds (45.4 to 56.2 kg.) and no tubercle bacilli had been found on repeated examinaions of sputum for about two months.

When the patient was observed one month subsequently he appeared to be in robust health and he had gained an additional 10 pounds (4.5 kg.). His larynx was healed but some injection still remained. Physical examination of the thorax revealed râles over both upper lobes anteriorly and no

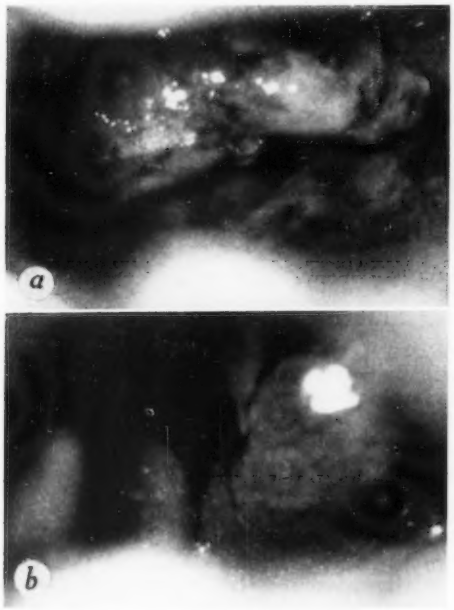



FIG. 2 (case 2) *a*. Markedly edematous epiglottis with ulceration of tip and right border due to tuberculosis. Treatment with streptomycin had been given for a week and considerable improvement had already occurred. *b*. Marked edema of the arytenoids. There was also ulceration of the right arytenoid and of both vocal cords.

FIG. 3 (case 2). Appearance of larynx six weeks after figure 2*a* and *b*. Ulcers were completely healed.

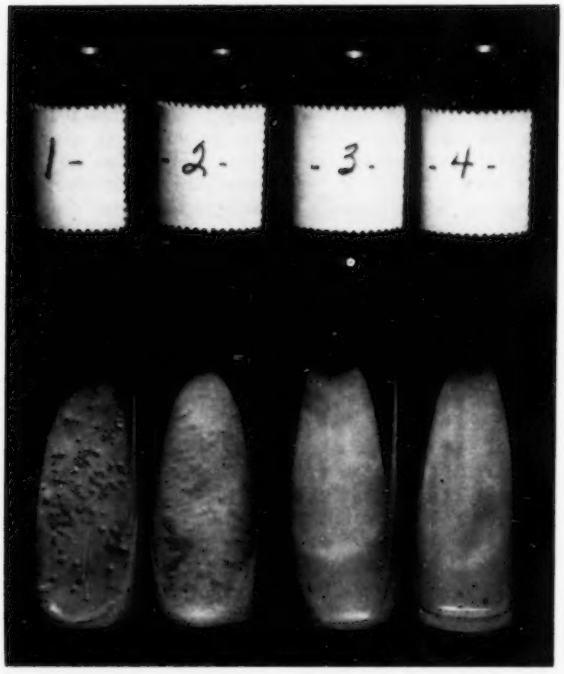



FIG. 4 (case 3). Cultures of sputum on glycerin egg medium showing two tubes (1 and 2) which yielded positive results, with numerous colonies of tubercle bacilli, before treatment (March 20, 1945) and two tubes (3 and 4) which yielded negative results following treatment (April 24, 1945).

change could be made out in the roentgenograms of the thorax. However, further examination on September 9, 1946, revealed marked decrease in the physical findings, and roentgenograms of the chest showed striking evidence of improvement.

*Case 3.*—The patient, a woman thirty-two years of age, came because of hoarseness of a year's duration. Examination disclosed a papillary tuberculoma which involved the entire glottis including the interarytenoid region. This was most marked on the right vocal cord and movement of the cord was limited. Physical examination of the thorax revealed no evidence of active pulmonary tuberculosis and roentgenograms presented only slight increase in the markings about the right hilus. Tubercle bacilli were present in the sputum. With the aid of intravenous pentothal sodium anesthesia and suspension laryngoscopy a specimen was taken for biopsy and a photographic record was made of the laryngeal lesion. On microscopic study of the tissue removed from the larynx caseating tuberculosis and a few acid-fast bacilli were found in the caseous portion of the specimen (figs. 4 and 5).

Streptomycin was given by subcutaneous injection and by nebulization for six weeks. Considerable improvement in the lesion of the larynx was noted on the tenth day of treatment and at the end of eight weeks the verrucous overgrowth that had previously involved the entire glottis had disappeared. When the patient was observed five months after this treatment was begun her voice was normal, she was in excellent general health and examination of the larynx did not reveal anything of significance (fig. 6). In a recent letter the patient stated that she has been enjoying good general health and that her voice has been normal right along.

This case was reported in a previous article.[2]

*Case 4.*—This patient, a war veteran twenty-eight years of age, was sent to the clinic from a government hospital. He had been under treatment in and out of military hos-

pitals for pulmonary tuberculosis for two and a half years and had been hospitalized and was at rest in bed continuously for eight months prior to coming under our care. During this period he had been continuously hoarse, and frequently had had thoracic pain and the pulmonary disease had progressed. On examination at the clinic the patient was extremely weak, markedly emaciated, anemic and aphonic and was experiencing great difficulty and pain on swallowing. Laryngeal examination was unsatisfactory because of poor co-operation and the anatomic conformation but fleeting glimpses showed injection and edema of the arytenoids and hypertrophic thickening in the interarytenoid region. No ulceration was discernible, probably because of the tenacious mucus present. Roentgenograms of the thorax revealed evidence of bilateral active tuberculosis with extensive cavitation involving most of the left lung and extensive exudative and caseous tuberculosis involving most of the right lung. Mycobacterium tuberculosis was present in the sputum. The sedimentation rate was 126 mm. per hour. Since the left lung was essentially destroyed and the process in the right lung probably already was destructive, a more difficult test of the effectiveness of streptomycin could scarcely be devised. In this case penicillin administered by nebulization was used as a supplement to the streptomycin administered by nebulization; otherwise, treatment was carried out in the same manner as in the preceding cases.

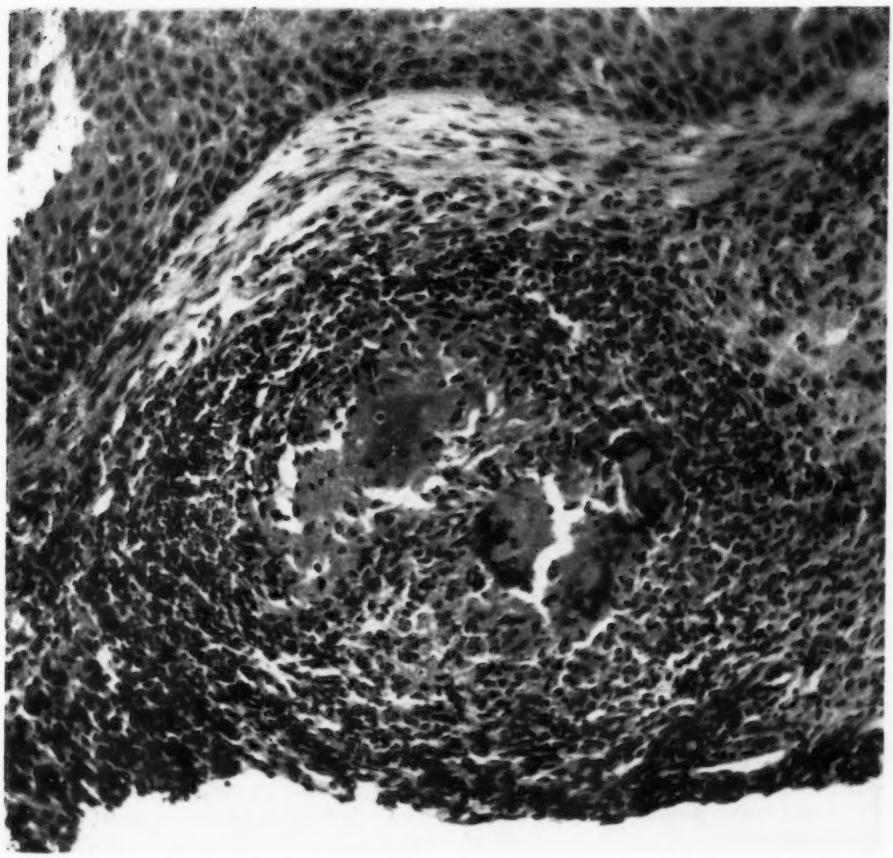


FIG. 5 (case 3). Typical tubercle with several giant cells in tissue removed for biopsy. A few acid-fast bacilli were present in the caseous central portion of the lesion in appropriately stained sections.

Within a week after treatment was initiated, the patient stated that his cough was improved 50 per cent. Within two weeks the

voice was improved and injection and swelling of the arytenoids had decreased. However, later the cough became worse and continued to be bad in spite of treatment. In addition the sputum continued to contain tubercle bacilli and the sedimentation rate remained high. Treatment was discontinued on the seventieth day as it was felt that the organism probably had become resistant to streptomycin and that not enough to be worth while was being accomplished. A short time ago in a telephone conversation with this patient's physician we were informed that the patient's general condition has recently improved markedly and that there has been no return of his laryngeal disease.

While this series is too small to permit us to draw any definite conclusions, it is interesting to note that amelioration of the symptoms referable to the throat occurred promptly in all of these cases. Moreover, it preceded evidence of improvement in the general condition and appeared to have a direct bearing on this. The prompt relief from the acute soreness and pain in the pharynx and larynx suggests the possibility that secondary infection was partly responsible for these symptoms. It is of interest also that the ulcers of the pharynx and larynx healed and showed no tendency to recur even in those cases in which tubercle bacilli continued to be present in the sputum.

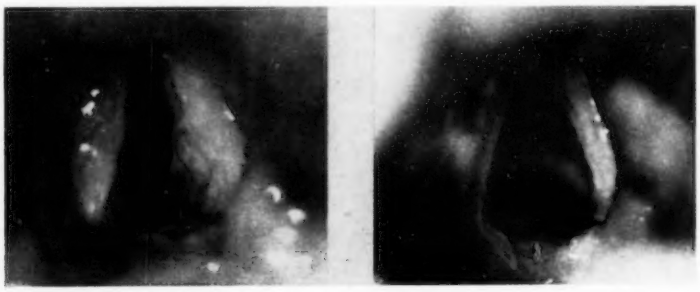


FIG. 6 (case 3). Larynx: *a*, on March 22, 1945, prior to starting treatment with streptomycin; marked papillary thickening, which was especially pronounced on the right vocal cord, involved the entire glottis; *b*, on May 22, 1945, following completion of treatment with streptomycin. The larynx appeared to be completely healed. Only slight fullness and injection of the right vocal cord remained.

## COMMENT

In all four of these cases the lesions of the pharynx and larynx had become progressively worse up to the beginning of treatment with streptomycin. In addition the general health of three patients had deteriorated for from two to several years preceding treatment with streptomycin.

The lesions in the throat for which streptomycin was administered were of three distinct types. Included were: (1) an indolent verrucous tuberculoma, (2) multiple superficial, painful, moth-eaten ulcers of the hypopharynx and (3) an extensive excavated, destructive ulcer of the epiglottis. The prognosis in this third type of lesion generally is bad.

The regression of these tuberculous lesions and their complete healing was more rapid than we have observed with any other type of treatment. This was true also of the improvement in the patient's general condition.

Further observation will be necessary in order to determine the ultimate effect of treatment with streptomycin on the pulmonary lesions in three of these patients. Improvement in the pulmonary process was not always commensurate with that of either the throat or the systemic condition. In fact, roentgeno-

graphic study of the thorax at times failed to reveal any definite change in the pulmonary findings even after the lesions in the throat had practically returned to normal. However, further observation later showed marked improvement in the pulmonary condition both on physical and on roentgenographic examination.

## REFERENCES

1. Brown, H. A. and Hinshaw, H. C.: Toxic reaction of streptomycin on the eighth nerve apparatus. Proc. Staff Meet., Mayo Clin. 21:347-352 (Sept. 4) 1946.

2. Figi, F. A., Hinshaw, H. C., and Feldman, W. H.: Treatment of tuberculosis of the larynx with streptomycin: report of case. Proc. Staff Meet., Mayo Clin. 21:127-130 Mar. 20) 1946.

3. Olsen, A. M.: Streptomycin aerosol in the treatment of chronic bronchiectasis: preliminary report. Proc. Staff Meet., Mayo Clin. 21:53-54 (Feb. 6) 1946.

4. Spencer, F. R.: Laryngeal tuberculosis. Philadelphia, J. B. Lippincott Company, 1927, 69 pp.

5. Thomson, St. Clair: The prognostic importance of tuberculosis of the larynx. Lancet. 2:689-690 (Oct. 18) 1919.

6. Thomson, St. Clair: Tuberculosis of the larynx: ten years' experience in a sanatorium. In: Medical Research Council, London, His Majesty's Stationery Office, 1924, no. 83, 91 pp.

## DISCUSSION

FRANK R. SPENCER, M.D., Boulder, Colo.: I did not have an opportunity to read Dr. Figi's paper before coming to this meeting as he didn't have his paper finished. He did send me a reprint of his article published last March, with a report of one patient cured with streptomycin. My discussion is, therefore, based on the articles from the Mayo Clinic about streptomycin. I believe I have read all of these.

The treatment of laryngeal tuberculosis has always been discouraging and that is doubtless one reason why so many laryngologists, all over the world, take so little interest in this disease.

When the sulfa drugs began to be used 10 years ago we were all hopeful of good results from these. At the Mayo Clinic the results showed that most of the patients manifested some improvement while taking the drug, from the lessening of secondary infection. The improvement was not always permanent. We used the sulfa drugs for the treatment of tuberculous otitis media at the University of Colorado School of Medicine in rabbits and guinea pigs. We obtained improvement only while the drug was being given. Our results were reported before the American Otological Society.

The improvement which Dr. Figi, and his coworkers, have obtained with streptomycin is the best I know of and in the very short period of six weeks. Very few patients have shown so much improvement in six months by the older methods of treatment. I am sure I have not cured any patient, with such marked laryngeal tuberculosis, in six months. Dr. Figi is quite frank in saying the absence of more than slight pulmonary disease helped and it undoubtedly did.

Over a period of years I have read, with a great deal of interest, the reports from the Mayo Clinic on their experimental work with tuberculosis. This paper by Figi is a well deserved reward for their years of research. Their frankness in not overstating their results is an outstanding characteristic of their achievement.

It is conceivable that even with positive sputum the secondary infection played the major role. Subsequent treatment of other patients, with more pulmonary tuberculosis, will help to decide this point. Dr. Figi and his coworkers are too keen not to have realized this. I shall await with a great deal of interest their subsequent reports.

In the Proceedings of the Staff Meetings at the Mayo Clinic for Wednesday, September 4th, Brown and Hinshaw reported the toxic reaction of streptomycin on the eighth nerve apparatus.

It is my understanding that patients may suffer damage to the vestibular apparatus, with varying degrees of vertigo, following the administration of streptomycin. Most of the patients had only mild vertigo. In one series of cases I understand 35 per cent of the patients had more or less severe vertigo with comparable hearing losses. So far as

I know no one else has reported this complication. We have used streptomycin, but in smaller doses to avoid labyrinthitis. We have not used enough or for a long enough period of time to justify reporting our results. Doubtless in the future a preparation of streptomycin will be made which will avoid this danger. The present preparation of streptomycin may be an impure product. The by products may be toxic for the 8th nerve.

FREDERICK A. FIGI, M.D., Rochester, Minn.: Relative to the toxic effects on the eighth nerve that we have observed in these cases, the patients whom we have had a chance to see some months later have shown marked lessening of these toxic manifestations, and from the progress that they have made to date, we anticipate that these symptoms probably will entirely disappear, although to date some of them have not entirely cleared up.

# TRANSACTIONS

## American Academy of Ophthalmology and Otolaryngology

*Official Publication of the American Academy of Ophthalmology and Otolaryngology*

Published under the direction of the Editorial Board

WILLIAM L. BENEDICT, Rochester, Minnesota, *Editor*

W. HOWARD MORRISON, Omaha, Nebraska, *Associate Editor*

HENRY L. WILLIAMS, Rochester, Minnesota, *Associate Editor*

Printed by Douglas Printing Company, 108 North 18th Street, Omaha 2, Nebraska

All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, the American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota


### Letter to the Editor

Cullen Road, Alleppey, P. O.
Travancore, S. India
October 9, 1946

Dear Dr. Benedict:

I wish to introduce myself to you as a Christian medical practitioner and a recent specialist in oto-rhino-laryngology. I am the only practitioner in this specialty in this industrial town where 90 per cent of the population are labourers. I regret very much to inform you that I have no proper consultant nor any kind of special literature in oto-rhino-laryngology and my funds are inadequate to purchase any costly literature. Therefore, I request you to kindly help me with the TRANSACTIONS of your Society, reprints of important articles, illustrated chrats, booklets and used copies of good journals. So much as far as your official portfolio is concerned.

Further I wish to make certain requests to you for your personal help. I request you to kindly help me with some practical books on the above specialty, and also with some good journals which are used by you.

I request you to kindly excuse me for all the trouble I have given to you.

With extreme regards,

Yours very truly,

(Signed) T. K. THOMAS

## In Memoriam

Claude Worth McKee.....Greensburg, Pennsylvania
September 11, 1946

Harold Vincent Phelan.....Cleveland, Ohio
September 1, 1946

George William Schlindwein.....Erie, Pennsylvania
March 19, 1946

Andrew Timberman .....Columbus, Ohio
October 15, 1946

Richard Joseph Tivnen.....Chicago, Illinois
August 27, 1946

# OCCUPATIONAL OPHTHALMOLOGY

## INTRODUCTORY STATEMENT

*The relationship between the ophthalmologist and occupational industrial eye problems, is one of the less tangible but most vital phases of the multiple responsibilities presented to ophthalmologists by the industries in their communities. Dr. Snell has pointed out in his discussion of the ophthalmologist and his relation to occupational eye problems, some of the important aspects of these responsibilities. Let no one think that the psychology of a medical person's approach to industry and the basis of their cooperation with industry is not of first importance—it is sometimes the pivot on which all else hangs.*

*In the last six years, while our studies of industrial eye problems have broadened, we have found that Faber Birren, paralleling our many efforts, has worked out a very clear-cut, practical approach to the place color has in one's work environment. His technics and advice are simple, dependable and very inspiring. This short paper will be followed in a later number by a full discussion of color and illumination.*

*Medical people can never be given too many instances of the relationship between production and visual efficiency. We learn figures and statistics the hard way, but Dr. Wirt's discussion of production increases in solderers brings this work again to our attention.*

*The research work at Wilmer Institute on lime and caustic burns is interesting. Your secretary does not feel as is stated in this memorandum, that neutralization is important especially in those all important first seconds. Early and copious irrigation, followed by proper treatment, has been most effective in her experience. However, this report is very important.*

HEDWIG S. KUHN, M.D.
*Secretary, Joint Committee*
*Occupational Ophthalmology*

## INDUSTRIAL EYE PROBLEMS AND OPHTHALMOLOGISTS

A. C. SNELL, M.D.
ROCHESTER, N. Y.

OUR attention first is directed to only three phases out of the many eye problems which confront the ophthalmologist who is interested in the occupational aspects of ophthalmology.

On many occasions I have been asked: "In what respect does the practice of industrial (occupational) ophthalmology differ from the general practice of ophthalmology?" A complete answer to this question would indicate most of the industrial eye problems which are presented to the ophthalmologists. In considering this question we will all admit, I feel sure, that the science and practice of ophthalmology present many special phases; and that few of us have acquired complete knowledge of all these phases —example, ocular pathology, neuro-ophthalmology. In the field of industrial eye work, there also are special problems not encountered in the ordinary practice of ophthalmology.

Let me point out that basically occupational eye problems may be broadly divided into three classes; first, those relating to human nature (this applies not only to industrial employees but also to management, supervisory personnel and to ophthalmologists). You may notice that no distinction is made between the human characteristics of employees and ophthalmologists! The remark of a certain U. S. senator is opposite to this thought when he said: "It is my observation, sir, that one individual has as much human nature as the next man—if not more so." Second are those problems which relate to physical defects,

their discovery and correction; and third is the problem of efficient utilization of visual skills when these are brought to their maximum efficiency. My discussion concentrates on the first.

The characteristics of human nature are not all alike; and in industrial practice the most difficult problems encountered are based on the necessity of dealing with several equally important groups of interested people. Thus the industrial ophthalmologist must possess some of the characteristics found in a politician or diplomat as well as those qualities which make for a successful ophthalmologist. H. Clipper in a recent number of the *Scientific Monthly* describes some of the characteristics which one employed in professional service may be expected to display, and which are opposite to the industrial ophthalmologists. He writes that such a professional person is expected to display the following characteristics: '.The sympathetic understanding of a father confessor, the diplomacy of an embassy attache, the business acumen of a wholesale merchant, the salesmanship of a used-car dealer, the financial judgment of a stock broker, and the information of a radio-quiz expert." In no other field of the practice of medicine is there a greater demand for the use of tact. The industrial ophthalmologist must possess administrative ability and have a flair for the social-economic phases of industrial practice.

Some of the desirable characteristics which the occupational ophthalmologist should possess may be comprehended by directing attention to both the disadvantages and the advantages of practice in this field. Some of the disadvantages may be mentioned as follows: (1) When one practices in the industrial field he is not completely his own boss; the relationship is not solely between physician and patient, since the industrial ophthalmologist must also take into consideration the interest of management, including, of course, the normal function of placement personnel, safety engineer, illuminating engineer, insurance carriers and state or federal compensation officials. Thus, a successful occupational ophthalmologist must be informed to a degree at least in *all* these special fields. He must have the ability to use this information in a spirit of real helpful cooperation. Since many industrial eye problems which are presented to the ophthalmologist have a social-economic aspect the ophthalmologist must have a flair for the social-economic phases of industrial production. (2) The practice of industrial ophthalmology offers only a limited amount of eye surgery. (3) There are more routine procedures to follow including reports than one meets with in ordinary practice. With adequate clerical help this obligation need not be irksome. (4) One must admit that while a "company doctor" has not in the past always received from industrial employees the same respect and confidence that a patient places in his own chosen physician, the question of respect and confidence depends largely on the personality and integrity of the ophthalmologist. An attitude of personal interest in the problems presented by each employee, a sympathetic explanation of the condition found and of recommendations to be followed, together with an attitude which displays a desire to be helpful, will aid substantially in maintaining the traditional relationship between employee-patient and industrial ophthalmologist. Here as elsewhere, of course, the patient, though an employee, is the number one consideration.

There are many most interesting aspects and advantages in incorporating industrial work into one's practice — a sense of direct public responsibility and service, an opportunity of correlating professional care and its results, and an opportunity to make scientific contribu-

tions in a field which seems still unexplored.

To those ophthalmologists who desire to devote all their time to industrial practice, this field should be full of opportunities and of interest. Most ophthalmologists who wish to do industrial eye work do so on a part-time basis and this is the most satisfactory all the way around. Whether part-time or full-time, or as a consultant, this field offers an unusual opportunity for service to a section of our public which has been largely neglected.

We have called attention to those human factors in industrial practice which primarily concern the ophthalmologist. An exhaustive discussion of the human employee characteristics as these relate to the use and conservation of employees' vision would require a volume on psychology. The importance of the human factor in industry and the problem of securing full cooperation from employees is well illustrated by the observation that in the accident prevention field alone it has been reliably estimated that 90 per cent of eye accidents are due to human failures and only 10 per cent to mechanical failures. In other words, although protective programs of education and mechanical devices have been instituted in a large proportion of industrial practice, eye accidents are still far too numerous. The number of compensable eye injuries has not been substantially decreased during the past decade.

In order to clarify the *special* nature of industrial eye problems let me call to your attention some of these activities.

1. Aid in the establishment of a medical staff. An ophthalmologist should be included—full time, part time or as consultant.
2. Establishment of a system, or method of testing the vision of pre-employment applicants and of all employees. Provision should be made for rechecks at stated intervals.
3. The development of suitable records. These should contain all data obtained from the individual's history and from all tests and examinations. These tests should contain data which will aid in the proper placement of all potential or present employees.
4. The establishment of suitable programs for examining those found to have subnormal vision, for correction of these when possible, and for furnishing the indicated supplies.
5. Institution of protective programs and methods for their continued application.
6. Establishment of first-aid stations. This requires special equipment for examination and for treatment of eye injuries. A nurse specially instructed or trained is most suitable and her services are most valuable. All activities of the nurse should be under direction of an ophthalmologist.
7. The study of adequate illumination.
8. The study of occupational lenses. The demands made on vision by modern industry vary greatly with various types of employment, so that the industrial ophthalmologist must consider more than securing the best visual acuity at 20 feet or at 14 inches. Some types of employment require good vision at these and all intermediate points.
9. Assistance in placement. Visual ability or skill is not possessed at a maximum degree by all employees, even with the best correcting devices. Therefore, the ophthalmologist should indicate to the placement personnel, those jobs which are suitable to the best obtainable vision of each employee, keeping in mind efficiency and the risk of injury to his other organs or to other

persons. This implies a huge task of job classification.

10. Consideration of all the special problems corrected with any specific employment. In some occupations color vision is essential; in others skill in color discrimination is required; and in others good stereopsis is necessary.
11. Study of the relation of vision to general health and vice versa. These may include problems of ventilation, of posture, of nutrition, and of domestic and social relations.
12. Rehabilitation of those sustaining eye injuries by finding suitable jobs for these after proper training.
13. Giving aid in problems relating to vision in the medico-legal field which requires expert knowledge of the compensation laws of the several states, and the computation of the degree of visual disability resulting from injury.
14. Consultation and cooperation with management, placement, personnel, medical staff members and others.
15. A comprehension of the social-economic relations. In order to comprehend many of these listed problems the industrial ophthalmologist, especially when he devotes only part time to industry or when he acts only as a consultant, should become personally familiar with all working conditions presented by a specific plant by observing all working conditions and visual demands.

All plants in which visual surveys have been made show that from 10 to 30 per cent of employees have visual defects which interfere with efficient production. Much improvement has been made in employees' vision in many plants during the past six years. However, a large proportion of industrial employees have not had and are not now receiving adequate visual service, so that the outstanding problem which is presented to the ophthalmologist today is that of developing a program of ophthalmic service which can reach all employees in every kind and size of plant wherever located.

To SUMMARIZE, I have attempted to show:

1. Occupational or industrial ophthalmology presents special problems to one who practices in this field.
2. The occupational ophthalmologist must possess unusual human characteristics.
3. The special fields of activity which are presented.
4. There is still present a need for ophthalmic service in industry.

---

## COLOR AND PSYCHOTHERAPY

FABER BIRREN
NEW YORK, N. Y.

BECAUSE of its strong emotional appeal the application of color is likely to be beset by personal prejudices and preferences and by many unfounded claims. In the past many exaggerated theories have been propounded which have justified a skeptical attitude.

If color is to be used sensibly and functionally, it is perhaps best to forego the idea that it has any direct therapeutic value. While this point may be argued, the physical action of color or visible light on the human body has by no means been clearly proved.

It is therefore more reasonable to study color in its more psychological aspects, for here there is no denying that it has strong emotional appeal. Rightly used, it pleases and creates moods fa-

Reprinted from The Modern Hospital, 67:2, August, 1946.

vorable to improved morale and mental attitude.

### EFFECT CAN BE MEASURED

Fortunately, measurements of the effectiveness of color are not all intangible. In human vision, for example, the causes and effects of eye fatigue may be readily checked. Ideal seeing conditions may be "engineered" in accordance with straight scientific practice and no personal judgment or debate is required to determine whether or not color has been rightly applied.

When glare, inadequate levels of illumination, severe contrasts and the like exist, eye fatigue will be apparent. The pupils of the eyes may show severe dilation. The lids may blink more rapidly. There may be a collapse in the "visual-form fields," the outer boundaries of the retina growing less sensitive. There will be increased muscular tension, apparent fatigue and irritability and perhaps headache and nausea.

Ophthalmologists and seeing specialists have been able to check these facts by means of instruments. Hence, eye fatigue is not to be mistaken, nor can the benefits of any lighting installation or color scheme be denied if it overcomes or lessens those reactions so evident when the eye is abused.

In the new science of brightness engineering, reliable formulas have been worked out to assure comfortable seeing conditions. In this application of color (largely for its brightness) right and wrong need not be confused. In fact, the fundamental principle involved is quite simple.

Parry Moon states: "Investigations of the most diverse kind show that a human being sees best and visual fatigue is reduced to a minimum when the entire field of view is approximately the same luminosity as that to which the fovea (center of the retina) is adapted."

Matthew Luckiesh has further shown that brightness ratios smaller than 1 to 5 are desirable; ratios higher than 1 to 10 should be avoided. If these requirements are met, hospital color schemes will be essentially right and quite beyond technical criticism.

Theories of interior decoration often run contrary to the principles of brightness engineering. If for the sake of "dramatic" effects, the patient must look alternately at light ceiling, dark floor, severe contrasts in walls and furnishings, constant pupillar adjustments may be demanded which will cause unmistakable distress.

When proper attention is paid to brightness ratios, all areas and surfaces regardless of their hue will be more or less uniform in brightness. Ceilings should be tinted to relieve glare. Walls should be neither too light nor too dark, perhaps with a reflectance of about 50 per cent. Flooring materials, furniture and draperies should average not less than 20 or 25 per cent in brightness.

If the variety is in color, not in extreme lightness against extreme darkness (and if the illumination of the room is fairly uniform), there will be little or no eye fatigue and prolonged occupancy will not cause discomfort.

### SPECIAL VALUE IN SURGERY

This engineering of color holds special value in surgical departments where critical seeing tasks are performed. *(Surgery is an occupation as well as a profession. H.S.K.)* Walls, if anything, should be a trifle deeper than are working or operating areas. Mr. Luckiesh states: "It may be concluded that brightness somewhat lower than those of the central field are generally most desirable. All experimental evidence indicates that peripheral brightness higher than those of the central field are definitely undesirable."

The use of blue-green in operating rooms, medium in tone, helps to reduce glare, aids visibility and, at the same

time, heightens perception of blood and tissue through visual complementation. White here is obviously to be avoided, particularly if it is brightly illuminated.

Brightness engineering is a relatively new science, given impetus and importance during the war when severe eye tasks were encountered throughout industry. It has brought into the art of color a number of accurate principles and rules which are subject to measurement and which make it unnecessary to rely upon highly fugitive opinions or artistic "feeling." This sure approach has long been needed to divest color of its mysteries and to put it to work with real competence and assurance.

Further and significant work has been done in the field of color by a number of investigators. These studies again suggest a functional approach that sidesteps empiricism and relies upon scientific method in determining why and how color should be employed. Research, of course, is the only sound basis for any application of color which is meant to serve a purpose that goes beyond mere appearance.

### COLORS ARE WARM OR COOL

In the main, the colors of the spectrum divide themselves into two major regions: warm hues, such as red, orange and yellow, and cool hues, such as green, blue and violet. The neutral point is in yellow-green. All radiation striking the eye or the body appears to create a tonus condition (muscular tension).

Reactions are in two directions. With warm hues there is an attraction to stimulus. With green and blue there is a withdrawal from it.

Kurt Goldstein writes, "We find that green favors performance in general, in contrast to red. The effect of red probably goes more in the direction of an impairment of performance." This would mean that green (and blue) more or less is conducive to normal activity and response, while red (and orange) is more impulsive and distracting.

It is also known that under the influence of warm hues time will be overestimated and weights will be judged as being relatively heavy. With cool hues, time will be underestimated and weights will be judged as being relatively light. Goldstein asserts: "Under red and green lights, movements are carried on with a different speed, without subjectively experiencing the change in speed. Likewise, the estimates of traversed distances, time intervals and weights are judged differently under the influence of different colors."

Warm hues, being exciting, tend to "pack" more experience into a given period of time, while cool hues have a contrary effect. Feré noted that muscular tension increased from a normal 23 to 42 under red light, while with blue the increase was only from 23 to 24. Similarly, Gilbert Brighouse showed muscular reactions to be 12 per cent quicker under red light, while green light had a retarding influence.

Such investigations would justify a practical attitude toward the use of color. Warm tints became appropriate for otherwise bare and uninteresting rooms and corridors. Cool hues, because they tend to relax the mood and make time "speed by," become appropriate for utility and working spaces in general.

Many interrelations exist between color and the other senses of the human body. S. V. Kravkov writes: "We may consider it an established fact that the color sensitivity of our . . . vision is dependent in a definite way on the condition of the autonomic nervous system."

From his work, later confirmed by Frank Allen and Manuel Schwartz of Canada, "It may be safely inferred that stimulation of any sense organ influences all other sense organs in their excitability." In brief, stimulation of the retina, the influence of loud noise, strong

odors and tastes depress sensitivity to red and increase sensitivity to green.

Today color preferences and emotional reactions to color are finding plausible explanation. The functioning of the endocrine glands may help to account for seasonal variations in likes and dislikes for color: tints in spring, shades in fall.

E. R. Jaensch has related the effect of red radiation, predominant in the tropics, to pigmentation of the retina and to the general preference of brunet types for red hues. Conversely, retinal accommodations to ultraviolet light in more polar regions may account for the general preference of blond types for greens and blues.

These studies and speculations began to lead from psychologic phenomena to things physiologic. It has long been recognized that the body, aside from vision, has a radiation sense. Blue radiation affects only the superficial layers of the flesh, while yellow and red penetrate more deeply. In experiments performed by Dr. Mizutani of Japan (1940) red was found to accelerate the healing of wounds, while blue retarded it. "As for the effects of visible light upon tumor tissue within the body, red rays affect to prevent the growth and blue rays affect, though slight, to accelerate it."

### COLOR CAN BE USED RATIONALLY

However, it is no doubt safest and wisest to forego the attitude that color has any direct therapeutic benefits in its action upon the human body. To accept it in the realm of psychotherapy is fully defensible and perhaps sufficient for the purposes of hospital decoration or in industry. There are enough reliable facts and plenty of functional reasons to apply color rationally and not merely esthetically.

When the color program has its basis in such measurable technics as brightness engineering and in evident human responses to warm hues as against cool ones, resultful specifications are to be written and the needs are well served.

### BIBLIOGRAPHY

Following are a few excellent articles to be consulted for additional notes on the psychologic and therapeutic aspects of color. For a complete bibliography see "The Story of Color" by Faber Birren, Crimson Press, Westport, Conn., 1941.

Allen, Frank, and Schwartz Manuel: Effect of stimulation of the senses of vision, hearing, taste and smell upon the sensibility of the organs of vision, J. Gen. Physiol., Sept. 20, 1940.

Deutsch, Felix: Psycho-physical reactions of the vascular system to influence of light and to impressions gained through light, Folia clin. orient., 1:3-4, 1937.

Goldstein, Kurt: Some experimental observations concerning the influence of colors on the function of the organism, Occup. Therapy, June, 1942.

Luckiesh, M.: Brightness engineering, Illuminating engineering, February, 1944.

Prescott, Clark Daniels: Psychological analysis of light and color, Occup. Therapy, June, 1942.

Yogo, Eizo: Effect of the visible light upon the vegetative nervous system, Jap. J. Obst. & Gynec., 1940.

---

## EYE CARE INCREASES EARNINGS OF ELECTRIC SOLDERERS

S. Edgar Wirt, Ph.D.
LAFAYETTE, IND.

Professional eye care, to improve visual skills and visual comfort, paid dividends to a group of women employees at the Bausch and Lomb Optical Company. These women do electric gold soldering, joining the small parts that make gold filled spectacle frames.

The work is fine, precise and repetitive at a close working distance. It in-

Division of Education and Applied Psychology, Purdue University, Lafayette, Ind.

Reprinted from Optometric Weekly, August 8, 1946.

volves exact prepositioning of parts, judgment of the heat of the parts by their color, a deft touch with the very tip of the solder wire and close inspection to see that the right amount of solder has been applied. Operators are paid, over and above their base rate, in proportion to the amount of satisfactory work they do. If they turn out faulty work, which is rejected by the inspectors, they must repair it themselves—during the time when they could be producing more and earning more.

Those employees who had professional care during a period of a year, from whatever source and by whatever method, had gained 5 per cent in earnings over their fellow workmen who had no eye care. Eye care paid them a dividend averaging over seven dollars a month in earnings, while it paid the company a dividend of 5 per cent more production for each dollar of capital investment in the jobs for these people.

Only a minority had eye care. Considerable evidence, which will be discussed here, indicates that a more intensive and aggressive program of eye care would multiply this gain. Such gains from professional eye care are not unique on this job; they occur on many jobs. A similar benefit has been reported by Kephart[1] for women who match hosiery.

## THE FACT FINDING SURVEY

Over a year ago a fact-finding visual survey of this job was conducted in the three departments where this work is done.[2] Similar extensive studies have been made and continue to be made on other jobs in the plant. In this study each solderer was tested on the Ortho-rater for visual skills. Records were collected to classify each worker in terms of average hourly earnings, percentage of work rejected by the inspectors, ratings by the supervisors and the number of injuries (mostly burns) sustained during the preceding year. This broad range of information on job performance was available in whole, or for the most part, on 74 electric solderers.

The analysis of these figures was carried out in the Occupational Research Center at Purdue University by a well established procedure[3] which is applied to all such projects wherever a visual fact-finding system is in use. (*The instrument used for tests was the Ortho-rater.*—H.S.K.) This analysis indicated that certain degrees of proficiency on several of the visual skills were important for successful performance on this job.

The pattern of all the desirable visual characteristics for this job is shown in Figure 1. The background of this pattern in Figure 1 is a chart containing all possible scores on each of the vision tests. For each test that is related to successful performance on this job, a portion of the possible scores is shaded to indicate that they represent undesirable visual traits—traits that are found most frequently among the less satisfactory workers. It will be seen from this pattern that for this particular job near vision requirements are more severe and restrictive than far vision requirements. Such a pattern of visual skills is specific for this job. Other jobs, even close jobs, may have somewhat different visual requirements; some may have very similar requirements.

Solderers who met these requirements were found to be producing more work, to have a smaller proportion of unacceptable work, to be earning more, to be rated higher by their supervisors and to have had fewer accidents in the previous year than those who did not meet these requirements. Subsequent study indicated further that solderers who met the visual requirements had somewhat fewer absences and accidents in the following year, and that more of them were still on the job after a year. All these differences represent important aspects of

success in job performance. The advantage in favor of those who meet the visual standards means more earnings for the employees, more production and lower costs for the company.

### SELECTION OF NEW EMPLOYEES

This indicated pattern of visual skills was adopted as minimum requirements for this job. These particular requirements were reasonable in terms of the available labor, because a majority of solderers then on the job qualified on all these tests. Applicants were required to qualify on all these tests before they were assigned to this job, but with some exceptions that were necessary because of an acute shortage of qualified applicants during this last war year.

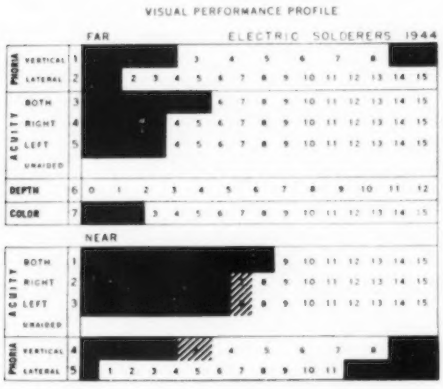



FIG. 1—Minimum visual requirements for electric solderers.

New employees starting this job do not earn at piece rates enough to make up their minimum guaranteed wage. The difference is made up by the company in cash outlay which is charged to learning cost. Some workers "outgrow" this minimum make-up payment in a few weeks; others require minimum make-up payments for months. Of those new employees during the following year who met the minimum visual requirements at the time they were hired, 75 per cent were earning their own pay in less than 10 weeks. Of those who did not meet all the minimum visual requirements, only 40 per cent were earning their own pay in less than ten weeks. This represents a marked difference in cash outlay that cannot be charged to any productive work. Those new employees who met the visual requirements were, on the average, earning more than their minimum guaranteed wage much sooner than those who did not meet the visual requirements.

### REFERRAL FOR EYE CARE

Solderers already on the job who did not meet these requirements were referred for eye care. This referral was made on the basis of their inadequate visual skills. Some others sought professional eye care for reasons of their own, such as ocular discomfort. Some went to the eye clinic operated at the Bausch and Lomb plant for the benefit of employees. Others went to their own optometrist or ophthalmologist in the community. In a few cases, no glasses or change of glasses or treatment was prescribed, presumably because in the doctor's opinion either nothing more needed to be done or nothing more could be done. In most cases, however, the doctors made an attempt towards improving either the comfort or the visual skills of these employees.

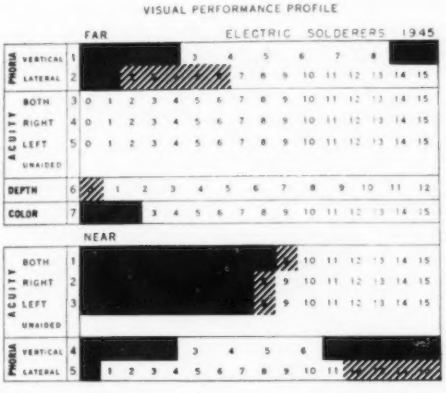



FIG. 2—Revised minimum and optimum visual requirements for electric solderers.

### FOLLOW-UP STUDY

A year later the Ortho-rater department restudied this job and extended the scope of the study to include a follow-up analysis of the year. On the basis of the new data, even more extensive than before, certain modifications were recommended in the pattern of visual requirements. The new basic pattern is shown by the solid shaded area in Figure 2.

In addition to this basic pattern, however, the re-study showed that still further restriction in the range of visual "tolerances" characterized the very superior workers. These additional restrictions are indicated by the cross-hatched areas in Figure 2. A minority of solderers could qualify on all these more severe requirements, but they were among the best producers and highest paid employees. While the revised basic pattern was adopted as minimum requirements, the more critical pattern has been added as optimum requirements for this job.

### EFFECTS OF EYE CARE

During the intervening year, average earnings of the experienced solderers who have stayed on the job increased for several months and then, during reconversion to peace time schedules, decreased. A net loss from beginning to end of the year averaged four cents an hour for the whole group. This change for those who had eye care and for those who did not, is shown in Figure 3. Those who had eye care held their own—they were earning 0.5 cents an hour more at the end of the year than at the beginning. Two cases who had eye care and could meet the new optimum standards at the end of the year had increased their earning 9.5 cents an hour. Those who had not had eye care decreased their earnings 4.1 cents an hour. The few who could, even without eye care, meet the optimum visual standards, increased their earnings only 0.6 cents an hour.

Those who did not have eye care lost on an average nearly eight dollars a month in earning capacity, while those who had eye care increased slightly, and those who also could meet the most exacting visual requirements increased 18 dollars a month.

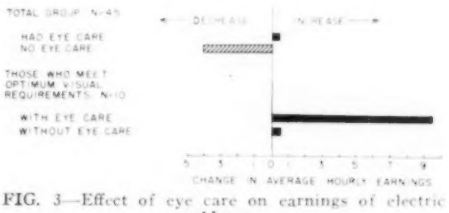



FIG. 3—Effect of eye care on earnings of electric solderers.

### NEED FOR FURTHER EYE CARE

While these are substantial gains in production and earnings due to eye care, additional evidence shows the possibilities of further repeated eye care for these solderers. One cannot expect that professional eye care on one occasion can qualify each employee from then on indefinitely for the visual requirements of any given job. Only a minority of the workers in this group had eye care. Some of them had visual conditions that could not be completely changed. In the following year there were, no doubt, some losses in visual skills with increasing age, a fact that has been demonstrated for all visual skills.[4] At the end of the year, many who had eye care needed further help. Due to a lack of qualified applicants, some new operators had been put on the job in spite of the fact that they did not meet all the visual requirements. At the close of the year there were still marked individual differences in visual performance that were related to production.

Those solderers with more than three months' experience who had best vision for the job were producing and earning

more, as shown in Figure 4. In this figure earnings are given as cents per hour above a constant base rate. Those who did not meet the minimum visual requirements were earning 11.4 cents an

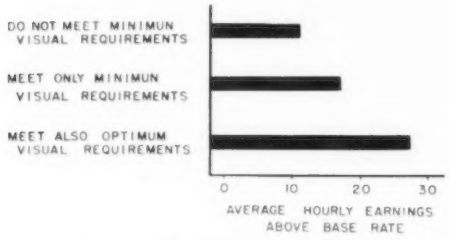



FIG. 4—Earnings in relation to vision of experienced electric solderers.

hour above the base rate. Those who met only the minimum requirements were earning 17.2 cents an hour above the base rate. Those who met the optimum visual requirements were earning 27.6 cents an hour above the base rate—a difference of more than sixteen cents an hour over the first group. The best qualified group visually could turn out more work than the unqualified group with the same amount of equipment and overhead expense, or could turn out as much work with proportionately less capital investment.

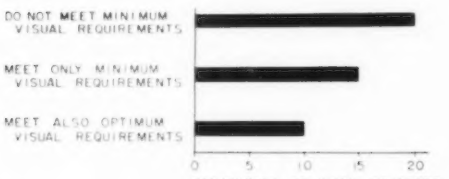



FIG. 5—Quality of work in relation to vision of experienced electric solderers.

Solderers with vision for the job were also better as to quality of work, as shown in Figure 5. Imperfect work is rejected by rigid inspection, has to be repaired and re-inspected, and sometimes scrapped. Experienced workers who did not meet the minimum visual requirements were turning out work of which 20 per cent was rejected; those who met only the minimum requirements were turning out work of which 15 per cent was rejected. Those who met the optimum visual requirements were turning out work of which 10 per cent was rejected—only half as much as the first group. Their total work required 8½ per cent less inspection than the work of the visually unqualified group.

The possible savings yet to be achieved in production, re-inspection and scrap indicate the need for further eye care for this group of employees. These remaining indifferences in production could be reduced if the visual skills of all the solderers could be brought up to a more uniform level of efficiency. The eye care problem is one that cannot be solved permanently in a year; continuing periodic eye care is required to keep these people at peak performance. The economic advantages to employees and company, as shown in the first year, far exceed the cost of such eye care.

## BIBLIOGRAPHY

1. Kephart, N. C.: Analysis of professional eye care and industrial efficiency, Trans. Am. Acad. O. & O., p. 166-170, March-April, 1946.
2. Stump, N. F.: Vision tests predict worker capability, Factory Management and Maintenance, February, 1946.
3. Tiffin, Joseph and Wirt, S. E.: Determining visual standards for industrial jobs by statistical methods, Trans. Am. Acad. O. & O., p. 72-93, Nov.-Dec., 1945.
4. Tiffin, Joseph: Industrial Psychology, Ch. 6, Prentice-Hall, Inc., New York, 1942.

## REPORT OF WILMER INSTITUTE ON "CAUSTIC EYE BURNS"

"Eye burns with lime or other caustic alkaline drugs are among the most serious of injuries. The Wilmer Ophthalmological Institute of John Hopkins University describes the steps to be fol-

lowed in treating such burns: 1. Immediate washing with copious amounts of water. 2. Removal of any residual alkali under the eyelids and instillation of a buffer solution to overcome the alkaline effect. 3. Use of a drug to dilate the pupil. 4. Washing the surface of the eye, after burns with lime, using a 10 per cent solution of ammonium tartrate or lactate. 5. Application of an ointment of sulfonamide or penicillin to prevent secondary infection. 6. Repeated visits to the specialist, so he may take the steps necessary to prevent scarring and adhesions."

---

## CONTINUING BIBLIOGRAPHY ON OCCUPATIONAL EYE PROBLEMS


ANON. *Eyes Right for Better Production.* Modern Industry, May 15, 1945.

BIRREN, FABER. *Color in the Plant.* Factory Management and Maintenance, Vol. 103, p. 2, Feb., 1945.

BRIDGES, C. D. *Job Placement of the Physically Handicapped.* McGraw-Hill, New York, 1946.

BRUCE, G M. *Simple Methods for the Detection of Ocular Malingering.* U. S. Naval M. Bull., Vol. 41, pp. 755-757, May, 1943.

CAPEL, ERNEST H. *Seeing the Job.* Medicine and Industrial Hygiene, July-August, 1945.

CAMPOS, E. *Cataract Produced by Electric Current.* Rev. Brasil de Oftal, Sept., 1944. (Review Arch. Ophthal., Aug., 1946)

CHECHIK-KUNINA. *Treatment of Perforating Wounds and Burns of the Eye with Lysozyme.* Vestnik Oftal., Vol. 24, pp. 42-43, 1945.

COGAN, DAVID G. AND KINSEY, V. E. *Action Spectrum of Keratitis Produced by Ultraviolet Radiation.* Arch. Ophthal., Vol. 35, No. 6, pp. 670-677, June, 1946.

DUNCAN, PERRY E. *Perforating Ocular Injuries.* Indust. Med., Vol. 15, No. 9, pp. 512-514, Sept., 1946.

FEINBERG, RICHARD. *Illumination and Vision Conservation in Industry.* Illuminating Engineering, Vol. 40, p. 5, May, 1945.

FRIEDENWALD, J. S., HUGHES, W. F. JR., and HERMANN, H.: *Acid Burns of the Eye.* Arch Ophthal., Vol. 35, pp. 98-108, Feb., 1946.

GREEAR, JAMES N., *and* MCGAVIC, JOHN S. *Visual Disturbances Associated with Head Injuries.* Arch. Ophthal., Vol. 36, No. 1, pp. 33-53, July, 1946.

HALL, RICHARD. *The Eyes Behind the Goggles.* Nat. Safety News, Sept., 1946.

HANMAN, BERT. *Placing the Handicapped.* Indust. Med., Vol. 15, No. 10, pp. 597-604, Oct., 1946.

HANMAN, BERT, ET AL. *The Worker and His Job.* Symposium at Annual Meeting of Western Assoc. Indust. Physicians and Surgeons, San Francisco, June 30, 1946. Indust. Med., Vol. 15, No. 9, p. 515, Sept., 1946.

HARDY, LEGRAND H.; RAND, GERTRUDE and RITTLER, M. CATHERINE. *Color Vision and Recent Developments in Color Vision Testing.* Arch. Ophthal., Vol. 36, No. 6, pp. 603-614, June, 1946.

KIRSCHBERG, L. S. S. *Depth Perception and Flying Ability.* Arch. Ophthal., Vol. 36, No. 2, pp. 155-170, Aug., 1946.

LEEDKE, HAZEL R. *The Health Program in a Pulp and Paper Mill.* The Paper Industry and Paper World, August, 1946.

MINTON, J. *Occupational Eye Diseases.* Brit. Med., Vol. 1, pp. 211-213, Feb., 1946.

NEW YORK SUPREME COURT. Colwell vs. Hammerlund Mfg. Co., Inc. *Loss of eye; Exposure to acid.* (Compensation granted.) New York Supreme Court, Appellate Div., 3rd Dept., March 12, 1946, New York City.

PERKINS, E. W. *Localization of Intraocular Foreign Bodies.* Bull. U. S. Army M. Dept., Vol. 5, pp. 215-217, Feb., 1946.

ROBBINS, LAURENCE L., ET AL. *Superficial "Burns" of Skin and Eyes from Scattered Cathode Rays.* Radiology, Vol. 46, pp. 1-23, Jan., 1946.

RODIGNA, A. M. *Surgical Treatment of Penetrating Wounds of the Eyeball.* Vestnik Oftal., Vol. 23, pp. 53-55, 1945. (Abs. Quarterly Rev. Ophthal., Vol. 2, No. 2, p. 137, June, 1946.)

ROBISON, C. A. *Some Problems of Lighting.* J. Indiana M. A., Vol. 39, No. 5, pp. 228-231, May, 1946.

SCHILLING, RICHARD. *The Present Work of the Industrial Health Research Board of Great Britain.* Indust. Med., Vol. 15, No. 2, pp. 121-127, Feb., 1946.

SMITH, VIRGINIA M. *Rehabilitation of the Partially Seeing.* Sight Saving Rev., Vol. 16, No. 1, pp. 31-35, Spring, 1946.

STREBEL, G. A. *Color Vision in Industry.* Indust. Med., Vol. 15, pp. 426-436, July, 1946.

TINKER, MILES A. *Illumination Standards.* Am. J. Pub. Health, Vol. 36, No. 9, pp. 963-973, Sept., 1946.

TOWNSEND, JAMES G. *Importance of Industrial Ophthalmology.* Proceedings of 1944 Seminar. N.S.P.B. Pamphlet 430.

WATROUS, R. M. *Some Techniques of Modern Industrial Medicine.* Indust. Med., Vol. 14, pp. 314-318, April, 1945.

WILLIAMS, JOSEPH. *Occupational Eye Disease.* Brit. M. J., Vol. 1, pp. 211-212, Feb., 1946.

WILSON, EUNICE. *Basic Eye Information in the Training of Workers.* Sight Saving Rev., Vol. 16, No. 1, pp. 12-20, Spring, 1946.

WOODARD, L. K., JR. *Electric Ophthalmia.* U. S. Med. Bull., Vol. 46, pp. 247-248, Feb., 1946.

WOOLFFLIN, E. *A New Apparatus for Testing Stereoscopic Vision: Preliminary Communication.* Ophthalmologica, Vol. 110, pp. 212-218, Sept.-Oct., 1945. (Review-Quarterly Review of Ophthal., Vol., 2, No. 2, p. 134, June, 1946.)

# COUNCIL MINUTES

## FIFTY-FIRST ANNUAL SESSION

OCTOBER 12 AND 16, 1946
Palmer House, Chicago

Present:

Gordon B. New, Alan C. Woods, Edmund B. Spaeth, Brittain F. Payne, Ralph I. Lloyd, Lawrence T. Post, William L. Benedict, O. E. Van Alyea, Algernon B. Reese, Dean M. Lierle, A. D. Ruedemann, Lawrence R. Boies, Erling W. Hansen, Derrick Vail, Carl H. McCaskey, James M. Robb, C. D. Blassingame, Frank E. Burch.

The meeting was called to order at 9:30 a.m. by Dr. Gordon B. New, President.

I

The Executive Secretary, Dr. W. L. Benedict, submitted the Secretary's report as follows:

1. Fellowship as of October 1, 1946:

| | |
|---|---|
| Juniors | 3,026 |
| Seniors | 239 |
| Life | 303 |
| Honorary | 19 |
| | 3,587 |

2. Elevations to Senior Fellowship on January 1, 1947:

Albright, Paul Morris, Corona, Calif.
Bentley, Neil, Detroit, Mich.
Berens, Conrad, New York, N. Y.
Blickenstaff, A. J., Peoria, Ill.
Charles, Joseph W., St. Louis, Mo.
Douglas, Frederick A., La Crosse, Wis.
Fairchild, Nora M., Los Angeles, Calif.
Forbes, Sherman B., Tampa, Fla.
Fust, John H., Erie, Pa.
Gailey, W. W., Bloomington, Ill.
Goux, Raymond S., Detroit, Mich.
Hardesty, John F., St. Louis, Mo.
Hurst, V. Reeves, Longview, Tex.
Kauffman, A. B., Syracuse, N. Y.
Kemler, Joseph I., Baltimore, Md.
Kully, B. M., Los Angeles, Calif.
LaRue, C. L., Shreveport, La.
Loftus, John Edward, Philadelphia, Pa.
March, Clara A., Buffalo, N. Y.
Peeler, C. N., Charlotte, N. C.
Russell, J. A. Merle, Erie, Pa.
Siegel, Francis X., Cincinnati, Ohio
Steinfeld, A. L., Toledo, Ohio
Sulzman, Frank M., Troy, N. Y.
Todd, Grover C., Pittsburgh, Pa.
Welch, Robert S. G., Annapolis, Md.
Winter, George E., Jackson, Mich.

3. Elevations to Life Membership on January 1, 1947:

Baum, H. L., Denver, Colo.
Bernatz, C. F., Pittsburgh, Pa.
Binger, H. E., St. Paul, Minn.
Binkley, Roy S., Dayton, Ohio
Blair, W. W., Pittsburgh, Pa.
Dean, Walter, Louisville, Ky.
Donoher, Wm. D., Los Angeles, Calif.
Dulaney, Octavus, Louisville, Ky.
Farnsworth, M. A., La Junta, Colo.
Judd, H. B., New York, N. Y.
Kaufman, A. Spencer, Philadelphia, Pa.
McKee, Claude W., Greensburg, Pa.
McKennan, James W., Washington, Pa.
Markel, J. Clyde, Pittsburgh, Pa.
Merrill, H. G., San Diego, Calif.
Naftzger, J. B., Hollywood, Calif.
Orcutt, D. C., Chicago, Ill.
Orton, Henry B., Newark, N. J.
Patterson, Ellen J., Pittsburgh, Pa.
Pitkin, C. E., Cleveland, Ohio
Sanderson, David D., Lincoln, Nebr.
Sedgwick, O. W., Ionia, Mich.
Shahan, W. E., St. Louis, Mo.
Shellman, John L., Pacific Palisades, Calif.
Shoemaker, S. A., Orlando, Fla.
Shuman, G. H., Pittsburgh, Pa.
Stueber, Paul J., Lima, Ohio
Taylor, M. C., Pittsburgh, Pa.
Tobey, George L., Jr., Boston, Mass.
Tooker, C. W., St. Louis, Mo.
Weisser, E. A., Pittsburgh, Pa.
Whitaker, Joel, Indianapolis, Ind.
Zemer, S. G., Lincoln, Nebr.

4. Deaths reported since last Council Meeting:

Agan, William B., Brooklyn, N. Y.
Andrews, Albert H., Chicago, Ill.
Barlow, Aaron, Philadelphia, Pa.
Blum, Henry N., New Orleans, La.
Braswell, James C., Tulsa, Okla.
Bruno, Joseph M. L., Brooklyn, N. Y.
Butler, Robert H., Bellefontaine, Ohio

Some items are omitted from this report. Complete minutes are on file in the office of the Executive Secretary-Treasurer.

Carmody, Thomas E., Denver, Colo.
Carson, W. E., Pittsburgh, Pa.
Clarke, George L., Detroit, Mich.
Crockett, R. L., Oneida, N. Y.
Clay, Grady E., Atlanta, Ga.
Comee, W. C., Green Bay, Wis.
Curtin, John F., Minneapolis, Minn.
Dyer, Benjamin C., Manhattan, Kans.
Eagleton, Wells P., Newark, N. J.
Elliot, R. H., London, England
England, Welch, Parkersburg, W. Va.
Foster, Hal, Kansas City, Mo.
Friesner, Isidore, Katonah, N. Y.
Frost, Albert D., Columbus, Ohio
Fullenwider, C. M., Muskogee, Okla.
Geiger, C. W., Kankakee, Ill.
Griffith, Charles M., New York, N. Y.
Hall, G. C., Louisville, Ky.
Herriman, L. L., Pueblo, Colo.
Holt, E. Eugene, Jr., Portland, Me.
Howe, Spencer S., Bellingham, Wash.
Jackson, Rufus, Baton Rouge, La.
Kennon, W. G., Nashville, Tenn.
Keys, M. J., Victoria, B. C., Canada
Kistler, O. F., Wilkes-Barre, Pa.
Klinedinst, John F., York, Pa.
McGregor, D'Arcy, Buffalo, N. Y.
MacKenzie, George W., Philadelphia, Pa.
Moore, G. A., Palmer, Mass.
Moore, Robert R., Little Rock, Ark.
O'Connor, Frederick J., Syracuse, N. Y.
Pettee, Howard W., Detroit, Mich.
Poser, Max, Rochester, N. Y.
Shoemaker, Ferdinand, Kansas City, Mo.
Shreve, Owen M., Erie, Pa.
Stewart, Marshall Bruce, Dormansville, N. Y.
Stewart, T. M., Cincinnati, Ohio
Stieren, Edward, Pittsburgh, Pa.
Tangeman, H. F., Cincinnati, Ohio
Terry, Theodore L., Boston, Mass.
Toms, S. W. S., Nyack, N. Y.
von der Heydt, Robert, Chicago, Ill.
White, James W., New York, N. Y.

5. The Program Committee met in Chicago on January 5 and 6, 1946. Plans were made for the annual convention to be held October 13 to 18 at the Palmer House. In anticipation of a large attendance, changes in the type of program usually presented were deemed necessary. The facilities of the hotel for meeting places and instruction rooms made it possible to use assembly halls and class rooms all day. The Committee made the following arrangements:

a. The Council to meet on Saturday preceding the opening of the convention, and the previous custom of holding Council meetings in the morning of each day to be discontinued. All business not completed on Saturday to be taken up at a luncheon meeting on Wednesday. The business meeting to be held Wednesday evening.

b. All scientific assemblies to be held in the Grand Ballroom, otolaryngologic in the morning and ophthalmologic in the afternoon.

c. Instruction courses to run all day—ophthalmologic in the mornings and otolaryngologic in the afternoons.

d. Scientific papers and motion pictures on scientific program to be given as continuous program alternating in sequence.

e. Scientific sessions to open Monday morning and close Friday noon.

f. Instruction courses to open Monday noon and close Friday noon.

g. Dr. George Morrison Coates was nominated as Guest of Honor.

h. A special program by the Committee on the Conservation of Hearing to be presented at 2:30 p.m. on Sunday. Programs by the Committee on Industrial Ophthalmology and the American Orthoptic Council and American Association of Orthoptic Technicians to be presented at 8 o'clock Sunday evening.

i. It was suggested that the Academy sponsor a Wherry Memorial Lecture each year and that the expenses of this lecture be defrayed by the Wherry Memorial Fund, since the original plan for the income from the Wherry Memorial Fund to pay the application fee and dues of an otolaryngologist each year has not been practicable for numerous reasons. It was moved that a mail vote be sent to the Council asking that the original action of the Council in regard to the Wherry Memorial Fund be rescinded and that the Wherry Memorial Lecture be set up as outlined above. The vote was unanimous in favor of the motion.

j. Scientific exhibit to be held in a portion of the Red Lacquer Room, the remainder of the room to be used by the hotel as a restaurant.

k. The Pan-American Congress of Otolaryngology will open Thursday evening, October 17th, at 7:30 with a formal dinner. The first scientific session will be held jointly with the Academy's otolaryngologic Scientific Session on Friday morning.

l. An editorial committee composed of Dr. A. W. Proetz, chairman, Dr. G. M. Coates,

# COUNCIL MINUTES

## FIFTY-FIRST ANNUAL SESSION

OCTOBER 12 AND 16, 1946
Palmer House, Chicago

Present:

Gordon B. New, Alan C. Woods, Edmund B. Spaeth, Brittain F. Payne, Ralph I. Lloyd, Lawrence T. Post, William L. Benedict, O. E. Van Alyea, Algernon B. Reese, Dean M. Lierle, A. D. Ruedemann, Lawrence R. Boies, Erling W. Hansen, Derrick Vail, Carl H. McCaskey, James M. Robb, C. D. Blassingame, Frank E. Burch.

The meeting was called to order at 9:30 a.m. by Dr. Gordon B. New, President.

I

The Executive Secretary, Dr. W. L. Benedict, submitted the Secretary's report as follows:

1. Fellowship as of October 1, 1946:

| | |
|---|---|
| Juniors | 3,026 |
| Seniors | 239 |
| Life | 303 |
| Honorary | 19 |
| | 3,587 |

2. Elevations to Senior Fellowship on January 1, 1947:

Albright, Paul Morris, Corona, Calif.
Bentley, Neil, Detroit, Mich.
Berens, Conrad, New York, N. Y.
Blickenstaff, A. J., Peoria, Ill.
Charles, Joseph W., St. Louis, Mo.
Douglas, Frederick A., La Crosse, Wis.
Fairchild, Nora M., Los Angeles, Calif.
Forbes, Sherman B., Tampa, Fla.
Fust, John H., Erie, Pa.
Gailey, W. W., Bloomington, Ill.
Goux, Raymond S., Detroit, Mich.
Hardesty, John F., St. Louis, Mo.
Hurst, V. Reeves, Longview, Tex.
Kauffman, A. B., Syracuse, N. Y.
Kemler, Joseph I., Baltimore, Md.
Kully, B. M., Los Angeles, Calif.
LaRue, C. L., Shreveport, La.
Loftus, John Edward, Philadelphia, Pa.
March, Clara A., Buffalo, N. Y.
Peeler, C. N., Charlotte, N. C.
Russell, J. A. Merle, Erie, Pa.
Siegel, Francis X., Cincinnati, Ohio
Steinfeld, A. L., Toledo, Ohio
Sulzman, Frank M., Troy, N. Y.
Todd, Grover C., Pittsburgh, Pa.
Welch, Robert S. G., Annapolis, Md.
Winter, George E., Jackson, Mich.

3. Elevations to Life Membership on January 1, 1947:

Baum, H. L., Denver, Colo.
Bernatz, C. F., Pittsburgh, Pa.
Binger, H. E., St. Paul, Minn.
Binkley, Roy S., Dayton, Ohio
Blair, W. W., Pittsburgh, Pa.
Dean, Walter, Louisville, Ky.
Donoher, Wm. D., Los Angeles, Calif.
Dulaney, Octavus, Louisville, Ky.
Farnsworth, M. A., La Junta, Colo.
Judd, H. B., New York, N. Y.
Kaufman, A. Spencer, Philadelphia, Pa.
McKee, Claude W., Greensburg, Pa.
McKennan, James W., Washington, Pa.
Markel, J. Clyde, Pittsburgh, Pa.
Merrill, H. G., San Diego, Calif.
Naftzger, J. B., Hollywood, Calif.
Orcutt, D. C., Chicago, Ill.
Orton, Henry B., Newark, N. J.
Patterson, Ellen J., Pittsburgh, Pa.
Pitkin, C. E., Cleveland, Ohio
Sanderson, David D., Lincoln, Nebr.
Sedgwick, O. W., Ionia, Mich.
Shahan, W. E., St. Louis, Mo.
Shellman, John L., Pacific Palisades, Calif.
Shoemaker, S. A., Orlando, Fla.
Shuman, G. H., Pittsburgh, Pa.
Stueber, Paul J., Lima, Ohio
Taylor, M. C., Pittsburgh, Pa.
Tobey, George L., Jr., Boston, Mass.
Tooker, C. W., St. Louis, Mo.
Weisser, E. A., Pittsburgh, Pa.
Whitaker, Joel, Indianapolis, Ind.
Zemer, S. G., Lincoln, Nebr.

4. Deaths reported since last Council Meeting:

Agan, William B., Brooklyn, N. Y.
Andrews, Albert H., Chicago, Ill.
Barlow, Aaron, Philadelphia, Pa.
Blum, Henry N., New Orleans, La.
Braswell, James C., Tulsa, Okla.
Bruno, Joseph M. L., Brooklyn, N. Y.
Butler, Robert H., Bellefontaine, Ohio

Some items are omitted from this report. Complete minutes are on file in the office of the Executive Secretary-Treasurer.

Carmody, Thomas E., Denver, Colo.
Carson, W. E., Pittsburgh, Pa.,
Clarke, George L., Detroit, Mich.
Crockett, R. L., Oneida, N. Y.
Clay, Grady E., Atlanta, Ga.
Comee, W. C., Green Bay, Wis.
Curtin, John F., Minneapolis, Minn.
Dyer, Benjamin C., Manhattan, Kans.
Eagleton, Wells P., Newark, N. J.
Elliot, R. H., London, England
England, Welch, Parkersburg, W. Va.
Foster, Hal, Kansas City, Mo.
Friesner, Isidore, Katonah, N. Y.
Frost, Albert D., Columbus, Ohio
Fullenwider, C. M., Muskogee, Okla.
Geiger, C. W., Kankakee, Ill.
Griffith, Charles M., New York, N. Y.
Hall, G. C., Louisville, Ky.
Herriman, L. L., Pueblo, Colo.
Holt, E. Eugene, Jr., Portland, Me.
Howe, Spencer S., Bellingham, Wash.
Jackson, Rufus, Baton Rouge, La.
Kennon, W. G., Nashville, Tenn.
Keys, M. J., Victoria, B. C., Canada
Kistler, O. F., Wilkes-Barre, Pa.
Klinedinst, John F., York, Pa.
McGregor, D'Arcy, Buffalo, N. Y.
MacKenzie, George W., Philadelphia, Pa.
Moore, G. A., Palmer, Mass.
Moore, Robert B., Little Rock, Ark.
O'Connor, Frederick J., Syracuse, N. Y.
Peirce, Howard W., Detroit, Mich.
Poser, Max, Rochester, N. Y.
Shoemaker, Ferdinand, Kansas City, Mo.
Shreve, Owen M., Erie, Pa.
Stewart, Marshall Bruce, Dormansville, N. Y.
Stewart, T. M., Cincinnati, Ohio
Stieren, Edward, Pittsburgh, Pa.
Tangeman, H. F., Cincinnati, Ohio
Terry, Theodore L., Boston, Mass.
Toms, S. W. S., Nyack, N. Y.
von der Heydt, Robert, Chicago, Ill.
White, James W., New York, N. Y.

5. The Program Committee met in Chicago on January 5 and 6, 1946. Plans were made for the annual convention to be held October 13 to 18 at the Palmer House. In anticipation of a large attendance, changes in the type of program usually presented were deemed necessary. The facilities of the hotel for meeting places and instruction rooms made it possible to use assembly halls and class rooms all day. The Committee made the following arrangements:

a. The Council to meet on Saturday preceding the opening of the convention, and the previous custom of holding Council meetings in the morning of each day to be discontinued. All business not completed on Saturday to be taken up at a luncheon meeting on Wednesday. The business meeting to be held Wednesday evening.

b. All scientific assemblies to be held in the Grand Ballroom, otolaryngologic in the morning and ophthalmologic in the afternoon.

c. Instruction courses to run all day—ophthalmologic in the mornings and otolaryngologic in the afternoons.

d. Scientific papers and motion pictures on scientific program to be given as continuous program alternating in sequence.

e. Scientific sessions to open Monday morning and close Friday noon.

f. Instruction courses to open Monday noon and close Friday noon.

g. Dr. George Morrison Coates was nominated as Guest of Honor.

h. A special program by the Committee on the Conservation of Hearing to be presented at 2:30 p.m. on Sunday. Programs by the Committee on Industrial Ophthalmology and the American Orthoptic Council and American Association of Orthoptic Technicians to be presented at 8 o'clock Sunday evening.

i. It was suggested that the Academy sponsor a Wherry Memorial Lecture each year and that the expenses of this lecture be defrayed by the Wherry Memorial Fund, since the original plan for the income from the Wherry Memorial Fund to pay the application fee and dues of an otolaryngologist each year has not been practicable for numerous reasons. It was moved that a mail vote be sent to the Council asking that the original action of the Council in regard to the Wherry Memorial Fund be rescinded and that the Wherry Memorial Lecture be set up as outlined above. The vote was unanimous in favor of the motion.

j. Scientific exhibit to be held in a portion of the Red Lacquer Room, the remainder of the room to be used by the hotel as a restaurant.

k. The Pan-American Congress of Otolaryngology will open Thursday evening, October 17th, at 7:30 with a formal dinner. The first scientific session will be held jointly with the Academy's otolaryngologic Scientific Session on Friday morning.

l. An editorial committee composed of Dr. A. W. Proetz, chairman, Dr. G. M. Coates,

and Dr. Theodore E. Walsh was appointed to edit the papers presented at the Congress. All papers will be published in the TRANSACTIONS.

6. The Board of Secretaries met in Chicago on June 15 and 16, 1946, for completion of the details of the program. The chairman of the Pan-American Congess of Otolaryngology met with the Program Committee and the Board of Secretaries and cooperated in the arrangements for the inaugural meeting of this society.

7. Home Study Courses: Registration, Income and Expense:

| *Year* | *Registration* | | | *Income* | *(Calendar year)* | *Expense* |
|---|---|---|---|---|---|---|
| 1940-1941 | Ophthalmology | 301 | | | | |
| | Otolaryngology | 114 | 415 | $4,570.00 | | $1,324.03 |
| 1941-1942 | Ophthalmology | 238 | | | | |
| | Otolaryngology | 117 | 355 | 3,690.00 | | 3,081.85 |
| 1942-1943 | Ophthalmology | 163 | | | | |
| | Otolaryngology | 81 | 244 | 2,510.00 | | 2,191.95 |
| 1943-1944 | Ophthalmology | 126 | | | | |
| | Otolaryngology | 74 | 200 | 1,995.00 | | 3,373.45 |
| 1944-1945 | Ophthalmology | 181 | | | | |
| | Otolaryngology | 88 | 269 | 2,765.00 | | 3,083.95 |
| 1945-1946 | Ophthalmology | 290 | | | | |
| | Otolaryngology | 112 | 402 | 3,971.00 | | 3,181.87 |
| 1946-1947 | Ophthalmology | 288 | | | | |
| (to Sept. 19) | Otolaryngology | 143 | 431 | 4,505.06 | | 3,059.95 |
| | | | 2,316 | $24,006.06 | | $19,297.05 |

Income in excess of expense: $4,709.01

II

The Treasurer, Dr. W. L. Benedict, submitted the audit for 1945, made by Shannon & Byers, Certified Public Accountants, and the financial status of the Academy as of August 31, 1946 as follows:

April 12, 1946

The American Academy of
Ophthalmology and Otolaryngology
Rochester, Minnesota

Gentlemen:

As requested, we have examined the accounts and records of The American Academy of Ophthalmology and Otolaryngology, an unincorporated association, for the calendar year ended December 31, 1945, and have prepared therefrom the accompanying Balance Sheet, Statement of Operations, and Analysis of Net Worth, together with a schedule showing a list of securities owned on December 31, 1945.

We have also reviewed the system of internal control and accounting procedures, and without making a detailed audit of cash receipts and disbursements and other transactions, have examined or tested accounting records and other supporting evidence, by methods and to the extent we deemed appropriate.

Cash on deposit at the various banks was verified by communicating with the depositories, and the petty cash fund was verified by actual count.

Membership dues receivable, amounting to $1,135.00, represent delinquent membership dues for 1945 and prior years and were verified from the records only.

Investments in stocks and bonds had a market value of $150,052.70 on December 31, 1945, as shown in Schedule No. 1.

Securities in the Research Fund, which are held for safekeeping by the Trust Department of the First National Bank, Minneapolis, Minnesota, were verified by communicating with the custodian of the securities. U. S. Savings bonds in the Wherry Memorial Fund are held by the Executive Secretary-Treasurer and were inspected by us. Interest earned on bonds since the dates on which the last payments became due has not been accrued.

Furniture, fixtures and equipment amounting to $3,699.89 represents equipment acquired by the Academy over a period of years. Equipment acquired in years prior to September 1, 1942, which had been charged to operating expenses as purchased, was inventoried and appraised at a market value of $1,420.28 on January 1, 1944 and set up on the books for that amount. Equipment purchased since Sept. 1, 1942, amounting to $2,279.61, was for the most part verified through an inspection of supporting vouchers and of the latter amount, $465.06 was purchased during the current year. Depreciation has been deducted and charged to operations at a rate that appears to be adequate.

Unexpired insurance premiums totaling $435.19 were computed by us. Prepaid bond interest of $12.24 represents accrued interest on bond purchases.

Current liabilities of $2,306.57 consist of accrued expenses totaling $1,654.97 for unpaid December printing and other minor items, federal witholding tax deductions totaling $396.60, and membership dues paid in advance, totaling $255.00.

Upon examining the insurance policies in force, we found Owners' & Tenants' Public Liability insurance in the amount of $25,000.00/$25,000.00 coverage, and Workmen's Compensation subject to statutory amounts. The Executive Secretary-Treasurer and Chairman of the Finance Committee are bonded for $25,000.00 each, while office employees are bonded for $1,000.00 each. Furniture, fixtures and equipment are insured for $4,000.00 fire and extended coverage.

In our opinion, the accompanying balance sheet and related statement of operations and net worth present fairly the position of the Association at December 31, 1945, and the results of its operations for the year, in conformity with generally accepted accounting principles applied on a basis consistent with that of the preceding year.

Respectfully submitted,

SHANNON & BYERS

Certified Public Accountants
By L. J. Byers, C.P.A.

*EXHIBIT "A"*

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

*Balance Sheet as at December 31, 1945*

ASSETS

| | | |
|---|---|---|
| *Current Assets*: | | |
| Cash, First National Bank, Rochester, Minn | $ 552.75 | |
| Cash, First National Bank, Minneapolis, Minn. | 6,421.08 | |
| Cash, Bank of Montreal, Toronto, Ont. | 441.90 | |
| Cash, petty cash fund | 25.00 | |
| Membership dues receivable (Delinquent) | 1,135.00 | |
| Total current assets | | $ 8,575.73 |
| *Investments*: (Schedule 1) | | |
| Stocks and bonds (at market value) | | 150,052.70 |
| *Furniture, Fixtures and Equipment* | 3,699.89 | |
| Less reserve for depreciation | 756.44 | 2,943.45 |
| *Deferred Charges*: | | |
| Prepaid insurance | 435.19 | |
| Prepaid bond interest | 12.24 | |
| | | 447.43 |
| Total Assets | | $162,019.31 |

LIABILITIES AND NET WORTH

*Current Liabilities*:

| | | |
|---|---|---|
| Accrued expenses | 1,654.97 | |
| Accrued withholding taxes | 396.60 | |
| Deferred income, membership dues | 255.00 | |
| Total current liabilities | | 2,306.57 |
| *Net Worth* (Exhibit "C") | | 159,712.74 |
| Total Liabilities and net worth | | $162,019.31 |

*EXHIBIT "B"*

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

*Statement of Operations*
*Year Ended December 31, 1945*

*Revenues*:

| | | |
|---|---|---|
| Membership dues | | $17,920.00 |
| Membership applications | | 2,244.00 |
| Atlases | | 1,140.40 |
| Certificates | | 27.00 |
| Transactions | | 18,986.21 |
| Home Study Courses | | 3,971.00 |
| Convention | | 1,254.42 |
| Income from securities: | | |
| Interest, Research Fund | $2,129.20 | |
| Dividends, Research Fund | 1,435.25 | |
| | 3,564.45 | |
| Interest, Wherry Memorial Fund | 115.00 | 3,679.45 |
| Sundry receipts | | 73.25 |
| Total Revenue | | $49,295.73 |

*Operating Expenses*:

| | | |
|---|---|---|
| General administrative | 20,968.54 | |
| Transactions | 13,844.53 | |
| Home Study Courses | 3,211.00 | |
| Research | 892.60 | |
| Conventions and meetings | 4,477.87 | |
| Total Operating Expenses | | 43,394.54 |
| *Net Income* | | $5,901.19 |

*EXHIBIT "C"*

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

*Analysis of Net Worth Account for Period Ended December 31, 1945*

| | | |
|---|---|---|
| *Net Worth, December 31, 1944* | | $146,305.78 |
| *Additions*: | | |
| Net income, year ended December 31, 1945 | $5,901.19 | |
| Increase in market value of securities from 1/1/45 to 12/31/45 (*) | 7,505.77 | |
| Total additions | | 13,406.96 |
| *Net Worth, December 31, 1945* | | $159,712.74 |

(*) *Increase in Market Value of Securities*:

| | *Cost* | *Market* | *Increase Decrease* |
|---|---|---|---|
| 12/31/44 | $142,043.62 | $130,066.93 | $11,976.69 |
| 12/31/45 (Schedule 1) | 154,523.62 | 150,052.70 | 4,470.92 |
| Difference | $ 12,480.00 | $ 19,985.77 | $ 7,505.77 |

SCHEDULE No 1

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

## Securities Owned, December 31, 1945

RESEARCH FUND

| BONDS | RATE | MATURITY | PAR VALUE | COST | MARKET VALUE |
|---|---|---|---|---|---|
| Brooklyn Edison Co., Inc. Cons. | 3¼ | 5/15/66 | $ 3,000.00 | $ 3,045.00 | $ 3,150.00 |
| Citizens Building Co. of Cleveland—1st Leasehold Sinking Fund | 6 | 1/1/46 | 5,000.00 | 5,000.00 | ..... |
| Dominion of Canada Conv. Loan, Ser. R8 | 4½ | 11/1/58 | 5,000.00 | 4,950.00 | (*) 4,850.00 |
| Dominion of Canada, 8th Victory Loan P5 | 3 | 10/1/63 | 2,500.00 | 2,500.00 | (*) 2,350.00 |
| Union Pacific R. R. Co., 35-yr. Debenture | 3½ | 5/1/71 | 6,000.00 | 5,947.50 | 6,180.00 |
| Van Sweringen Co., Certificate of Indebtedness, Series 1925 | 6 | 12/31/48 | 6,000.00 | 6,031.33 | 1,320.00 |
| U.S.A. Savings, Series "D" | ... | 4/1/49 | 10,000.00 | 7,500.00 | 8,700.00 |
| U.S.A. Savings, Series "D" | ... | 3/1/50 | 10,000.00 | 7,500.00 | 8,500.00 |
| U.S.A. Savings, Series "F" | ... | 12/1/55 | 13,500.00 | 9,990.00 | 10,111.50 |
| U.S.A. Treasury 1967-72 | 2½ | 12/15/71 | 10,000.00 | 10,000.00 | 10,100.00 |
| U.S.A. Savings, Series "G" | 2½ | 8/1/53 | 10,000.00 | 10,000.00 | 10,000.00 |
| U.S.A. Savings, Series "G" | 2½ | 6/1/55 | 4,000.00 | 4,000.00 | 4,000.00 |
| U.S.A. Savings, Series "G" | 2½ | 3/1/56 | 6,000.00 | 6,000.00 | 6,000.00 |
| U.S.A. Savings, Series "G" | 2½ | 5/1/56 | 8,000.00 | 8,000.00 | 8,000.00 |
| U.S.A. Savings, Series "G" | 2½ | 7/1/56 | 2,000.00 | 2,000.00 | 2,000.00 |
| U.S.A. Savings, Series "G" | 2½ | 11/1/56 | 25,000.00 | 25,000.00 | 25,000.00 |
| U.S.A. Savings, Series "G" | 2½ | 1/1/57 | 2,000.00 | 2,000.00 | 2,000.00 |
| Total Bonds | | | | $119,463.83 | $112,261.50 |

| STOCKS | KIND | PAR VALUE | SHARES | | |
|---|---|---|---|---|---|
| Citizens Bldg. Co., Cleveland | Shares | No Par | 50 | None | ..... |
| Commonwealth Edison Co. | Common | $25.00 | 100 | 2,619.00 | 3,300.00 |
| E. I. du Pont de Nemours & Co | Common | 20.00 | 15 | 2,347.31 | 2,790.00 |
| Louisville Gas & Electric Co. | 5% Prf. | 25.00 | 200 | 5,450.00 | 6,000.00 |
| Massachusetts Investors Trust | | 1.00 | 600 | 17,513.00 | 17,400.00 |
| Ophthalmic Publishing Co. | Capital | 20.00 | 5 | None | ..... |
| Philip Morris & Co. Ltd. | Common | 5.00 | 60 | 2,530.48 | 3,900.00 |
| Total Stocks | | | | $ 30,459.79 | $ 33,390.00 |
| Total Research Fund | | | (A) | $149,923.79 | $145,651.50 |

WHERRY MEMORIAL FUND

| | | | | | |
|---|---|---|---|---|---|
| U. S. A. Savings, Series G | | | (B) | 4,600.00 | 4,401.20 |
| Total | | | | $154,523.62 | $150,052.70 |

(*) U. S. Dollars

(A) Held for safekeeping by Trust Dept. of First National Bank, Minneapolis, Minn.

(B) Held by Dr. W. L. Benedict, Executive Secretary-Treasurer, Rochester, Minn.

*Statement of Operations for the First Eight Months of the Current Fiscal Year:*

*EXHIBIT "A"*

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

*Balance Sheet as of August 31, 1946*

ASSETS

| | | |
|---|---|---|
| *Current Assets:* | | |
| Cash, First Nat'l Bank, Rochester | $ 17,940.18 | |
| Cash, Bank of Montreal | 1,191.55 | |
| Cash, First Nat'l Bank, Minneapolis | 2,971.26 | |
| Checks for Collection | 4.62 | |
| Petty Cash Fund | 25.00 | |
| Membership Dues Receivable | 433.00 | |
| Total Current Assets | | $ 22,565.61 |
| *Investments:* | | |
| Research Fund | 171,445.64 | |
| Wherry Memorial Fund | 4,600.00 | |
| | 176,045.64 | |
| Less: Reserve for Mkt. Fluctuations | 2,352,42 | |
| Total Investments | | 173,693.22 |
| *Fixed Assets:* | | |
| Furniture & Fixtures | 4,327.52 | |
| Less: Reserve for Depreciation | 996.44 | |
| Total Fixed Assets | | 3,331.08 |
| Prepaid Insurance | | 315.19 |
| Prepaid Postage | | 200.00 |
| Prepaid Interest: | | 12.24 |
| Total Assets | | $200,117.34 |

LIABILITIES

| | | |
|---|---|---|
| *Current Liabilities:* | | |
| Accrued Withholding Taxes | $ 252.82 | |
| Deferred Income: Membership Dues | 260.00 | |
| *Net Worth:* | | |
| Balance, January 1, 1946 | 161,831.24 | |
| Excess, Income over Expenses, Year to Date | 37,773.28 | |
| Total | | 199,604.52 |
| Total Liabilities | | $200,117.34 |

*EXHIBIT "B"*

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

*Statement of Income and Expenses for Month Ending August 31, 1946 and Year to Date*

| | August 1946 | 1/1/46 to 8/31/46 |
|---|---|---|
| *Revenues:* | | |
| Membership Dues | $ 129.00 | $18,304.00 |
| Membership Applications | 544.00 | 3,635.00 |
| Atlases | 419.38 | 2,259.63 |
| Certificates | | 8.00 |
| Transactions | 287.72 | 16,935.48 |
| Home Study Courses | 1,460.06 | 3,905.06 |
| Convention | 70.95 | 12,072.30 |
| Other Income | 15.00 | 77.03 |
| Rent | | 50.00 |
| Income from Securities | | |
| Research Fund: | | |
| Interest | 125.00 | 1,336.74 |
| Dividends | 35.00 | 648.00 |
| Profit, Securities Sold | | 1,104.01 |
| Wherry Memorial Fund: | | |
| Interest | 50.00 | 107.50 |
| Total Revenues | $3,136.11 | $60,442.75 |
| *Operating Expenses:* | | |
| General Administrative | $1,177.06 | $11,736.05 |
| Transactions | | 5,541.46 |
| Home Study Courses | 581.98 | 2,982.87 |
| Research | | 12.75 |
| Convention and Meetings | 481.05 | 2,421.84 |
| Total Operating Expenses | $2,240.09 | $22,669.47 |
| Excess Income Over Expenses | $ 896.02 | $37,773.28 |

## American Academy of Ophthalmology and Otolaryngology

## Securities Owned, August 31, 1946

RESEARCH FUND

| BONDS | RATE | MATURITY | PAR VALUE | COST |
|---|---|---|---|---|
| Brooklyn Edison Co., Inc. | 3¼ | 5/15/66 | $ 3,000.00 | $ 3,045.00 |
| Canada, Dominion of — 1931 Conv. Loan | 4½ | 11/1/58 | 5,000.00 | 4,950.00 |
| Canada, Dominion of — Series P5 | 3 | 10/1/63 | 2,500.00 | 2,500.00 |
| Citizens Bldg. Co. of Cleveland, 1st Mtg. | 6 | 1/1/46 | 5,000.00 | 5,000.00 |
| U. S. Savings Bonds — Series D | | 4/1/49 | 10,000.00 | 7,500.00 |
| U. S. Savings Bonds — Series D | | 3/1/50 | 10,000.00 | 7,500.00 |
| U. S. Savings Bonds — Series F | 2½ | 12/1/55 | 13,500.00 | 9,990.00 |
| U. S. Savings Bonds — Series G | 2½ | 8/1/53 | 10,000.00 | 10,000.00 |
| U. S. Savings Bonds — Series G | 2½ | 6/1/55 | 4,000.00 | 4,000.00 |
| U. S. Savings Bonds — Series G | 2½ | 3/1/56 | 6,000.00 | 6,000.00 |
| U. S. Savings Bonds — Series G | 2½ | 5/1/56 | 8,000.00 | 8,000.00 |
| U. S. Savings Bonds — Series G | 2½ | 7/1/56 | 2,000.00 | 2,000.00 |
| U. S. Savings Bonds — Series G | 2½ | 11/1/56 | 25,000.00 | 25,000.00 |
| U. S. Savings Bonds — Series G | 2½ | 1/1/57 | 2,000.00 | 2,000.00 |
| U. S. Savings Bonds — Series | 2½ | 12/15/72 | 10,000.00 | 10,000.00 |
| U. S. Savings Bonds — Series G | 2½ | 7/1/58 | 30,000.00 | 30,000.00 |
| Van Sweringen Co., Cert. of Indebtedness | 6 | 12/31/48 | 6,000.00 | 6,031.33 |
| Total Bonds | | | | $143,516.33 |

| STOCKS | KIND | SHARES | |
|---|---|---|---|
| Citizens Bldg. Co. of Cleveland | | 50 | None |
| Commonwealth Edison Co. | Common | 100 | $ 2,619.00 |
| DuPont de Nemours and Co. | Common | 15 | 2,347.31 |
| Louisville Gas and Electric Co. | 5% Preferred | 200 | 5,450.00 |
| Massachusetts Investors Trust | | 600 | 17,513.00 |
| Ophthalmic Publishing Co. | Capital | 5 | None |
| Total Stocks | | | $ 27,929.31 |
| Total | | (A) | $171,445.64 |

WHERRY MEMORIAL FUND

| | | |
|---|---|---|
| U. S. Savings Bonds — Series G | (B) | 4,600.00 |
| | | $176,045.64 |

(A) Held for safekeeping by Trust Dept., First National Bank, Minneapolis, Minn.
(B) Held by Dr. W. L. Benedict, Executive Secretary-Treasurer, Rochester, Minn.

SECURITIES PURCHASED AND SOLD

*October*, 1945 — *October*, 1946

| *Date* | *Description* | *Par Value* | *Purchase Price* | *Selling Price* | *Gain* | *Loss* |
|---|---|---|---|---|---|---|
| BONDS PURCHASED: | | | | | | |
| 12/3/45 | $10,000 U.S. Sav. Bd. Ser. G 2½% | $10,000.00 | $10,000.00 | | | |
| 7/6/46 | $30,000 U. S. Sav. Bd. Ser. G 2½% | $30,000.00 | $30,000.00 | | | |
| BONDS SOLD: | | | | | | |
| 12/3/45 | $5,000 U.S. Sav. Bd. Ser. G 1½% | $ 5,000.00 | $ 5,065.63 | $5,075.00 | $ 9.37 | |
| 12/3/45 | $ 5,000 U.S. Sav. Bd. Ser. G 2¼% | $ 5,000.00 | $ 5,076.56 | $5,029.69 | | $46.87 |
| 3/1/46 | Union Pacific RR Co. Deb. 3½% | $ 6,000.00 | $ 5,947.50 | $6,180.00 | $232.50 | |
| STOCKS SOLD: | | | | | | |
| 2/18/46 | 60 Shs Philip Morris & Co. Com. Stock | | $ 2,530.48 | $3,401.99 | $871.51 | |

Motion made by Dr. J. M. Robb and seconded by Dr. C. H. McCaskey that the reports of the Executive Secretary-Treasurer be accepted as read. Carried.

III

As Editor-in-Chief of the TRANSACTIONS, Dr. W. L. Benedict submitted the following report:

The average bimonthly publication of the TRANSACTIONS was 4,600 copies. Five hundred were allotted to bound volumes: Bound volumes sold in 1944-1945, 503; in 1945-1946, 445 to date. An average of 4,000 copies were mailed bimonthly. From September 1, 1945 to August 31, 1946, the average number of pages of paid advertising was 18½, representing about 28 advertisers.

The *income* from the TRANSACTIONS was as follows:

| | |
|---|---|
| Amount set aside from Fellows' dues | $ 9,536.00 |
| Amount set aside from Candidates' fees | 693.00 |
| Subscribers (nonmembers) | 298.85 |
| Single issues sold | 42.00 |
| Reprints | 155.82 |
| Orders for Bound Volumes | 2,035.00 |
| Advertisers | 6,821.24 |
| Total | $19,581.91 |

*Expenditures* were as follows:

| | |
|---|---|
| Binding Bound Volumes | $ 594.95 |
| Printing 4,600 copies bimonthly | 6,651.42 |
| Cuts and Engravings | 126.99 |
| Supplies | 146.73 |
| Reprints | 509.48 |
| Postage | 324.50 |
| Editor's Honorarium | 2,000.00 |
| Manuscript Editor's Salary | 2,000.00 |
| Total | $12,354.07 |

Excess Income over Expenditure: $7,227.84

It was moved by Dr. Lawrence T. Post and seconded by Dr. Derrick Vail that the report be accepted. Carried.

IV

A communication from Dr. Olaf Larsell for a request for financial assistance in research was referred to the Executive, Research and Finance Committee.

V

*Report of Activities Committee*—Dr. Derrick Vail, Chairman. (*Full reports of the standing and joint committees are printed following the report of the Business Meeting.*)

Reports were received from the following committees and reviewed by the Activities Committee:

1. Advisory Committee to the Registry of Ophthalmic Pathology.
2. Advisory Committee to the Registry of Otolaryngic Pathology.
3. Conservation of Hearing
4. Revision of Pathologic Alases. Dr. Brittain F. Payne recommended that the three atlas committees be dismissed and a new committee created, to be called the "Advisory Committee to the Registries of Pathology." It was moved by Dr. Alan C. Woods and seconded by Dr. O. E. Van Alyea that his recommendation be accepted. Carried.
5. Standardization of Tonometers
6. Advisory Committee to the Eye Health Committee of the American Student Health Association.
7. American Board of Ophthalmology
8. American Board of Otolaryngology
9. American Committee on Optics & Visual Physiology.
10. American Orthoptic Council
11. Industrial Ophthalmology. In accordance with the recommendation of the Committee it was moved by Dr. W. L. Benedict and seconded by Dr. Edmund B. Spaeth that the name of the Committee be changed from the Joint Committee on Industrial Ophthalmology to the Joint Committee on Occupational Ophthalmology. Carried.

No reports were received from the following committees:

1. Pan-American Congress of Otolaryngology.
2. Scientific Exhibit
3. American Association for the Advancement of Science.

VI

*Report of Board of Councillors*—Dr. Derrick Vail, Chairman.

1. Nomination of officers for 1947:
   President-Elect: Carl H. McCaskey, Indianapolis, Ind.
   1st Vice President: A. C. Jones, Boise, Idaho

2nd Vice President: Phillips Thygeson, San Jose, Calif.
3rd Vice-President: Norton Canfield, New Haven, Conn.
Councillor: W. Ivan Lillie, Philadelphia, Pa.
Executive Secretary-Treasurer: William L. Benedict, Rochester, Minn.

Dr. W. L. Benedict stated that he wished to renominate the secretaries with the exception of Dr. Harry S. Gradle who would be replaced by Dr. Lawrence R. Boies of Minneapolis as Secretary for Home Study.

2. Applications of 160 candidates passed by the Council in 1945 were reviewed and accepted.

The applications for Fellowship received in 1946 were reviewed. It was recommended that 224 candidates be accepted. *(The names of candidates* (1945-1946) *for Fellowship approved by the Council are published in the account of the Business Meeting.)*

It was moved by Dr. Ralph I. Lloyd and seconded by Dr. Edmund B. Spaeth that the reports of the Activities Committee and the Board of Councillors be accepted as read. Carried.

## VII

*Report of Executive, Research and Finance Committee*—Dr. Gordon B. New, Chairman.

The Committee made the following recommendations:

1. That the entrance fee and annual dues remain unchanged.

4. That Scott N. Reger, Ph.D., Iowa City, Ia.; Dr. Justo M. Alonzo of Montevideo, Uruguay; Dr. Ricardo Tapia y Fernandez of Mexico City; and Dr. George Morrison Coates, Philadelphia, Pa., be made Honorary Fellows of the Acadmy.

5. *Nominations to Standing and Joint Committees:*

To the Advisory Committee to the Registries of Pathology, Dr. Brittain F. Payne, chairman; Col. J. E. Ash, M.C., ex officio; Dr. Andrew Eggston; Dr. Edmund P. Fowler; Dr. Jonas S. Friedenwald; Stacy Guild, Ph.D.; Dr. Lyman Heine; Dr. Michael J. Hogan; Dr. John E. L. Keyes; Dr. John McGavic; Dr. Bernard J. McMahon; Dr. Frank J. Novak, Jr.; Dr. Algernon B. Reese; Dr. Benjamin Rones; Dr. Theodore E. Sanders; Dr. LeRoy A. Schall; Dr. Herman Z. Semenov; Dr. Georgiana Dvorak-Theobald.

To the Committee on Conservation of Hearing, present committee continued, Dr. Dean M. Lierle, chairman.

To the Committee on Scientific Exhibit, Dr. James H. Maxwell, chairman; and Dr. Kenneth L. Roper.

To the Committee on Standardization of Tonometers, the present committee continued, Dr. Jonas S. Friedenwald, chairman.

To the Advisory Committee to the Eye Health Committee of the American Student Health Association, Dr. Lawrence T. Post, continued.

To the American Association for the Advancement of Science, Dr. Elliot M. Randolph.

To the American Board of Ophthalmology, Dr. Algernon B. Reese, continued.

To the American Board of Otolaryngology, Dr. C. H. McCaskey.

To the American Committee on Optics & Visual Physiology, Dr. Frederick C. Cordes.

To the American Orthoptic Council, Dr. Frank Costenbader.

To the Joint Committee on Occupational Ophthalmology, Dr. Edmund B. Spaeth, Dr. John B. Hitz, and Dr. Richard G. Scobee.

6. That the present Editor-in-Chief of the TRANSACTIONS, Dr. W. L. Benedict, be reappointed.

7. That the next annual meeting of the Academy be held at the Palmer House, Chicago, October 12-17, 1947.

8. That the request from Dr. Olaf Larsell for financial assisance for studies on the development of the human cranial nerves be denied on the grounds that it would set a precedent contrary to previous Council action.

*Recommendations for expenditures:*

To the Registry of Pathology, a sum not to exceed $2,500.

To the Committee on the Conservation of Hearing, a sum not to exceed $3,000.

To the Committee on the Standardization of Tonometers, a sum not to exceed $2,000.

To the American Committee on Optics & Visual Physiology, $100.

To the American Orthoptic Council, a sum not to exceed $100.

To the Joint Committee on Occupational Ophthalmology, a sum not to exceed $250.

9. That the office salaries be left to the Executive Secretary, who can confer with the Subcommittee on Research and Finance regarding necessary or advisable changes.

10. That an honorarium of $2,000 be made to the Editor of the TRANSACTIONS.

It was moved by Dr. Lawrence T. Post and seconded by Dr. C. H. McCaskey that the report be accepted. Carried.

VIII

*New Business*:

Dr. Lawrence T. Post presented a proposal that the Academy set up committees to formulate what they consider ideal standards of undergraduate instruction. This proposal was accepted without motion and the following committees were set up: Committee on Undergraduate Instruction in Ophthalmology, Dr. Lawrence T. Post, chairman; Dr. Alan C. Woods; Dr. Edwin P. Dunphy; and Dr. Derrick Val. To the Committee on Undergraduate Instruction in Otolaryngology, Dr. James H. Maxwell, chairman; Dr. Lawrence R. Boies; Dr. Fletcher D. Woodward; and Dr. Dean M. Lierle.

Dr. Dean M. Lierle, chairman of the Committee on Conservation of Hearing, introduced Dr. E. S. Dalton of Atlantic City. Dr. Dalton stated that within the past two or three years the Kiwanis International had become interested in the care of handicapped children with hearing difficulties. He stated that it was the purpose of Kiwanis International to make the care of such children one of the outstanding projects of that group. The Kiwanis International will cooperate with the Academy Committee on Conservation of Hearing and will lend financial support. Dr. Lierle asked the permission of the Council to cooperate with Kiwanis International and to accept financial support in the furtherance of projects promoted by the Committee. It was moved by Dr. Edmund B. Spaeth and seconded by Dr. C. H. McCaskey that the Committee on Conservation of Hearing be authorized to cooperate with Kiwanis International. No arrangement for the handling of funds from the Kiwanis International was suggested.

Dr. Alan C. Woods proposed that the Academy make a survey of available postgraduate courses in our specialties along the following lines:

a. List of special and general hospitals which offer services in ophthalmology and otolaryngology and give the basic courses required by specialty boards.
b. List of special and general hospitals offering services in ophthalmology and otolaryngology who do not give the required courses.
c. List of institutions giving basic courses in ophthalmology and otolaryngology which are open to candidates not on their individual staffs.

Dr. Derrick Vail moved that Dr. Benedict be empowered to carry out the ideas set forth to the best of his ability. Seconded by Dr. Brittain F. Payne.. Carried.

Dr. W. L. Benedict read the following report of the *Golf and Diversion Club*, which was accepted and tabled:

The Council in 1941 approved the recommendation made by the Activities Committee that a golf committee be formed, the rules of which be left to Dr. C. H. McCaskey and the Executive Secretary. The plan was approved. In 1942 Dr. McCaskey suggested the name "Diversion Club" rather than "Golf Club" and that the activities be extended to include entertainment features connected with the Academy activities. It was also suggested that the activities of the Diversion Club be subsidized to a reasonable extent from Academy funds. The Council also directed by consent without motion that the funds of the Diversion Club be kept separate from those of the Academy so that there will be no appearance on the Academy books of receipts or expenditures of the Diversion Club. The proposal was unanimously approved by Council consent without motion.

Dr. W. P. Wherry was made Secretary-Treasurer of the Diversion Club and Dr. McCaskey, President. Membership in the Diversion Club are sold at $10.00 each. Thirty-one memberships have been sold, none since 1943. The total receipts for 31 members at $10.00 each was $310.00. The disbursements for printing and supplies were $14.60, leaving a balance of $295.40.

In 1942, following the death of Dr. Wherry, Dr. W. L. Benedict was made Secretary-Treasurer and the Diversion Club funds transferred to his custody. The Diversion Club has not been active during the past four years.

The following report on the *Honor Society* was read:

The Honor Society had its inception in an action of the Council in 1938. The Executive Committee offered the following proposal: "Recognition of Past Presidents: For some time it has been realized that with the increasing problems confronting the Council, and also because of widespread importance of some, that the Past Presidents of the Academy should be called upon to serve

as an advisory committee. With this in mind, the Council has asked the Program Committee to arrange some time and place during each convention when the Past Presidents, gathered together, can review such matters as will be brought before them."

The Academy Honor Society was proposed in a brief announcement by Dr. Wherry, published in the *Bulletin* of the Academy, Vol. VIII, No. 3, p. 3, August, 1939. The paragraph reads as follows: "Culminating many suggestions of the past few years the Council has authorized the organization of an Academy Honor Society. A suitable key will be the insignia stamping the recipient as a member. The formulating of rules, regulations and policies of this group has been left to the Past Presidents who will be called together during the Chicago convention for this purpose. The Past Presidents who have retired from the Council activities will comprise the 'Founder group.'"

In the *Bulletin*, Vol. VIII, No. 4, p. 8, December, 1939, appears a REPORT OF PRELIMINARY MEETING IN CONNECTION WITH FORMATION OF ACADEMY HONOR SOCIETY, by S. Hanford McKee, Secretary. The first paragraph of this report is as follows: "Pursuant to a communication received from the Executive Secretary of the American Academy of Ophthalmology and Otolaryngology a number of Past Presidents of the society met today, October 8, for the purpose of organization." Dr. S. Hanford McKee was made secretary of the society and Dr. Joseph C. Beck was made president. In addition to the Past Presidents who had retired from Council activities, the following were elected members of the society: Secord H. Large, W. P. Wherry, John L. Myers, W. L. Benedict and Ralph A. Fenton.

The chair appointed a committee consisting of Drs. Lancaster and Greenwood "to consider the matter of by-laws and constitution for the society and asked that they report at the next meeting." In the *Bulletin*, Vol. IX, No. 1, p. 4, February, 1940, there was published under the caption ACADEMY HONOR SECTION, a proposed constitution and by-laws of the Honor Section of the American Academy of Ophthalmology and Otolaryngology. This article is unsigned.

In subsequent years revisions of the constitution and by-laws were presented, none of which ever was officially adopted. Several members of the Academy were elected to the Honor Society until at the present time there have been enrolled approximately 50.

On several occasions the Honor group has met in official session and attempted to formulate an organization that would be of assistance to the Academy in the development of its educational program. The results of these efforts have not satisfied the members of the Honor group and it is quite generally agreed that there is not sufficient function for such a group to warrant its existence on the present basis.

After considerable correspondence with members of the Honor group, it has been concluded that the Honor group as it is at present constituted has not functioned and that there is no purpose in preserving it as a unit. The purposes in the minds of the founders have proved to be of no practical value.

Dr. W. L. Benedict proposed that the Academy Honor group (the Academy Honor Society) be redefined as follows: The Academy Honor Society shall consist of Fellows of the Academy who, because of their contribution to the activities and purposes of the Academy, have merited special recognition. Elections to the Honor Society shall be made by the Council and the number elected in any one year will not be limited. Upon election to the Honor Society, the Academy will award the Honor Society Key.

It was moved by Dr. Alan C. Woods that the report and the proposal therein be accepted and adopted. The motion was seconded by Dr. Dean M. Lierle. Carried.

The motion was made by Dr. W. L. Benedict and seconded by Dr. A. D. Ruedemann that Life and Senior ribbons on badges be discontinued in the future. Carried.

# ANNUAL BUSINESS MEETING

WEDNESDAY EVENING, OCTOBER 16, 1946

The annual business meeting of the American Academy of Ophthalmology and Otolaryngology, held during the Fifty-First Annual Session, at the Palmer House, Chicago, on Wednesday evening, October 16, 1946, convened at eight-fifteen o'clock in the Grand Ballroom. Dr. Gordon B. New, President, presided.

PRESIDENT NEW: Gentlemen, we will start the business meeting of the Academy. The first order of business is the report of the Executive Secretary-Treasurer, Dr. Benedict.

EXECUTIVE SECRETARY-TREASURER BENEDICT: Mr. President and Members: Active and Honorary Fellows, October 1, 1946, 3,587; candidates for election at this meeting, 384, which will make a total membership of 3,971. Twenty-seven Fellows will be elevated to Senior Fellowship on January 1, 1947, and 33 to Life Fellowship.

The Secretary publishes early in each year a Directory, which contains the names, addresses and classification of all Fellows.

The Secretary has kept records of all official meetings of the Program Committee and of the Council that have occurred during the year, and these records are on file in the home office. The Secretary has received reports from Council Committees, Standing and Joint Committees, which have been submitted to the Council for consideration and will be referred to this body for final action by the Senior Member of the Council.

The full report of the Treasurer will appear in the November issue of the TRANSACTIONS. (*See minutes of Council Meeting*).

The fiscal year of the Academy is from January 1 to December 31. Total revenues from January 1, 1946 to August 31, 1946, were $60,442.75 and total expenditures for the same period were $22,669.47. Excess income over expenses, therefore, amounting to $37,773.28.

PRESIDENT NEW: The report of the Necrologist.

EXECUTIVE SECRETARY-TREASURER BENEDICT: From October 1, 1945, to October 1, 1946, the Academy lost 50 members through death. From issue to issue the names are published in the TRANSACTIONS and obituaries are published in the July-August issue.

PRESIDENT NEW: Will the members please stand for a moment in reverence of those who have passed on?

PRESIDENT NEW: The next report is the report of the Editor of the TRANSACTIONS.

EXECUTIVE SECRETARY-TREASURER BENEDICT: The average bimonthly publication of the TRANSACTIONS was 4,600 copies. Five hundred were allotted to bound volumes: Bound volumes sold in 1944-45, 503; for 1945-46, 445 to date. An average of 4,000 copies were mailed bimonthly. From September 1, 1945 to August 31, 1946, the average number of pages of paid advertising was 18½, representng about 28 advertisers.

The income from the TRANSACTIONS, including subscriptions and advertising, to date is $19,581.91, and the expenditures are $12,354.07, leaving a total profit on the TRANSACTIONS to date of $7,227.84.

PRESIDENT NEW: The next order of business are the reports of the American Boards of Ophthalmology and Otolaryngology. (*These are printed following the report of the Business Meeting.*)

PRESIDENT NEW: The next report is that of the Senior Member of the Council. Dr. Lloyd is not here. The Secretary will read the report.

EXECUTIVE SECRETARY-TREASURER BENEDICT: "Report of Senior Member of Council. The Council has received and approved the report of the Executive Secretary-Treasurer, Necrologist, and the Editor of the TRANSACTIONS. A summary of these reports has just been read to you by the Secretary.

"The report of the Committee on Research and Finance (a subcommittee of the Executive Research and Finance Committee) has been accepted. A detailed report of the functions of this committee will appear in the annual audit at the close of the fiscal year.

"The Activities Committee has reviewed the reports of the Standing and Joint Committees. (*These are printed following the report of the Business meeting.*) The following nominations were made by the Executive Research & Finance Committee.

*"Nominations to Standing and Joint Committees:*

Conservation of Hearing—Dean M. Lierle, Chairman. Other members of the committee to be appointed by Dr. Lierle.

Standardization of Tonometers—Jonas S. Friedenwald, Chairman. Other members of the committee to be appointed by Dr. Friedenwald.

Advisory Committee to the Eye Health Committee of the American Student Health Association—Lawrence T. Post.

American Association for the Advancement of Science—Elliott Randolph.

American Board of Ophthalmology—Algernon B. Reese.

American Board of Otolaryngology— C. H. McCaskey.

American Committee on Optics and Visual Physiology—Frederick C. Cordes.

American Orthoptic Council—Frank Costenbader.

The following appropriations were made for the year, 1947:

Committee on Conservation of Hearing—a sum not to exceed $3,000.

Committee on Standardization of Tonometers—a sum not to exceed $2,000.

American Committee on Optics and Visual Physiology—a sum not to exceed $100.

American Orthoptic Council—a sum not to exceed $100.

Committee on Occupational Ophthalmology —$250.

Registries of Pathology—a sum not to exceed $2,500.

"The Joint Committee on Industrial Ophthalmology has recommended that the name of the committee be changed to Joint Committee on Occupational Ophthalmology. This recommendation was approved, the same action having been taken by the Section on Ophthalmology of the A.M.A.

"The Committee on Revision of Pathologic Atlases has approved the reprinting of 1,000 copies of the Atlas of Ophthalmic Pathology and 700 copies of the Atlas of Otolaryngic Pathology. Plans are being made to revise the present atlases but work required will delay the publication of new atlases for more than one year. The new atlases will be prepared by Colonel J. E. Ash, Director of the Registries of Pathology, and Mrs. Helenor Wilder, with the cooperation of the Editorial Board of the Committee.

"The Committee on the Conservation of Hearing has recommended and the Council has approved the re-publication of "Syllabus of Audiometric Procedures in Administration of a Program for the Conservation of Hearing of School Children." The committee has prepared and adopted an audiogram form which it hopes will be universally adopted. The committee has recommended the establishment of additional training programs for audiologists in the various universities. It has recommended that the word 'audiology' be recognized by the Academy and incorporated in dictionaries. The committee recommended that an attempt be made by the Academy to set up a bureau to certificate and regulate audiologists.

"The Executive, Research and Finance Committee recommended that the entrance fee and annual dues remain unchanged. William L. Benedict was appointed Editor-in-Chief of the TRANSACTIONS. Changes in membership were ordered by the committee as follows:

Dropped for non-payment of dues—3 Fellows.

Withheld for further consideration—3 Fellows four years delinquent in dues.

Resignations—5.

The dues of 11 Fellows were remitted for cause.

*"Nominated for Honorary Fellowship:*

Scott N. Reger, Ph.D., Iowa City, Iowa.

George Morrison Coates, Philadelphia, Pennsylvania.

Justo M. Alonso, Montevideo, Uruguay.

Ricardo Tapia y Fernandez, Mexico City.

"The Council have recommended that the Committee on Ophthalmic Pathology, the Committee on Otolaryngic Pathology, and the Committee on Revision of Pathologic Atlases be discontinued. They recommended the appointment of a Standing Committee to be known as the Advisory Committee to the Registries of Pathology. This was occasioned by changes that have taken place in Washington, including the establishment of new registries separate from the Army Medical Museum. Dr. Brittain F. Payne is nominated for Chairman. Other members of the committee to be appointed by Dr. Payne with the approval of the Council.

"The Council recommends the appointment of a Committee on Undergraduate Teaching of Ophthalmology, Chairman, Lawrence T. Post.

"The Council recommends the appointment of a Committee on Undergraduate Teaching of Otolaryngology, Chairman, James Maxwell.

"The Council recommended that the Executive Secretary prepare data for consideration relative to graduate school courses and residencies in ophthalmology and otolaryngology.

"The Council recommends the redefinition of the Academy Honor Society. (*The report of Dr. W. L. Benedict (see Council Minutes) was read at this time.*)

"The Council nominates for Fellowship in the Academy 384 candidates. The names of these candidates are listed on the sheets which were handed to you at the door. One hundred and sixty of these candidates were approved by the Council in 1945, and their election to Fellowship will be retroactive."

PRESIDENT NEW: Gentlemen, the credentials of these candidates have been carefully gone into by the Council, and it is their recommendation that the candidates listed here for 1945-46 be elected. Will someone please make a motion to that effect?

DR. DERRICK VAIL (Chicago, Ill.): I so move.

. . . The motion was seconded . . .

PRESIDENT NEW: All those in favor say "aye"; opposed. It is so ordered.

*Newly elected Fellows of the Academy:*

**1945**

**Abrams, Murray, Major (MC), 100 Woodruff Ave., Brooklyn, N. Y., ALR.**
**Altmann, Franz, 10 East 85th St., New York, N. Y., ALR.**
**Ashley, Richard Warner, 5811 8th Ave., Kenosha, Wis., OP.**
**Atkins, Joseph Preston, 250 S. 18th St., Philadelphia, Pa., ALR.**
**Auslander, Milton Manuel, 6738 N. Broad St., Philadelphia 26, Pa., ALR.**

**Bancks, Howard Raymond, 75 Lenox Rd., Brooklyn, N. Y., ALR.**
**Bau, Hyman William, 3216 W. Roosevelt Road, Chicago 24, Ill., ALR.**
**Beetham, William P., 108 Bay State Road, Boston, Mass., OP.**
**Bender, Rosarie Rossell, 195 Linwood Avenue, Buffalo, N. Y., ALR.**
**Bernasconi, Ezio Joseph, 716 River Ave., Providence, R. I., OP.**
**Black, Vernon Anderson, 105 Hawes St., Wharton, Texas, OP.**
**Bogdasarian, Robert Michael, 108 Oak St., Binghamton, N. Y., ALR.**
**Brennan, James William, 11 Horton Place, Buffalo, N. Y., OP.**
**Brikates, Peter, 140 East 54th St., New York, N. Y., OP.**
**Burman, Daniel, 121 First St., Newburgh, N. Y., ALR.**
**Busby, William, Jr., EENT Clinic, Madigan General Hosp., Ft. Lewis, Wash., ALR.**

**Carey, Edward Thomas, 415 Howes Bldg., Clinton, Iowa, ALR.**
**Cerney, Frank Joseph, Capt. (MC), Vetans Administration, Dayton, Ohio, OP**
**Chamberlin, Ira Iowa, 4 Chestnut St., Springfield, Mass., ALR.**
**Chester, Lewis, 179 Allyn St., Hartford 3, Conn., ALR.**
**Chevigny, Julius James, 504 Broadway, Gary, Ind., ALR.**
**Cockrum, William Marion, 908 Hulman Bldg., Evansville, Ind., OP.**
**Cole, John Gordon, 780 Park Ave., New York, N. Y., OP.**
**Collins, Arthur D., 1007 Guardian Bldg., Cleveland 14, Ohio, OP.**
**Craig, Stuart Lessley, 135 East 65th St., New York 21, N. Y., ALR.**
**Crandall, Alan Slade, 313 S. 17th St., Philadelphia 3, Pa., OP.**
**Croll, Maurice, 1659 Longfellow Ave., Detroit, Mich., OP.**
**Crowe, Samuel James, Johns Hopkins Hospital, Baltimore, Md., ALR.**
**Cuthbert, Marvin Peare, Lt. (MC), Station Hosp., Camp Carson, Colo., OP.**
**Cutler, Max Helix, 636 Church St., Evanston, Ill., ALR.**

**Davis, Ralph Allison, 4753 Broadway, Chicago, Ill., OP.**
**Duclos, Gaston Normand, Capt. (RCAM C), No. 3 Du Moulin St., Drummondville, Que., Canada, ALR.**

**Ehrlich, Leon Herbert, 211 Central Park West, New York 24, N. Y., OP.**
**Eilert, John Harold, 391 E. 149th St., New York, N. Y., OP.**
**Elwyn, Herman, 239 Central Park West, New York, N. Y., OP.**
**Estlick, Richard E., 4223 Beaver Ave., Ft. Wayne, Ind., ALR.**

**Farmer, William Dempsey, Major (MC), 119 North Elm St., Greensboro, N. C., ALR.**
**Flack, Clarence Elbert, 715 Lake St., Oak Park, Ill., OP.**
**Flickinger, Roger R., Park Hosp. Clinic, Mason City, Iowa, ALR.**
**Flora, William Kenneth, 406 Main St., Johnstown, Pa., ALR.**
**Fox, Nathan H., 4010 W. Madison, Chicago, Ill., OP.**

Frazer, John Paul, 224 Aberdeen St., Rochester, N. Y., ALR.
Fried, Joseph John, 5 West 86th St., New York 24, N. Y., OP.

Gershon, Nathan Isaac, Lt. Comdr. (MC), 26 Linde- Ave. N. E., Atlanta, Ga., ALR.
Goldhammer, Samuel, Republic Bldg., Denver 2, Colo., OP.
Gordon, Dan Morris, 525 E. 68th St., New York 21, N. Y., OP.
Graham, George Albert, 842 Carrol St., Brooklyn 15, N. Y., OP.
Gromet, Maurice T., 105 Lincoln Rd., Brooklyn 25, N. Y., ALR.
Guerry, Du Pont, 3rd, 503 Professional Bldg., Richmond, Va., OP.

Gunderson, Harvey Clifford, 2001 Collingwood Ave., Toledo, Ohio, ALR.
Gurwin, Bernard J., 900 Seventeenth N. W., Washington, D. C., OP.
Gutkin, Morton Lewis, Capt. (MC), 6734 Sixth Ave., Brooklyn, N. Y., ALR.

Hanson, A. George, 1528 Medical-Dental Bldg., Seattle 1, Wash., OP.
Harper, DeWitt Lawrence, 204 North Main, Greenville, S. C., ALR.
Helbling, Franklin Kirkland, 34 No. Ash St., Ventura, Calif., ALR.
Henderson, Charles Henry, 800 Park Ave., Norton, Va., OP.
Hipp, Walter, 140 East 54th St., New York 22, N. Y., OP.
Hooker, Lyle, 446 Esperson Bldg., Houston 2, Texas, OP.
Horsfield, Russell Thomas, 715 Medical-Dental Bldg., Seattle 1, Wash., OP.
Howley, Bartley Maxwell, 67 Livingston Ave., New Brunswick, N. J., ALR.

Jaeckle, Charles Eisele, 136 Evergreen Pl., East Orange, N. J., OP.
Jervey, James W., Jr., 101 Church St., Greenville, S. C., OALR.
Johnson, Carl Cordes, 5 Bay State Road, Boston 15, Mass., OP.
Johnson, Hobart Clemens, 839 Belgrade Ave., Mankato, Minn., OP.
Johnson, Roger Harry, 522 Cobb Bldg., Seattle, Wash., OP.

Katzin, Herbert Maurice, 1148 Fifth Ave., New York 28, N. Y., OP.
Kelly, Francis B., 519 Med. Prof. Bldg., Corpus Christi, Texas, OP.
Kennon, Beverley Randolph, 3rd., 908 Medical Arts Bldg., Norfolk, Va., OP.
Kilmann, Martin H., 118 E. 54th St., New York 22, N. Y., OP.
Kuhn, Clifford W., Major (MC), 9545 S. W. 62nd Dr., Portland, Ore., ALR.
Kuntz, Herman William, 501 Hume-Mansur Bldg., Indianapolis, Ind., ALR.

Lee, Otis S., Jr., 112 Rimbach St., Hammond, Ind., OP.
Lenzen, Aloysius Francis, 206 Marquette St., LaSalle, Ill., OP.
Levin, Bert G., 1210 Lowry Bldg., St. Paul 5, Minn., ALR.
Lewis, Charles Kelley, 2109 Sterick Bldg., Memphis, Tenn., ALR.
Lieberman, George Emanuel, 1721 Pine St., Philadelphia, Pa., ALR.
Lodge, Edmund Anderson, 79½ Prospect St., Gloucester, Mass., ALR.

McEwen, James Woods, 670 Cherry St., Terre Haute, Ind., OP.
McKenzie, George Edward, 602 DuPont Bldg., Miami, Fla., ALR.

Maier, Haskel Levitt, 936 Medical Arts Bldg., Tacoma 2, Wash., ALR.
Mather, Homer Raymond, Jr., Capt. (MC), 172nd Station Hosp., APO 547, c/o PM, San Francisco, Cal., OP.
Meer, Nadhim Edward, Lt. (MC), Wyckoff, N. J., ALR.
Meyer, Monte F., 645 Canal Bldg., New Orleans, La., ALR.
Miears, Claude H., 713 Scarbrough, Austin, Texas, ALR.
Miller, Claude K., Widby-Hadley Bldg., Wenatchee, Wash., OP.
Miller, Harold Rexford, 114 E. 54th St., New York 22, N. Y., OP.
Minsky, Armen A., Capt. (MC), 626 La Salle, Minneapolis, Minn., ALR.
Monsees, Wayne Emerson, 233 A St., San Diego, Calif., OP.
Morse, Leo M., 25 East Washington St., Suite 1523, Chicago, Ill., ALR.
Muirhead, Robert Mowatt, 8 East Broadway, Salt Lake City 1, Utah, ALR.
Munoz - MacCormick, Carlos Eduardo, Monterry Bldg., Park Terrace, Santurce, Puerto Rico, ALR.

Nantz, Frank A., 225 Bodmann Ave., Cincinnati, Ohio, OP.
Naugle, Thomas Calvert, 618 College St., Columbus, Miss., ALR.
Nicholson, Charles T., 422 Tyler Pl., Alexandria, Va., ALR.
Noonan, Paul Ecret, Capt. (MC), 238 N. Park Ave., Warren, Ohio, ALR.

Osherwitz, Morris Stanford, 456 Doctors Bldg., Cincinnati 2, Ohio, OP.
Ostrom, Louis, 124 18th St., Rock Island, Ill., ALR.

Paradis, Charles Ovide, 625 Cleveland Ave. N.W., Canton 3, Ohio, ALR.
Parker, Harry Caunter, 673 Broadway, Gary, Ind., ALR.
Parkinson, Harold O., 626 Flood Bldg., San Francisco, Calif., ALR.
Petix, Dominick Gilbert, 2615 Grand Concourse, Bronx, N. Y., OP.
Pfeiffer, Guy Oliver, 19-A South High St., Belleville, Ill., ALR.
Phillips, Warren Charles, 801 N. Second, Harrisburg, Pa., OP.
Pitkin, York Nones, Lt. Col. (MC), 10515 Carnegie Ave., Cleveland, Ohio, ALR.
Pohlman, David Alexander, Major (MC), Address unknown, ALR.
Popovich, Stephen J., 1930 Wilshire Blvd., Suite 617, Los Angeles, Calif., OP.
Price, F. Raymond, 118 Rutledge Ave., Charleston 16, S. C., OP.

Raggio, Francis Warren, Jr., Gordon Bldg., Lake Charles, La., ALR.
Rathgeber, Van D., 1305 Medical Arts Bldg., Ft. Worth, Texas, OP.
Ray, Joseph Carr, 914 Frances Bldg., Louisville, Ky., ALR.
Rein, Walter John, 501 E. Franklin St., Richmond, Va., OP.
Riesenbeck, Leo Henry, 831 Carew Tower, Cincinnati, Ohio, ALR.
Ripsteen, Joel Stanley, Capt. (MC), 200 Parkview Ave., Allentown, Pa., ALR.
Rohm, Jack Zeth, 7002 Jenkins Arcade, Pittsburgh, Pa., ALR.
Roman, Stanley John, Capt. (MC), Address unknown, OALR.
Rosenberg, William, 25 E. Washington St., Rm. 1523, Chicago, Ill., OP.
Ross, Perry W., 5426 N. Sheridan Rd., Chicago, Ill., OP.
Royce, Thomas L., 904 Holman, Houston, Texas, ALR.
Rucker, Ralph Weller, Major (MC), Union Nat'l Bank Bldg., Bartlesville, Okla., ALR.
Ruehl, William W., 7106 Jenkins Arcade, Pittsburgh 22, Pa., ALR.

Sannella, Lee George, 124 Waterman, Providence, R. I., OP.
Schachat, Walter S., 30 E. 60th St., New York 22, N. Y., OP.
Scovis, I. John, U. S. National Bank Bldg., The Dalles, Ore., OP.
Shepard, Willis, B., Miner Bldg., Eugene, Ore., ALR.
Shofstall, Charles K., Balcony Bldg., Kansas City 2, Mo., ALR.
Shroder, Luther Null, R.R. 5, Greenville, Ohio, ALR.
Siniscal, Arthur Alfred, M. Trachoma Hospital, Rolla, Mo., OP.
Sirles, Wayne Pulley, 4850 Scott Ave., St. Louis 10, Mo., ALR.
Skinner, Merton Byron, 30 E. Washington, Chicago, Ill., ALR.
Slaughter, Howard Clarence, 908 Hulman Bldg., Evansville, Ind., OP.
Sommer, Ignatius George, 981 S. Westmoreland, Los Angeles 6, Calif., OALR.
Southgate, Paul T., 302 Professional Bldg., Long Beach, Calif., OP.
Stagman, Joseph, 55 E. Washington St., Chicago, Ill., ALR.
Staman, Harry H., 49 W. Church St., Uniontown, Pa., ALR.
Stamm, Carl, 1737 Chestnut St., Philadelphia 3, Pa., ALR.
Staton, Younger A., 715 Harvey Bldg., West Palm Beach, Fla., OALR.
Steinberg, Nathan, 253 Sunrise Highway, Rockville Centre, N. Y., ALR.
Stelle, Harry L., 207 Commerce Bldg., Pittsburg, Kans., OALR.
Stolzar, Irwin H., Capt. (MC), 831 Guardian Bldg., Cleveland, Ohio, OP.
Surber, John Roger, 119 E. 74th St., New York 21, N. Y., ALR.
Swenson, Rudolph E., 1 Carver St., Plymouth, Mass., ALR.

Tait, Edwin Forbes, 1324 W. Main St., Norristown, Pa., OP.
Teasdale, Laurie Redmond, 311 Professional Bldg., Charleston, S. C., ALR.
Teixler, Victor Adolph, 224 Hume Mansur Bldg., Indianapolis, Ind., OP.
Thompson, Chase S., Capt. (MC), West Texas Hosp., Lubbock, Texas, ALR.
Thornell, William Clyde, 1002 Carew Tower, Cincinnati, Ohio, ALR.
Tisher, Paul Winslow, 99 W. Main St., New Britain, Conn., OP.
Tobin, Henry Loughlin, 74 Front St., Binghamton, N. Y., OP.
Tulloch, George M., 410-12 McCulloch Bldg., Okmulgee, Okla., ALR.

U'ren, Harold Merle, 1735 N. Wheeler Ave., Portland 12, Oregon, OP.

Viger, Roland Joseph, 1414 Drummond St., Montreal, P. Q., Canada, OP.
Voorhees, Darrell Graham, 135 E. 65th St., New York, N. Y., ALR.

Wall, Walton B., 320 N. Main St., Orlando, Fla., OP.
Webb, Walter Whayne, Third Nat'l Bldg., Dayton, Ohio, ALR.
Wilcoxen, William B., Bowling Green, Mo., ALR.
Wild, Joseph Peter, 161 W. Wisconsin Ave., Milwaukee, Wis., ALR.
Wolff, Jerome Marshall, 822 Park Ave., Plainfield, N. J., OP.
Wood, George Harden, Petroleum Bldg., Big Spring, Texas, ALR.
Wright, R. Wesley, 300 Homer Ave., Palo Alto, Calif., ALR.

Zavalia, Alberto Urrets, 27 de Abril 244, Cordoba, Argentina, OP.

**1946**

Aagesen, Walter J., 622 Citizens Bank Bldg., Anderson, Ind., ALR.
Abrams, Henry, 195 Nassau St., Princeton, N. J., Op.
Agatston, Howard James, 57 West 57th St., New York 19, N. Y., Op.
Allen, Benjamin Israel, 1070 Park Ave., New York, N. Y., ALR.
Alpert, Max, 881 Lafayette St., Bridgeport, Conn., ALR.
Alter, Francis William, Jr., 23 Second Ave., San Mateo, Calif., ALR.
Amdur, Louis A., 2540 Boulevard, 15 Exch. Place, Jersey City 4, N. J., Op.
Anderson, Ernest Richard V., 1052 W. Sixth St., Los Angeles 14, Calif., ALR.
Arnold, Leon M., 1235 Park Ave., New York 28, N. Y., ALR.
Auten, Hanford Louis, Jr., Dartmouth Eye Institute, Hanover, N. H., Op.

Baker, Lauchlin J., 310 Franklin St., Ogdensburg, N. Y., ALR.
Barnaby, Henry C., Capt. (M.C.), Walter Reed General Hospital, Washington, D. C., ALR.
Barnet, Emmanuel, 214 W. Grand Ave., Wisconsin Rapids, Wis., ALR.
Bauer, Charles E., 11 Cannon St., Poughkeepsie, N. Y., Op.
Bellucci, Richard John, 121 East Sixtieth St., New York 22, N. Y., ALR.
Bergida, Jerome J., Comdr. (MC), 1615 Dorchester Rd., Brooklyn, N. Y., ALR.
Berkley, William Leneave, U. S. Naval Hospital, St. Albans, N. Y., Op.
Berman, Harry, 128 Lincoln St., Salt Lake City 2, Utah, ALR.
Bizzell, Marcus Edward, 139 W. Walnut Street, Goldsboro, N. C., ALR.
Bloom, Samuel M., 216 E. 15th St., New York 3, N. Y., ALR.
Bolton, Wilbur Minnis, 1020 S. W. Taylor St., Portland 1, Ore., ALR.
Brav, Solomon S., 2027 Spruce St., Philadelphia 41, Pa., Op.
Brody, Yale, 919 Bank of America Bldg., Stockton, Calif., Op.
Brown, Harold Ethan, 581 Bulen Ave., Columbus 9, Ohio, Op.
Brumm, Lewis Patrick, 726 Carew Tower, Cincinnati, Ohio, Op.
Burns, Coleman Craig, Medical Arts Bldg., Abilene, Texas, ALR.

Calhoun, Ferdinand Phinizy, Jr., 478 Peachtree St., Atlanta, Ga., Op.
Cameron, Walter Courtney, 1103 Medical Arts Bldg., Tacoma 2, Wash., Op.
Cappeta, Domenico, 125 East 84th, New York, N. Y., Op.
Cavanor, Frank Tracy, 1818 Medical Arts Bldg., Minneapolis 2, Minn., ALR.
Chait, Robert A., 20 Plaza St., Brooklyn, N. Y., ALR.
Chamberlin, Clyde G., 300 Rentschler Bldg., Hamilton, Ohio, ALR.
Chessen, James, Capt. (MC), 763 Race St., Denver 8, Colo., ALR.
Chinn, Chester W., 209 W. 125 St., New York, N. Y., OALR.
Christenberry, Henry Edward, Jr., 501 W. Church St., Knoxville, Tenn., ALR.
Cirillo, Anchise Anthony, 89-21 153rd St., Jamaica, N. Y., ALR.
Clark, William H., 120 N. Lafayette Blvd., South Bend, Ind., ALR.
Cobey, James Francis, 1210 Chapel St., New Haven 11, Conn., OALR.
Cody, Claude Carr, III, 1304 Walker Ave., Houston, Texas, ALR.
Covell, Kermit Wendell, 610 Main St., Racine, Wis., ALR.
Cowell, Edward Huntington, 427 Biltmore Way, Coral Gables 34, Fla., Op.
Crane, Donald Van Denburg, Medical Arts Bldg., Tulsa 3, Okla., Op.
Curts, James H., 330 South Washington St., Saginaw, Mich., Op.
Cutler, Morton, 122½ Main, South, Twin Falls, Idaho, Op.

Dan, Julius Martin, 1362 President St., Brooklyn 13, N. Y., Op.
Davies, Charles Edgar, 925 West Georgia St., Vancouver, B. C., Canada, Op.
Della Rocca, Anthony J., 1400 Benson St., New York 61, N. Y., ALR.
Diehl, William Harold, 701 Chestnut St., Lebanon, Pa., ALR.

Donoghue, William Francis, Jr., 69 Chestnut St., Springfield, Mass., ALR.
Drell, Maurice Joseph, 630 Medical Arts Bldg., Seattle 1, Washington, Op.
Duncan, Frank Benbow, 301 Polk St., Amarillo, Tex., Op.

Edwards, David Lloyd, 203 Stanolind Bldg., Tulsa 3, Okla., Op.
Edwards, Thomas Marvin, 322 Citizens Bldg., Tampa 2, Fla., ALR.
Emrich, Paul Stanley, 64 Mt. Vernon, Oshkosh, Wis., ALR.
Ersay, Emil Francis, 1030 Euclid Ave., Cleveland 14, Ohio, ALR.
Esterly, Daniel Beard, 104 North Madison, Pasadena 4, Calif., Op.

Fawcett, Keith R., 205 W. 2nd St., Duluth, Minn., Op.
Fein, Norman Nathaniel, Trinity Hosp., Little Rock, Ark., ALR.
Fields, Jack, 104 South Michigan Ave., Chicago 1, Ill., Op.
Fisher, Harry Richard, 465 West End Ave., New York, N. Y., ALR.
Friedman, Joseph Jay., 57 Midwood St., Brooklyn 25, N. Y., ALR.
Fringer, Robert Chatterton, 216 News Tower, Rockford, Ill., Op.
Fritz, Milo H., 927 Park Ave., New York 28, N. Y., OALR.

Gaines, Shelley Rice, American Bank Bldg., New Orleans 12, La., Op.
Gardiner, Charles Ernest, 701 Chestnut St., Lebanon, Pa., ALR.
Garlichs, Richard Walton, 216 North Manoa Rd., Havertown, Pa., ALR.
Garvey, Thomas Quincy, 115 E. 61st St., New York, N. Y., ALR.
Gettes, Bernard Charles, 2028 Pine St., Philadelphia, Pa., Op.
Gilligan, John Henry, 1831 Wilson Blvd., Arlington, Va., ALR.
Goldman, Jerome, 1 Johnson Ave., Newark 8, N. J., Op.
Gordon, George Raoul, 913 S. 19th St., Birmingham, Ala., ALR.
Gordon, John Simpson, 950 E. 59th St., Chicago 19, Ill., ALR.
Greene, Philip Burkland, 517 Old National Bank Bldg., Spokane, Wash., Op.
Greetham, James S., Capt. (MC), Wakeman General Hospital, Camp Atterbury, Ind., Op.
Gromer, Terry John, 110 Metropolitan Bldg., Denver, Colo., ALR.
Gurau, Henry H., 215 Bankers Trust Bldg., Des Moines, Iowa, Op.

Hanson, Frederick Charles, 162 Angell St., Providence 6, R. I., Op.
Harley, Robison D., 101 South Indiana Ave., Atlantic City, N. J., Op.
Harris, Herman Lee, Reynolds Arcade Bldg., Bristol, Va., ALR.
Hart, Verling Kersey, 106 W. 7th St., Charlotte 4, N. C., ALR.
Harvey, Elmer Frank, 1422 Euclid Ave., Cleveland 15, Ohio, Op.
Hays, Arthur Vernon, 54th Femeway, Boston, Mass., ALR.
Henderson, James Alexander, Comdr. (MC), U. S. Naval Hospital, Philadelphia 45, Pa., ALR.
Henrich, Mary Ingeborg, 40 North St., Buffalo 2, N. Y., Op.
Henrickson, John William, 2001 Fourth Ave., San Diego, Calif., ALR.
Hester, Marion William, 317 Marble Arcade Bldg., Lakeland, Fla., Op.
Hetzel, Clarence Charles, Jr., 721 Eccles Bldg., Ogden, Utah, ALR.
Hickey, Cornelius S 39 Bay State Road, Boston 15, Mass., ALR.
High, Howard C., University of Michigan, Ann Arbor, Mich., ALR.
Hilger, Jerome Andrew, 444 Lowry Medical Arts Bldg., St. Paul, Minn., ALR.
Hogan, Michael John, 450 Sutter St., San Francisco 8, Calif., Op.
Holmes, Dorothy Behner, 1816 R Street N. W., Washington 9, D. C., Op.
Huffman, William Clyde, University Hospitals, Iowa City, Iowa, ALR.

Inciardi, James A., 2415 Beverly Rd., Brooklyn 26, N. Y., Op.

Johnson, Thomas W., 529, 39 East Ohio, Indianapolis 4, Ind., ALR.
Jones, John Allen, 401 S. Court St., Montgomery, Ala., ALR.
Jordan, Harvey Ernest, Jr., 1912 Spruce St., Philadelphia 3, Pa., ALR.
Jordan, Raymond Ellsworth, 121 University Place, Pittsburgh 13, Pa., ALR.

Kallen, Leo A., 667 Madison Ave., New York 21, N. Y., ALR.
Kaufman, Aaron, 483 Beacon St., Boston 7, Mass., ALR.
Keim, William Franklin, Jr., 15 Washington St., Nowark 2, N. J., ALR.
Key, Samuel Newton, Jr., University Hospitals, Iowa City, Iowa, Op.
King, James Thomas, 803 Medical Arts Bldg., Atlanta 3, Ga., ALR.
Klaff, Daniel David, 3720 Washington Blvd., St. Louis 8, Mo., ALR.

Koff, Raphael Joseph, 1919 Wilshire Blvd., Los Angeles, Calif., Op.
Kornblut, Alfred, 1539 Fairfield Ave., Bridgeport 5, Conn., ALR.
Kulesher, Henry T., 3825 Pershing Drive, Arlington, Va., ALR.

Lamb, Andrew J., 10006 Carnegie Ave., Cleveland, Ohio, ALR.
Larose, Joseph Henry, 1317 Whitney Bldg., New Orleans 12, La., Op.
Lavine, Oscar, Suite 620, 1801 Eye St., N. W., Washington 6, D. C., Op.
Lees, Charles Ray, 806 Medical Arts Bldg., Fort Worth, Texas, Op.
Lennarson, Vincent A., 215 North Sheridan Road, Waukegan, Illinois, ALR.
Levitt, Edward J., 840-842 Maccabees Bldg., Detroit 2, Mich., ALR.
Lieberman, Harold, 175 June St., Worcester, Mass., ALR.
Linhart, William Oran, 521 Medical Arts Bldg., Pittsburgh, Pa., Op.
Lisman, Jack Victor, 654 Madison Ave., New York, N. Y., Op.
Livshin, Norman, 611 University Bldg., Syracuse, N. Y., ALR.
Locke, William Walter, 507 Empire Bldg., Birmingham, Ala., ALR.
Long, Albert Emanuel, 169 Main St., Weston, W. Va., Op.

McCrory, James Lynn, 607 North Grand, St. Louis 3, Mo., ALR.
McFarland, Corley B., 122 N. Lafayette Blvd., South Bend, Ind., Op.
McGraw, James Lorenz, 619 University Bldg., Syracuse, N. Y., Op.
McNally, John Joseph, 121 E. 60th St., New York, N. Y., ALR.
Marden, Philip Ayer, 1912 Spruce St., Philadelphia 3, Pa., ALR.
Marmor, William A., 607 N. Grand Blvd., St. Louis, Mo., ALR.
Merrill, Rowland Hyde, 1010 First National Bank, Salt Lake City, Utah, Op.
Mings, Dwain Edward, 1684 N. Prospect Ave., Madison, Wis., Op.
Missal, Sylvester Clemens, 2323 Prospect Ave., Cleveland, Ohio, ALR.
Mitchell, Douglas Gatlin, 703 Medical Arts Bldg., Fort Worth, Texas, Op.
Morris, Francis O'Neil, 820 S. Cainter Ave., Whittier, California, ALR.
Neivert, Harry, 555 Park Ave., New York 21, N. Y., ALR.
Nelson, John Russell, Wyoming National Bank Bldg., Casper, Wyo., ALR.
Nicholson, John Bixby, 401 Savings Bank Bldg., Marquette, Mich., ALR.
Norris, Charles Morgan, 3401 N. Broad St., Philadelphia 40, Pa., ALR.
Noyes, Leighton Helmuth, 67 Main St., Brattleboro, Vt., ALR.

O'Brien, James Miles, Dartmouth Eye Institute, Hanover, N. H., Op.
O'Donnell, Merrill Charles, 725 Jenkins Bldg., Pittsburgh 22, Pa., ALR.
Offenbach, Bertha, 270 Commonwealth Ave., Boston, Mass., Op.
O'Keefe, John Joseph, 255 S. 17th St., Philadelphia 3, Pa., ALR.
Olsen, Arthur Roy, 1421 State St., Santa Barbara, Calif., ALR.
Owens, William Councilman, Johns Hopkins Hospital, Baltimore 5, Md., Op.

Page, Donald Franklin, Boulder-Colorado Sanitarium, Boulder, Colo., ALR.
Peck, Howard Burgess, 610 South Carlisle, Alberquerque, N. Mex., Op.
Peele, James Clarendon, Box 636, Goldboro, N. C., ALR.
Penta, Arthur Quito, 1301 Union St., Schenectady, N. Y., ALR.
Percy, Archibald Edward, 1404 Murray St., Alexandria, La., ALR.
Perfect, Fred Rolleston, 1258 Penn Ave., Wyomissing, Pa., ALR.
Perkins, Edward Williamson, 312 Medical Arts Bldg., Richmond, Va., Op.
Pfingst, Harry A., 332 W. Broadway, Louisville 2, Ky., Op.
Piercy, Robert Lee, 613 Home Savings and Loan Bldg., Youngstown, Ohio, ALR.
Pliss, Aaron, 40 North St., Buffalo, N. Y., Op.
Presberg, Max Harold, 35 Chestnut St., Rochester 4, N. Y., Op.
Preston, William O., 733 South 44th St., Louisville, Ky., ALR.
Prindle, Ronald Edon, Capt. (MC), 20 Courtland St., Norwich, N. Y., ALR.
Pulliam, Robert Lee, Jr., Bowdine-Contril Clinic, 1735 N. Wheeler Ave., Portland 12, Ore., ALR.
Putney, Floyd Johnson, 255 S. 17th St., Philadelphia 3, Pa., ALR.

Rambo, Victor Clough, 110 Harvey St., Philadelphia 44, Pa., Op.
Rasgorshek, Robert Houston, 425 Aquila Court, Omaha, Nebr., Op.
Reed, Paul Henry, 727 W. 7th St., Los Angeles 14, Calif., Op.
Reel, Frederick Clifton, 432 Medical Arts Bldg., Charleston 1, W. Va., ALR.
Reese, Frederic Miller, Professional Bldg., 330 N. Charles, Baltimore 1, Md., Op.

Reid, Frederick Kessinger, Major (MC), 119 Manhatten Ave., Crestwood 7, N. Y., Op.

Reid, Wesley Grattan, 974 Fisher Bldg., Detroit, Mich., Op.

Rice, Theodore A., 390 Main St., Worcester, Mass., Op.

Ringer, Merritt Gartley, Col., (MC), Wm. Beaumont Gen. Hosp., El Paso, Tex., ALR.

Rodenberg Elmer Julius, 319 N. Elm St., Henderson, Ky., ALR.

Ross, Ervin Scott, 477 Riddle Rd., Cincinnati, Ohio, ALR.

Roth, Aaron, 133 E. 58th St., New York. N. Y., ALR.

Roth, Leslie William, 203 Commercial Bldg., Belleville, Ill., Op.

Rothstein, David Louis, 57 W. 57th St., New York 19, N. Y., ALR.

Sage, Thomas, 7815 E. Jefferson Ave., Detroit, Mich., ALR.

Sale, George Goble, 216 Dixon Bldg., Missoula, Mont., ALR.

Schmidtke, Reinhardt L., 441 Medical Arts Bldg., St. Paul 2, Minn., Op.

Schwartz, Herbert Norman, 99 Pratt St., Hartford, Conn., Op.

Seeley, Ralph Hunt, Jr., 79 Center St., Rutland, Vt., ALR.

Seeley, Robert Charles, 133 East 58th St., New York, N. Y., ALR.

Seitz, Howard M., 9 Main St., Wapakoneta, Ohio, ALR.

Seligman, Maurice, 405 N. Bedford Dr., Beverly Hills, Calif., ALR.

Shoemaker, Robert E., 1248 Hamilton St., Allentown, Pa., Op.

Shulruff, Harry I., 3701 Main St., East Chicago, Ind., Op.

Simmons, John T., 1501 First Ave., Jasper, Alabama, ALR.

Sleight, James Duane, 401 Security Bank Bldg., Battle Creek, Mich., Op.

Smith, Guy William, 1014 Republic Bldg., Denver, Colo., ALR.

Smith, Homer Edwards, 1005 Medical Arts Bldg., Salt Lake City, Utah, Op.

Smith, Thomas Leonard, 800 Broadway, Lorain, Ohio, ALR.

Snell, Albert Conrad, Jr., 53 South Fitzhugh St., Rochester 4, N. Y., Op.

Sooy, Francis A., Suite 808, 490 Post St. San Francisco, Calif., ALR.

Spiller, Arthur Eliot, 1148 5th Ave., New York, N. Y., Op.

Sputh, Carl Brosius, 224 N. Meridian, Indianapolis, Ind., ALR.

Stahl, Richard Holmes, 143 Portage Trail, Cuyahoga Falls, Ohio, ALR.
pitals, Iowa City, Iowa, ALR.

Steele, Clarence Hart, Ochsner Clinic, New Orleans, La., ALR.

Steele, Wendell M., Major (MC), Lawson General Hospital, Atlanta, Ga., Op.

Steine, Ben Zion, Medical Arts Bldg., Montreal, P.Q., Canada, ALR.

Sternberg, Paul, 58 E. Washington, Chicago 2, Ill., Op.

Stump, Robert Byron, 408 Hynds Bldg., Cheyenne, Wyo., Op.

Swan, Otis Dwight, Forum Bldg., 9th and K, Sacramento 14, Calif., ALR.

Tanner, Owen Ralph, 627 University Ave., Palo Alto, Calif., Op.

Toland, Virgil A., 509 W. McDowell Rd., Phoenix, Ariz., Op.

Treynor, Jack Vernon, 532 First Ave., Council Bluffs, Iowa, ALR.

Truitt, John William, 161 W. Wisconsin, Milwaukee, Wis., ALR.

Tyler, William H., University of Iowa Hospitals, Iowa City, Iowa, ALR.

Urmston, Paul Robert, 303 Davidson Bldg., Bay City, Mich., ALR.

Voltz, Vernon Carl, 303 N. Main St., Rockford, Ill., Op.

Von Leden, Hans Victor, 1046 Loyola Ave., Chicago, Ill., ALR.

Von Sallmann, Ludwig J. K., 635 W. 165th St., New York 32, N. Y., Op.

Walsh, Francis Mark, 330 Lowry Medical Arts Bldg., St. Paul 2, Minn., Op.

Weeks, Carrol Lamont, 1930 Wilshire Blvd,. Los Angeles 5, Calif., Op.

Weinstein, Lee, 1104 N. 2nd St., Harrisburg, Pa., ALR.

Weiss, Leon M., 30 East 60th St., New York 22, N. Y., Op.

Weiss, Louis, 7085 Jenkins Arcade, Pittsburg, Pa., ALR.

Welch, Robert Francis, 232 Main St., Brockton, Mass., ALR.

Weseman, Chester M., 2140 Shattuck Ave., Berkeley, Calif., ALR.

Whitmer, Kenneth Sweet, 711 duPont Bldg., Miami 32, Fla., Op.

Wickstrom, Otto William, U.S.N. Hosp. Quarters M., Great Lakes, Ill., ALR.

Wilcox, John Guinan, 624 N. Los Angeles St., Anaheim, Calif., ALR.

Wilson, James Edward, 899 Madison Ave., Memphis 3, Tenn., Op.

Wilson, John McCullough, 207 Watauga Ave., Johnson City, Tenn.,OP.

**Wilson, Warren Andrew, 1930 Wilshire Blvd., Los Angeles 5, Calif., Op.**
**Witchell, Ira S., 2021 Grand Concourse, New York 53, N. Y., ALR.**
**Witter, Gordon L., 2 West Fern Ave., Redlands, Calif., OALR.**
**Wong, Robert Tuck, 1147 Union St., Honolulu, T. H., Op.**
**Workman, Joseph B., Jr., 1515 Bull St., Columbia, S. C., Op.**

**Young, Clifton Aurelius, U. S. Naval Hospital, Great Lakes, Ill., ALR.**

**Zonderman, Bernard, 29 Bay State Rd., Boston, Mass., ALR.**

EXECUTIVE SECRETARY-TREASURER BENEDICT: "The Council submits the following nominations for officers for 1947:
President-Elect—Dr. C. H. McCaskey, Indianapolis, Indiana
First Vice-President—Dr. A. C. Jones, Boise, Idaho
Second-Vice-President—Dr. Phillips Thygeson, San Jose, California
Third Vice-President—Dr. Norton Canfield, New Haven, Connecticut
Councillor—Dr. W. Ivan Lillie, Philadelphia, Pennsylvania
Executive Secretary-Treasurer—Dr. William L. Benedict, Rochester, Minnesota
Secretary for Otolaryngology—Dr. O. E. Van Alyea, Chicago, Illinois
Secretary for Ophthalmology—Dr. Algernon B. Reese, New York, New York
Secretary for Instruction in Otolaryngology and Maxillofacial Surgery—Dr. Dean M. Lierle, Iowa City, Iowa
Secretary for Instruction in Ophthalmology —Dr. A. D. Ruedemann, Cleveland, Ohio
Secretary for Home Study Courses—Dr. Lawrence R. Boies, Minneapolis, Minnesota
Secretary for Public Relations—Dr. Erling W. Hansen, Minneapolis, Minnesota

PRESIDENT NEW: Will someone move that the officers recommended by the Council be elected?

. . . It was regularly moved and seconded that the officers recommended by the Council be elected . . .

PRESIDENT NEW: All those in favor say "aye"; opposed. It is so ordered.

EXECUTIVE SECRETARY-TREASURER BENEDICT: Time and place of next meeting: Palmer House, Chicago, October 12-17, 1947."

Mr. President, the report of the Senior Member of the Council is open for adoption.

PRESIDENT NEW: The report of the Senior Member of the Council is open for adoption. If it is your wish, will someone move that the report be adopted.

DR. J. MILTON ROBB: I move that the report be adopted

. . . The motion was seconded . . .

PRESIDENT NEW: All those in favor say "aye"; opposed. It is so ordered.

Will Dr. Robb and Dr. Ridpath please escort Dr. Woods, the President-Elect, to the platform?

PRESIDENT NEW: Dr. Woods, it gives me great pleasure to present you with the gavel and to wish you the greatest success during the coming year.

PRESIDENT-ELECT WOODS: Thank you, Dr. New.

I want to express my appreciation for the very great honor that I feel has been conferred on me and express my enormous devotion to what I think is the very high ideal of the Academy and the great good that this Academy has already done and I feel will mark its future.

I trust that next year will be a successful year and as successful a meeting as this very splendid meeting under the direction of Dr. New.

. . . President-Elect Woods then read his prepared paper, the Jackson Memorial Lecture. (See page 75.)

PRESIDENT-ELECT WOOD: There is one more item on the program, a motion picture.

. . . Showing of motion picture . . .

PRESIDENT-ELECT WOODS: This closes the meeting. Thank you very much.

. . . The business meeting adjourned at ten o'clock . . .

# COMMITTEE REPORTS

## REPORT OF THE ADVISORY COMMITTEE TO THE REGISTRY OF OPHTHALMIC PATHOLOGY

J. E. Ash, Colonel, M.C., U.S.A., Registrar
Brittain F. Payne, M.D., Chairman

July 1, 1945 — June 30, 1946

There were 2,446 accessions added to the Registry during the year, bringing the total number of cases to 20,272.

Material in the Registry was used in the preparation of the Seminar of the American Society of Clinical Pathologists held in San Francisco in June. The Seminar was conducted by Colonel J. E. Ash under the title "Pathology of the Special Senses".

It was necessary to have a new printing of the Atlas, which was a great improvement over the last one because of the use of better paper and improved facilities. The revision of the Atlas is getting under way again.

The Registry conducted the course in Pathology for the George Washington University Post Graduate Course in Ophthalmology; the ninth year for this activity. In addition, a number of doctors have spent varying times at the Registry studying the material and receiving individual instruction.

A chapter on Comparative Ophthalmology was prepared for inclusion in "Ophthalmology in the War Years" edited by Dr. Meyer Wiener of California.

---

## REPORT OF THE ADVISORY COMMITTEE TO THE REGISTRY OF OTOLARYNGIC PATHOLOGY

J. E. Ash, Colonel, M.C., U.S.A., Registrar
Frank J. Novak, Jr., M.D., Chairman

July 1, 1945 — June 30, 1946

On July 1, 1946, there were 2,804 cases in the Registry, 126 accessions having been added during the year.

The material in the Registry was used in the preparation of the Seminar of the American Society of Clinical Pathologists held in San Francisco in June, the subject of which was "Pathology of the Special Senses". The Seminar was conducted by Colonel J. E. Ash.

The number of loan sets has proved entirely inadequate to meet the demand and we are reconstituting several old sets that had a number of missing slides and have begun to cut ten additional sets. The revision of the Atlas is just getting under way again. There was a new printing of the Atlas, a great improvement over the last one because of the use of better paper and improved facilities.

Contracts have been made by the Office of the Surgeon General with Dr. John R. Lindsay of Chicago and Dr. Edmund P. Fowler of New York for assistance in cutting a large collection of temporal bones from aviators, acquired during the War. This will greatly facilitate this important study.

## REPORT OF THE COMMITTEE FOR REVISION OF PATHOLOGIC ATLASES

Brittain F. Payne, M.D., Chairman

The meeting of the Committee for Revision of Pathologic Atlases was held in the Palmer House October 13, 1946, with the following members present:

Brittain F. Payne, M.D., New York, N. Y., Chairman
Col. J. E. Ash, M.C., Washington, D. C.
Howard Douglas, Omaha, Neb.
Jonas S. Friedenwald, M.D., Baltimore, Md.
Frank J. Novak, Jr., M.D., Chicago, Ill.
Algernon B. Reese, M.D., New York, N. Y.
Benjamin Rones, M.D., Washington, D. C.
LeRoy A. Schall, M.D., Boston, Mass.
Herman Semenov, M.D., Beverly Hills, Calif.
Georgiana Dvorak-Theobald, M.D., Oak Park, Ill.
Mrs. Helenor Campbell Wilder, Washington, D. C.

The immediate demand for both Ophthalmic and Otolaryngic Atlases will be satisfied by the reprinting of 1,000 copies of the Atlas of Ophthalmic Pathology and 700 copies of the Atlas of Otolaryngic Pathology. Delivery of the reprinted atlases should begin within a period of six weeks, according to Col. J. E. Ash of the Registry of Pathology.

The details of the actual revision of the atlases were discussed at great length. It was decided that editorial boards will have actual control of the planning and scope of the new atlases with frequent consultations with other members of the Advisory Committee to the Registries of Pathology. (See Council Minutes, V, 4).

The Editorial Board for Ophthalmic Atlases will consist of:

Benjamin Rones, M.D., Chairman
Jonas Friedenwald, M.D.
Mrs. Helenor Campbell Wilder

The Editorial Board for Otolaryngic Atlases will consist of:

Herman Semenov, M. D., Chairman
J. E. Ash, Colonel, M.C., U.S.A.
Stacy Guild, Ph.D.

The Chairman of the Advisory Committee will be a member of each of the editorial boards ex-officio.

The duties of the editorial boards will be:

1. Preparation of the general outline to be followed throughout the atlases to be submitted to the Committee as a whole for additional comments.
2. After the outlines and plans of the revised atlases are accepted by the committee the editors, Mrs. Wilder and Colonel Ash, will begin their work on the texts.
3. The texts will be submitted to the editorial boards for suggestions and constructive criticism.
4. Samples of printing, etc., will be submitted by Colonel Ash and Mrs. Wilder for preparation of the "dummy" in order that estimates on costs may be secured from printers. The initial printing of 2,000 copies of each atlas and the costs of such printing will be submitted to the Committee as a whole.
5. Final recommendations of the Advisory Committee to the Registries of Pathology will be submitted to the Council for definite action.

---

## REPORT OF THE COMMITTEE ON THE CONSERVATION OF HEARING

DEAN M. LIERLE, M.D., Chairman

The Executive Committee of the Committee on the Conservation of Hearing held a meeting at the Palmer House in Chicago on May 26, 1946.

There has been a great deal of interest in regard to the organization of complete hearing clinics both in the larger cities and smaller towns. The Committee is very much encouraged and feels that much progress has been made in this direction.

During the year a manual to be used as a guide for the fitting of heaing aids has been written and will soon be ready for publication.

It is recognized that the average otologist in private practice does not have the facilities in many instances to fit hearing aids. Therefore, a working statement regarding the fitting of hearing aids was prepared by the Committee and published in the July-Aug. issue (p. 215) of the TRANSACTIONS.

The Committee has prepared and adopted an audiogram form. It is hoped that the chart may be universally adopted.

The Committee has recommended the establishment of additional training programs for audiologists in university clinics. A detailed outline of a practical course has been adopted as a pattern for proposed courses.

The Committee has recommended that the word "audiology" be recognized by the Academy and that the word be incorporated in the new dictionaries.

The Committee has recommended that an attempt be made to have the Academy set up a bureau to certificate and regulate audiologists.

A new kodachrome sound film for propaganda purposes has just been completed and will be shown during the scientific program at this (1946) meeting. The film was designed to acquaint the public with the treatment of early hearing losses. The Departments of Dramatic Arts and Otolaryngology of the State University of Iowa have cooperated with the Committee on the Conservation of Hearing in the production of the film.

It was generally agreed that the licensing of hearing aid salesmen was not a function of the Academy.

A directive for state representatives to aid in the establishment of hearing clinics has been proposed by the Committee.

A Committee has been appointed for the solicitation of funds for the activities of the Section on the Conservation of Hearing. It was agreed that a full-time otologist employed by the Committee is necessary to assist the state representatives in developing hearing clinics.

The question of affiliation of the Otosclerosis Study Group with the Committee on the Conservation of Hearing was considered again. It was the consensus that each group should function independently and the Oto-

sclerosis Study Group be recognized by the Academy as a separate organization.

A special program for the state representatives has been proposed by Dr. Raymond Carhart of Northwestern University for the meeting of the Conservation of Hearing on Sunday morning, October 13.

The scientific program for the Section on the Conservation of Hearing for the 1946 meeting of the Academy is as follows:

1. Fitting of Hearing Aids—Dr. Raymond Carhart and Lt. Eva Thompson.
2. Public School Hearing Program and Demonstration—Dr. C. E. Kinney
3. Demonstration in Lip-reading — Mr. Howard Carter and Dr. H. Westlake
4. Moving picture film on the treatment of early hearing losses.

The following problems will be considered during 1946-47:

1. Acoustic trauma in industry
2. Further development of complete hearing clinics
3. Universal program for the testing of school children
4. Development of courses for the teaching of audiologists
5. Registration of audiologists by the American Academy of Ophthalmology and Otolaryngology
6. Further propaganda for the hard of hearing program
7. Procurement of a paid, full-time otolaryngologist to assist state representatives in organizing hearing programs
8. Further cooperation of the American Academy of Ophthalmology and Otolaryngology with the Hard of Hearing Society

The Committee recommends that the sum of $3,000 be allocated to defray expenses for their activities during the ensuing year.

## REPORT OF THE COMMITTEE ON STANDARDIZATION OF TONOMETERS

JONAS S. FRIEDENWALD, M.D., Chairman

The work of our Committee has continued along the three main projects previously undertaken. I. Assisting manufacturers in the production of certifiable instruments, II. Checking instruments for ophthalmologists, III. Recalibration of standard instruments.

### *I. Certification of Tonometers*

Several manufacturers have cooperated with our Committee in developing standard specifications for Schiotz tonometers, and are attempting to produce instruments which will be submitted for certification. Our Committee is ready and eager to assist manufacturers in problems of design and structure, and has already been consulted by three of the leading manufacturers in regard to special problems that they have encountered. The chief difficulty in the manufacture of certifiable tonometers is not in the technical problems of design and production, but in the shortage of personnel in the industry.

In cooperation with the manufacturers of the Gradle tonometer, specifications for a new standard design have been worked out, and a model instrument of this design has been approved by the Committee.

In a previous report, our Committee has reported on the arrangements made for the certification of tonometers which conform to acceptable specifications. A commercial testing laboratory,—The Electrical Testing Laboratories, Inc., has been authorized to certify tonometers which are submitted to them by the manufacturers, and which are found on measurement to correspond to the specifications which our Committee has worked out in collaboration with the cooperating manufacturers. The Electrical Testing Laboratories, Inc., has undertaken to make these measurements at cost, and has worked out a sliding scale of charges per tonometer tested depending on the number of instruments submitted. The precise nature of the measurements to be made was worked out by our Committee in collaboration with the cooperating manufacturers, and the Electrical Testing Laboratories, Inc.

### *II. Tonometer Testing Stations*

Three tonometer testing stations are now in operation to which ophthalmologists can send instruments for testing:

1) Dr. Adolph Posner, 667 Madison Ave., New York 21, N. Y.
2) Dr Peter Kronfeld, 904 W. Adams Street, Chicago 7, Ill.
3) Dr. Frederick C. Cordes, Dr. Robert Shaffer, University of California Eye Clinic, Third Avenue and Parnassus, San Francisco 22, Calif.

At these stations instruments are weighed and measured and examined for structural imperfections. Owing to the fact that instruments have, in the past, been manufactured without any control of their structural characteristics and owing to the fact that most manufacturers have, in the past, failed to appreciate the significance of accuracy in design, a very large proportion of those instruments which are submitted to our testing

TABLE

| | *No. of Instruments Measured* | *No. Repaired* | *No. Found Satisfactory Without Recalibration* | *No. Recalibrated* | *Total No. Returned Serviceable* | *Condemned* |
|---|---|---|---|---|---|---|
| New York Testing Station | 19 | 4 | 7 | 8 | 17 | 2 |
| Chicago Testing Station | 124 | 28 | 22 | 98 | 120 | 4 |
| San Francisco Testing Station | 15 | 4 | 2 | 11 | 13 | 2 |
| Totals | 158 | 36 | 31 | 117 | 150 | 8 |

stations are found to deviate from Schiotz original design by very large factors. Plunger weights 20 per cent or more in error are the rule. Efforts have been made by our Committee to develop correction factors for the Schiotz scale to be applied to instruments that are mechanically satisfactory, but differ from the Schiotz standard in plunger weight or over all weight. When it is possible to correct for errors in design in this way, special tables or charts are supplied to the owner for use with his instrument. It should be clear that these recalibrations are applicable only individually to the particular instruments for which they are supplied.

In order to make these recalibrations, it has been necessary for the Committee to decide on a standard calibration for standard Schiotz instruments. When Schiotz first produced his tonometer, he made calibration studies and supplied charts based on these calibrations. The average normal intraocular pressure assumed in his calibration was 18-20 mm Hg. This calibration was subsequently criticized by a number of investigators, and in 1821 Schiotz published a second "corrected" chart in which the pressure readings were higher. The average normal intraocular pressure with this second calibration was 22 mm Hg. In 1937, the theory of tonometry was restudied by Friedenwald, and a further correction on Schiotz calibration was found necessary. With this calibration the average normal intraocular pressure was estimated to be 25 mm Hg.

The Committee in reviewing these three different calibrations, has concluded that the latter is probably nearest to the true values and has, in consequence, furnished to the owners of tonometers submitted for checking, calibrations of their instruments based on the Friedenwald chart.

*III. Recalibration of Standard Instruments*

The discussion in the previous section makes it clear that we still do not know with any reasonable accuracy what is the normal intraocular pressure, and while each ophthalmologist can determine for himself what is the normal range of reading with his tonometer, and while correction factors can be introduced in order to make different tonometers read equally on the same eye, a new investigation is required to avoid these ambiguities. The problem is the more urgent because the Gradle instruments were never directly calibrated manometrically, and the scale furnished with these instruments was based on a comparison of clinical readings made with a standard Gradle and a standard Schiotz. However, in the course of time, successive modifications in the design of the Gradle instrument have been made by the manufacturer without any corresponding adjustment in the calibration chart. Finally, the independent calibration studies of William McLean used in establishing the scale for his instrument are, as is well known, in wide disagreement with those of Schiotz.

In view of these large uncertainties in the calibration of tonometers, the Committee has undertaken an experimental restudy of the whole calibration problem. Special reference standard instruments have been obtained from manufacturers of the Schiotz, Gradle and McLean instruments, and direct measurements on enucleated eyes at manometrically determined intraocular pressures are being made with all three of these reference standard instruments. Several thousand measurements will be required in order to reach reliable calibration data, and the work is still in progress. It is to be hoped that acceptable new standard calibrations will result by which all three types of instruments can be brought

into agreement with each other. This calibration study is being conducted by Dr. Friedenwald.

*IV.*

In view of the fact that further work is required in the fields that we have been authorized to undertake, it is recommended that the Committee be continued.

## REPORT OF THE ADVISORY COMMITTEE OF THE EYE HEALTH COMMITTEE OF THE STUDENT HEALTH ASSOCIATION

LAWRENCE T. POST, M.D.

During my term as representative of the Academy on this Committee, there has been no meeting and no activity. Letters have recently been sent from the Chairman of the Committee and from Mrs. Merrill of the National Society for the Prevention of Blindness that we may expect activities to be resumed this fall.

## REPORT OF THE AMERICAN BOARD OF OPHTHALMOLOGY

A. B. REESE, M.D.

As senior member on the American Board of Ophthalmology of those appointed by the American Academy of Ophthalmology and Otolaryngology, I should like to report to the Council on the activities of the Board during the past year.

The following examinations were held and the results listed:

| | *Passed* | *Failed* | *Conditioned* |
|---|---|---|---|
| New York City<br>June 12-16, 1945 | 24 | 26 | 25 |
| Chicago<br>Jan. 18-22, 1946 | 22 | 12 | 16 |
| New York City<br>April 14-18, 1946 | 29 | 15 | 20 |
| San Francisco<br>June 18-22, 1946 | 20 | 12 | 11 |

*The officers for* 1945-46 *are*:

Chairman: Dr. Edward C. Ellett, Memphis, Tenn. V. Chairman: Dr. Georgiana D. Theobald, Oak Park, Ill.

Secretary-Treasurer: Dr. S. Judd Beach, Portland, Maine.

Assistant Secretary: Dr. Theodore L. Terry, Boston, Mass.

*The personnel of the Board for* 1946 *are*:

| *Term ends* | *Sec. A.M.A.* | *A.O.S.* | *Academy* |
|---|---|---|---|
| 12/31/46 | Berens | Ellett | Reese |
| 12/31/47 | Terry | Post | Cordes |
| 12/31/48 | Goar | Beach | Masters |
| 12/31/49 | Dunningon | Theobald | Vail |

*Examinations scheduled for the future are*:

October 9-13, 1946, Chicago

June, 1947 (AMA) New York or Philadelphia

October, 1947 (Academy) Chicago.

*Statistics for the Board are as follows*:

Eighty-seven examinations have been held and the total number of certificates issued to date are 2,522.

Forty-four ophthalmologists have served on the Board since its organization in 1917.

One hundred applications have been received during the fiscal year ending April 30, 1946.

## REPORT OF AMERICAN BOARD OF OTOLARYNGOLOGY

DEAN M. LIERLE, M.D.

In May, 1946, in Chicago 55 candidates were examined. Of this number 13 were conditioned and 5 were failed. Passed, 37.

In October, 1946, in Chicago 74 candidates were examined. Of this number 19 were conditioned and 5 were failed. Passed, 50.

## REPORT OF THE AMERICAN COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY

CONRAD BERENS, M.D.

A meeting was held June 27, 1946, in San Francisco. Dr. Berens reported the activities of the Subcommittee on Research on Myopia and submitted a financial report for the year's expenses (1945). This report was accepted as read.

A second meeting was held July 3, 1946. A report prepared by Dr. Lester Stein was read suggesting an investigation of contact lens manufacturing along the lines of approval-testing practiced by the American Medical Association and an evaluation of existing ethics, methods of and personnel responsible for dispensing contact lenses. An article is to be prepared by Dr. Conrad Berens and Dr. F. A. Rosa for publication in the Journal of the American Medical Association and is to include the following points: 1. Historical account. 2. Should contact lenses be recommended. 3. If so, to whom—indications and contra-indications. 4. By whom should contact lenses be prescribed. 5. By whom fitted. 6. Glass versus plastic lenses. 7. Technic of fitting. 8. Impressions and adjustment. 9. Solutions to be used.

The Committee (in connection with its function as consultants to the Council on Physical Medicine of the A.M.A.) discussed the following optical merchandise:

*Ortho-Rater*: 1. Physically and optically reasonably accurate. 2. Advertising claims slightly exaggerated at present. Committee suggested changes in advertising copy. It was moved, seconded and carried that the previous report on the Ortho-Rater be reconsidered. 3. Dr. Swan is to prepare a report on the Ortho-Rater.

*Orthoptic Apparatus*: It was moved, seconded and carried that the Orthoptic Council may pass upon orthoptic apparatus at the request of the American Committee on Optics and Visual Physiology.

*Orthoptics-essentials of an approved school*: Referred to the Orthoptic Council.

*Artificial eyes*: Allowable claims in advertising and glass versus plastic. Moved, seconded and carried that this question be deferred.

*Prisms*: Allowable error in accuracy of prisms—Plus or minus 10 per cent error permissible was moved and carried.

*Sun glasses*: Allowable claims in advertising. It was moved, seconded and carried that advertising should specify how much more infra-red or ultraviolet radiation will be excluded by tinted lenses than by ordinary glass lenses.

---

## AMERICAN ORTHOPTIC COUNCIL

MURRAY MCCASLIN, M.D.

During the year 1945-46 no formal meeting of the American Orthoptic Council was held. However, on October 5th simultaneous oral-practical examinations were held for technician candidates in New York City and Chicago. In New York 5 candidates were examined by Doctors LeGrand Hardy, T. L. Boyes, and Frank Costenbader, and Misses Knauber, Stark, Enos, McPhail and Bair. In Chicago 4 candidates were examined by Doctor A. D. Prangen, Beulah Cushman, George Guibor, and T. D. Allen, and the Mrs. Darr and Kotlewsky, and Miss Heinen All candidates passed the examination and received certificates.

At the present time there are 81 certified orthoptic technicians, of whom 56 are active, and 69 are members of the American Association of Orthoptic Technicians.

On September 7th, 1946 written examinations were given to 14 new applicants in different cities in the U. S. Thirteen of these passed the written examinations and were permitted to take the oral-practical examinations held at the Northwestern University Medical School, Eye Department, on October 12th. Of this number 12 will be certified as proficient in the technical processes of orthoptics by award of the American Orthoptic Council technicians certificate.

Technicians are now being trained under the supervision of Dr. Francis H. Adler in Philadelphia, Drs. Ernest Sheppard and Frank Costenbader in Washington, Dr. Lawrence Post in St. Louis, and at centers in Milwaukee, Cleveland, Buffalo and Rochester. It becomes increasingly evident that additional training centers are needed, and should be established as soon as practicable. The demand for well trained orthoptic technicians is great.

During the spring of 1946 the first "Graduate Training Course" was given under the supervision of Dr. Lancaster and Miss Lancaster in Boston This was participated in by 26 certified technicians.

A Symposium Program, which was prepared for the joint technicians-ophthalmologists meeting in Chicago in 1945, was postponed until 1946.

The six period continuous course in orthoptics offered by Doctors Hardy, Boyes, Sheppard, Cushman, Guibor, and Costenbader for the meeting of the American Academy of Ophthalmology and Otolaryngology in 1945, was also postponed to 1946.

The prides for the 1945 major amblyoscope slide contest were awarded as follows: First and 2nd prizes to Mrs. Serena Herslof ($25 and $15), 3rd prize to Miss Dorothy Bair ($10).

Your committee respectfully recommends:

1. that the American Academy of Ophthalmology and Otolaryngology continue its sponsorship and support of the American Orthoptic Council and that it appoint for a period of three years, Dr. Frank Costenbader of Washington to replace Dr. Murray McCaslin, whose term of office expires.
2. that the American Academy of Ophthalmology and Otolaryngology continue its financial support of the American Orthoptic Council to the extent of $100.00, if such support becomes necessary during the year 1947.

---

## REPORT OF COMMITTEE ON INDUSTRIAL OPHTHALMOLOGY

HEDWIG KUHN, M.D.

During the past year we have lost from the Committee our beloved Chairman, Dr. Albert C. Snell, who has felt compelled to resign. He has been a great leader and the

wellspring of stimulating ideas and suggestions. Dr. Derrick Vail consented to take his place and will prove, we feel certain, to be a similar leader.

During the past several years your Committee has worked with a similar Committee from the American Medical Association as a Joint Committee on problems in industrial ophthalmology. Our activities may roughly be divided into three general categories:

a) Research
b) Education
c) Service

In research, numerous studies have been made of visual skills and of the visual defects existing in workers in many types of industrial operations. Technics have been studied and developed for visual screening of large numbers of employees, and for analyzing the findings in relation to job efficiency. We have been assisted in this work by the Bausch & Lomb Optical Company and Purdue University, Professor Joseph Tiffin of Purdue being an associate member of our Joint Committee. He has directed the statistical analyses, and he, together with his associates, have given us a great deal of assistance. Various comprehensive visual programs have been established in several large plants during the war and these have been studied in an attempt to evaluate the facts both as to better placement of individuals in jobs and as to efficiency in plant management.

Our findings have been presented to the profession and published in several periodicals; seminars have been instituted; and, courses of instruction in industrial ophthalmology have been started. This educational aspect is one which we hope will grow in scope and importance.

The service to industry consists in large part of:

1) Answering all sorts of specific inquiries. If sufficient information is at hand, we attempt to have some reseach started so that a scientific answer can be given.
2) Articles in certain lay journals.
3) Investigation of factory lighting in conjunction with illumination engineers.
4) An attempt to find out who among the ophthalmologists are now, and will be willing to enter the field of industrial ophthalmology.
5) Quality and types of eye protection.
6) Establishment of a blood serum bank for cases of epidemic keratoconjunctivitis for the middle west (Michael Reese Hospital).
7) Outlining the function of, and procedures in first aid for industrial nurses, with standard orders for first aid care of such injuries.
8) Cooperation with such organizations as the Public Health Service and the National Society for the Prevention of Blindness.
9) Cooperation with the Council of Physical Medicine of the American Medical Association.
10) Close association with Purdue Industrial Visual Institute.
11) Aid rendered those who are preparing articles and books on industrial medicine.
12) Many contacts with the British who are interesed in industrial eye problems.

Among other projects, we hope to proceed with investigative work on irradiation in modern industry:

a) Its hazards
b) Its exact effects on workers
c) Methods of reducing the hazards
d) A review of the problems in compensation and the furnishing to industry of adequate ophthalmologic service.

Two new members of the Committee, Dr. E B. Spaeth of Philadelphia and Dr. Richard G. Scobee of St. Louis, have recently been appointed to succeed Dr. Thomas Allen and Dr. Edwin B. Dunphy who are retiring.

At the last official meeting of the Committee, October 13, 1946, it was voted to change the name of the Joint Committee on Industrial Ophthalmology to the Joint Committee on Occupational Ophthalmology.

Our budget has been $500, half from the A.M.A. and half from the Academy. $250 was used to bring Hazel Hurst to the 1944 Academy meeting. The balance has not been touched. We would like to submit a bill of $101.64 for reprints widely distributed on request and otherwise. The Committee requests a continuance of the annual appropriation.

## OFFICERS *for* 1947

*President*
ALAN C. WOODS, M.D.
BALTIMORE, MARYLAND

*President-Elect*
CARL H. McCASKEY, M.D.
INDIANAPOLIS, INDIANA

*Executive Secretary-Treasurer*
WILLIAM L. BENEDICT, M.D.
ROCHESTER, MINNESOTA

*First Vice-President*
ARTHUR C. JONES, M.D.
BOISE, IDAHO

*Second Vice-President*
PHILLIPS THYGESON, M.D.
SAN JOSE, CALIFORNIA

*Third Vice-President*
NORTON CANFIELD, M.D.
NEW HAVEN, CONNECTICUT

*Secretary for Ophthalmology*
ALGERNON B. REESE, M.D.
New York, New York

*Secretary for Otolaryngology*
O. E. VAN ALYEA, M.D.
Chicago, Illinois

*Secretary for Instruction*
*Ophthalmology*
A. D. RUEDEMANN, M.D.
Cleveland, Ohio

*Secretary for Instruction*
*Otolaryngology*
DEAN M. LIERLE, M.D.
Iowa City, Iowa

***Secretary** for Home Study Courses*
LAWRENCE R. BOIES, M.D.
Minneapolis, Minnesota

*Secretary for Public Relations*
ERLING W. HANSEN, M.D.
Minneapolis, Minnesota

# American Academy of Ophthalmology and Otolaryngology

## THE COUNCIL — 1947

| | |
|---|---|
| Alan C. Woods, M.D., Baltimore, Md. | President |
| Carl H. McCaskey, M.D., Indianapolis, Ind. | President-Elect |
| Arthur C. Jones, M.D., Boise, Idaho | First Vice President |
| Phillips Thygeson, M.D., San Jose, Calif. | Second Vice President |
| Norton Canfield, M.D., New Haven, Conn. | Third Vice President |
| Lawrence T. Post, M.D., St. Louis, Mo. | Past President, Senior Member of Council |
| Gordon B. New, M.D., Rochester, Minn. | Past President |
| William L. Benedict, M.D., Rochester, Minn. | Executive Secretary-Treasurer<br>Editor and Business Manager of the Transactions |
| Algernon B. Reese, M.D., New York, N. Y. | Secretary for Ophthalmology |
| O. E. Van Alyea, M.D., Chicago, Ill. | Secretary for Otolaryngology |
| A. D. Ruedemann, M.D., Cleveland, Ohio | Secretary for Instruction—Ophthalmology |
| Dean M. Lierle, M.D., Iowa City, Iowa | Secretary for Instruction—Otolaryngology and Maxillofacial Surgery |
| Lawrence R. Boies, M.D., Minneapolis, Minn. | Secretary for Home Study |
| Erling W. Hansen, M.D., Minneapolis, Minn. | Secretary for Public Relations |
| Carl H. McCaskey, M.D., Indianapolis, Ind. | Councillor |
| James M. Robb, M.D., Detroit, Mich. | Councillor |
| C. D. Blassingame, M. D., Memphis, Tenn. | Councillor |
| W. Ivan Lillie, M.D., Philadelphia, Pa. | Councillor |

## COUNCIL COMMITTEES

### Executive, Research and Finance

| | |
|---|---|
| Alan C. Woods, M.D., *Chairman* | Baltimore, Md. |
| Lawrence T. Post, M.D. | St. Louis, Mo. |
| Gordon B. New, M.D. | Rochester, Minn. |
| Carl H. McCaskey, M.D. | Indianapolis, Ind. |
| William L. Benedict, M.D. | Rochester, Minn. |

### Research and Finance

(Subcommittee of Executive, Research and Finance Committee)

| | |
|---|---|
| Lawrence T. Post, M.D., *Chairman* | St. Louis, Mo. |
| Alan C. Woods, M.D. | Baltimore, Md. |
| William L. Benedict, M.D. | Rochester, Minn. |

### Activities

| | |
|---|---|
| Carl H. McCaskey, M.D., *Chairman* | Indianapolis, Ind. |
| James M. Robb, M.D. | Detroit, Mich. |
| C. D. Blassingame, M. D. | Memphis, Tenn. |
| W. Ivan Lillie, M.D. | Philadelphia, Pa. |
| Arthur C. Jones, M.D. | Boise, Idaho |

### Judicial

| | |
|---|---|
| Alan C. Woods, M.D., *Chairman* | Baltimore, Md. |
| Carl H. McCaskey, M.D. | Indianapolis, Ind. |
| James M. Robb, M.D. | Detroit, Mich. |
| C. D. Blassingame, M.D. | Memphis, Tenn. |
| W. Ivan Lillie, M.D. | Philadelphia, Pa. |

### Board of Councillors

| | |
|---|---|
| Carl H. McCaskey, M.D., *Chairman* | Indianapolis, Ind. |
| James M. Robb, M.D. | Detroit, Mich. |
| C. D. Blassingame, M.D. | Memphis, Tenn. |
| W. Ivan Lillie, M.D. | Philadelphia, Pa. |

### Board of Secretaries

| | |
|---|---|
| Wm. L. Benedict, M.D., *Chairman,* | Rochester, Minn. |
| Algernon B. Reese, M.D. | New York, N. Y. |
| O. E. Van Alyea, M.D. | Chicago, Ill. |
| A. D. Ruedemann, M.D. | Cleveland, Ohio |
| Dean M. Lierle, M.D. | Iowa City, Ia. |
| Lawrence R. Boies, M.D. | Minneapolis, Minn. |
| Erling W. Hansen, M.D. | Minneapolis, Minn. |

### Program

| | |
|---|---|
| Alan C. Woods, M.D., *Chairman* | Baltimore, Md. |
| Carl H. McCaskey, M.D. | Indianapolis, Ind. |
| Arthur C. Jones, M.D. | Boise, Idaho |
| William A. Mann, M.D. | Chicago, Ill. |
| General Chairman of Arrangements | |
| William L. Benedict, M.D. | Rochester, Minn. |
| Algernon B. Reese, M.D. | New York, N. Y. |
| O. E. Van Alyea, M.D. | Chicago, Ill. |
| A. D. Ruedemann, M.D. | Cleveland, Ohio |
| Dean M. Lierle, M.D. | Iowa City, Ia. |
| Lawrence R. Boise, M.D. | Minneapolis, Minn. |
| Erling W. Hansen, M.D. | Minneapolis, Minn. |

# STANDING COMMITTEES

## Advisory Committee to the Registries of Pathology

Brittain F. Payne, M.D., *Chairman,* New York, N. Y.
Col. J. E. Ash, M.C., *Ex Officio,* Washington, D. C.
Andrew A. Eggston, M.D. ..... New York, N. Y.
Edmund P. Fowler, M.D. ..... New York, N. Y.
Jonas S. Friedenwald, M.D. ..... Baltimore, Md.
Stacy Guild, Ph.D. ..... Baltimore, Md.
Lyman H. Heine, M.D. ..... Fremont, Nebr.
Michael J. Hogan, M.D. ..... San Francisco, Calif.
John E. L. Keyes, M.D. ..... Cleveland, Ohio
John S. McGavic, M.D. ..... Phoenixville, Pa.
Bernard J. McMahon, M.D. ..... St. Louis, Mo.
Frank J. Novak, Jr., M.D. ..... Chicago, Ill.
Algernon B. Reese, M.D. ..... New York, N. Y.
Benjamin Rones, M.D. ..... Washington, D. C.
Theodore E. Sanders, M.D. ..... St. Louis, Mo.
LeRoy A. Schall, M.D. ..... Boston, Mass.
Herman Z. Semenov, M.D. ..... Beverly Hills, Calif.
Georgiana Dvorak Theobald, M.D. ..... Oak Park, Ill.

## Conservation of Hearing

*Executive Committee*

Dean M. Lierle, M.D., *Chairman* ..... Iowa City, Ia.
Ernest M. Seydell, M.D., *Secretary,* Wichita, Kans.
Gordon Berry, M.D. ..... Worcester, Mass.
George M. Coates, M.D. ..... Philadelphia, Pa.
A. C. Furstenberg, M.D. ..... Ann Arbor, Mich.
William E. Grove, M.D. ..... Milwaukee, Wis.
Gordon D. Hoople, M.D. ..... Syracuse, N. Y.
Marvin F. Jones, M.D. ..... New York, N. Y.
Charles E. Kinney, M.D. ..... Cleveland, Ohio
Carl H. McCaskey, M.D. ..... Indianapolis, Ind.
Marion R. Mobley, M.D. ..... Florence, S. C.
Harris P. Mosher, M.D. ..... Marblehead, Mass.
Werner Mueller, M.D. ..... Boston, Mass.
C. Stewart Nash, M.D. ..... Rochester, N. Y.

*Consultants*

Raymond Carhart, Ph.D. ..... Evanston, Ill.
Howard Carter, B.S. ..... Chicago, Ill.
Stacy Guild, Ph.D. ..... Baltimore, Md.
Scott N. Reger, Ph.D. ..... Iowa City, Ia.
Lt. Eva Thompson ..... Philadelphia, Pa.
Harold Westlake, Ph.D. ..... Evanston, Ill.

## Standardization of Tonometers

Jonas S. Friedenwald, M.D., *Chairman* ..... Baltimore, Md.
Francis Heed Adler, M.D. ..... Philadelphia, Pa.
Harry S. Gradle, M.D. ..... Sherman Oaks, Calif.
Trygve Gundersen, M.D. ..... Boston, Mass.
D. O. Harrington, M.D. ..... San Francisco, Calif.
Peter C. Kronfeld, M.D. ..... Chicago, Ill.
John M. McLean, M.D. ..... New York, N. Y.
Adolph Posner, M.D. ..... New York, N. Y.
Algernon B. Reese, M.D. ..... New York, N. Y.

## Scientific Exhibit

James H. Maxwell, M.D., *Chairman,* ..... Ann Arbor, Mich.
Kenneth L. Roper, M.D. ..... Chicago, Ill.

## Undergraduate Instruction in Ophthalmology

Lawrence T. Post, M.D., *Chairman,* St. Louis, Mo.
Alan C. Woods, M.D. ..... Baltimore, Md.
Edwin P. Dunphy, M.D. ..... Boston, Mass.
Derrick T. Vail, M.D. ..... Chicago, Ill.

## Undergraduate Instruction in Otolaryngology

James H. Maxwell, M.D., *Chairman* ..... Ann Arbor, Mich.
Lawrence R. Boies, M.D. ..... Minneapolis, Minn.
Dean M. Lierle, M.D. ..... Iowa City, Ia.
Fletcher D. Woodward, M.D., Charlottesville, Va.

*Representatives of the Academy on:*

## Advisory Committee to the Eye Health Committee of the American Student Health Association

Lawrence T. Post, M.D. ..... St. Louis, Mo.

## American Association for the Advancement of Science

M. Elliott Randolph, M.D. ..... Baltimore, Md.

## American Board of Ophthalmology

Frederick C. Cordes, M.D., San Francisco, Calif.
Robert J. Masters, M.D. ..... Indianapolis, Ind.
Derrick T. Vail, M.D. ..... Chicago, Ill.
Algernon B. Reese, M.D. ..... New York, N. Y.

## American Board of Otolaryngology

Frank R. Spencer, M.D. ..... Boulder, Colo.
Carl H. McCaskey, M.D. ..... Indianapolis, Ind.

## American Committee on Optics and Visual Physiology

Alfred Cowan, M.D. ..... Philadelphia, Pa.
Kenneth C. Swan, M.D. ..... Portland, Ore.
Frederick C. Cordes, M.D. ..... San Francisco, Calif.

## American Orthoptic Council

Avery deH. Prangen, M.D. ..... Rochester, Minn.
John B. Hitz, M.D. ..... Milwaukee, Wis.
Frank D. Costenbader, M.D. ..... Washington, D. C.

## Joint Committee of Occupational Ophthalmology

Edmund B. Spaeth, M.D. ..... Philadelphia, Pa.
John B. Hitz, M.D. ..... Milwaukee, Wis.
Richard G. Scobee, M.D. ..... St. Louis, Mo.

# THE MANAGEMENT OF OTOGENIC INFECTION

MERRILL B. HAYES, M.D.
CHESTER, PA.

*and*

C. FREMONT HALL, M.D.
BY INVITATION
NEW YORK, N. Y.

OTOLOGIC practice in the temperate zones can be divided generally into two distinct seasonal phases. One is the summer phase, in which conditions are essentially the same both indoors and out as regards temperature and humidity. In the second, or winter phase, we live in different environments, the dry, warm indoors, and the variable, cold outdoors. During this phase the human economy is subject to great fluctuations in temperature and humidity over relatively short periods of time. As a result, we have our fall, winter and spring colds with their ensuing complications, not the least of which are otogenic infections such as otitis media and mastoiditis.

During the summer phase we have relatively little of this type of infection but a great increase in *external* otogenic infections. In addition, we frequently encounter exacerbations of chronic otogenic infections due to artificial activation of pathologic otologic changes resulting from swimming, excessive sweating and bathing.

If otologic infections are studied bacteriologically, it will be found, on the one hand, that the winter varieties are due predominately to gram-positive organisms. It has been noted by many observers, on the other hand, that the primary infective agents in the external or summer type of otogenic infection are not the fungus nor the gram-positive organisms but the pyocyaneus or proteus groups. Often in external otitis the infection is mixed with both gram-negative and gram-positive organisms present. With the recent advance in chemotherapy and antibiotic therapy, it has been found time and again that the gram-positive organisms can be eliminated and yet have the gram-negative organisms flourish unabated.

During the past five years we have observed a great many patients with ear infections of all types. In their treatment we have been able to eliminate, in a large measure, the gram-positive organisms as a cause of morbidity. In 6 cases of mastoid infections treated by surgery during the past five years, the infecting organism proved to be Pseudomonas aeruginosa. In view of these findings, we embarked on a search for a substance which would be as effective against the gram-negative group of organisms as the sulfa drugs and penicillin are against the gram-positive group.

We shall apply our findings to three essential groups of otologic pathology: (1) acute otitis media, (2) chronic suppurative otitis media, and (3) otitis externa.

By far the greatest number of ear infections are acute otitis media. The infective agent has been variously reported as from 65 per cent to 90 per cent streptococci, 5 per cent to 15 per cent staphylococci, 5 per cent to 20 per cent pneumococci, and the remaining a miscellane-

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Illinois.

ous group.[7,17] It is our belief that all chronically infected ears were due initially to invasion by a micro-organism. This does not discount the fact that anatomic variations and altered physiology may well be a major contributing factor in preventing resolution. Nevertheless if there were no infective agent, we would have extremely few chronically infected ears.

If acute otitis media is diagnosed and treated early in its course with our present readily available armamentarium, resolution takes place within a month and complications are few. When complications do occur, and if the condition becomes chronic, we have frequently noted the presence of Ps. aeruginosa against which our newer drugs are not effective. Initially, these infections were due probably to the usual streptococci or staphylococci. However, a culture from an acutely infected ear which under adequate modern treatment persists in discharging over six weeks, almost always will show the presence of a gram-negative organism.

Chronic suppurative otitis media is always difficult to relieve by medical treatment because so many pathologic factors may be involved, such as enlarged or diseased tubal lymphoid tissue, chronic infection of the eustachian tube, with resulting stricture and granulation, polypoid tissue in the middle ear cavity and mastoid antrum, necrosis of the ossicular chain, chronic infection of the membranous lining of the mastoid cells, bony necrosis of the mastoid cells and cholesteatomatous deposits in the middle ear and mastoid antrum.

To treat adequately these chronic ears, whether the drum be destroyed, perforated marginally or centrally, extreme cleanliness is the first prerequisite. There is little question that with local cleansing, removal of excessive tubal tissue, granulation and polyps, many cases will cease discharging for varying periods of time but the discharge will often recur. In such cases infection, as a rule, is deep-seated, many cellular layers being involved. These can be treated adequately only by a substance that circulates through the blood and lymphatic channels and thus reaches every part of the infective process. Local application will not do that; it must be systemic. This may be related to the observation of Abraham and Chain[1] and Woodruff and Foster[16] that such gram-negative organisms as Ps. aeruginosa (B. pyocyaneus), coliform bacteria and proteus produce an enzyme, penicillinase, which destroys the bacteriostatic action of penicillin. To combat this problem, a group of antiseptics were employed in otogenic infections which had proved to be active against the gram-negative bacteria, particularly those of the mucoid variety such as proteus and Ps. aeruginosa.

We have observed in our present study that in chronic suppurative otitis media local cleansing and systemic medication alone will often result in disappearance of the granulations and positive cultures for gram-positive organisms, but the gram-negative forms remain and flourish in many cases.

The third variety of otogenic infection, external otitis, has been shown by many observers[2,3,9] and by ourselves to be due predominately to the gram-negative Ps. aeruginosa. We feel that the concept of otitis externa has changed in recent years. In the past there has been overemphasis upon the fungi as the etiologic agent.[5,6,8] This present work suggests the significance of Ps. aeruginosa as an important etiologic factor. In the past this organism was referred to only as the ubiquitous organism which overgrew many of the cultures. The pathogenicity of the organism was generally disregarded. It is our opinion, in light of not only this work but also in view of several recent surveys, that the fungi are relatively secondary in importance to

Ps. aeruginosa. (Salvin and Lewis,[12] Senturia,[13] Quayle,[11] Nelson,[10] and Syverton, et al.[14])

The problem of otitis externa still remains a complicated one that frequently comes first to the attention of the dermatologist and later may be referred to the otologist. We have tried to group the cases in this study into otitis externa diffusa, furunculosis, eczematous type of otitis externa, and otomycosis. Of course, one type often merges with another. Those cases grouped under otitis externa diffusa ranged from severe cellulitis to chronic wet external auditory canals. Although external otitis is generally considered an acute condition in which the entire canal is inflamed and edematous, we included chronic cases, many of which had acute flare-ups. The typical "swimmers' ears" fell into this category. Those cases with exfoliation and a tendency to crusting and exudation, as well as those with dry scaling, we termed eczematous. Some were undoubtedly seborrheic dermatitis, others were impetigo and intertrigo, but they all were grouped under the eczematous otitis heading.

As this present study progressed, we were surprised to note the frequency of the presence of Ps. aeruginosa in ears whose condition we had felt previously was probably due to fungus infections, and in chronic otitis media which had been under treatment. We noted also that only a very few of these had the greenish pus commonly associated with pyocyaneus infections. Organisms isolated from these cases when placed in suitable artificial culture media did produce heavy pigmentation in many instances. Although only a few normal ears were cultured, our results confirm the findings of Syverton, et al.,[14] and Salvin and Lewis[12] in that Ps. aeruginosa rarely appeared in normal ears, but did appear frequently in pathologic ears. Recent investigators, especially Salvin and Lewis,[12] Syverton, et al.,[14] and Williams, et al.,[15] seem to corroborate this opinion.

In a group of 100 medically treated cases of external otitis surveyed by Friedman and Hinkel,[4] the acute cases, that is those under one week's duration, presented little or no problem in treatment. In the chronic group medical treatment failed in 72 per cent of the cases and required treatment for as long as six months in 19 per cent of the cases. In their group of roentgen-treated cases, 36 per cent were listed as failures and 20 per cent had recurrences.

In this study, cultures were taken from all ears after thorough cleansing of the concha and outer auditory canal, but before treatment was instituted. The cultures were taken with a sterile applicator and streaked on blood agar plates. Sabouraud's medium and thioglycollate media were inoculated. Details of cultivation and isolation will not be discussed at length but results are shown in *Tables 1* through *6*.

In our search for an effective agent against gram-negative bacteria* and, in particular, against the Ps. aeruginosa, many compounds were tried. The most satisfactory one found in the early part of this work was Monobromsaligenin.** This substance used in 5 per cent concentration in polyethylene glycol proved to be effective against the gram-negative organisms. In the course of this study, dibromsalicylaldehyde,*** which in the tables and for ease of reference will be termed "Compound No. 280" proved to be equally effective against the gram-negative organisms, and, in addition, was found to be very active against fungi.

*Contact was made with Dr. John H. Brewer, Director of Biological Research, Hynson, Westcott and Dunning, Baltimore, Maryland, who was conducting a survey of compounds active against gram-negative organisms. Arrangements were made with him for the bacteriologic work and also for the several compounds discussed in this paper.

**Bromsalizol.

***Dibromsalicylaldehyde is a crystalline compound, light yellow in color, of which the empirical formula is $C_7H_4O_2Br_2$. The molecular weight of the compound is 280, and its melting point is 81-83° C.

A comparison with similar fungistatic agents will be found in *Table 7*. Compound No. 280 is about ten times as active as the proprietary compounds tested containing sodium propionate and propionic acid or undecylenic acid and zinc undecylenate.

The antibiotic activity of Compound No. 280 was checked not only against a number of gram-negative and gram-positive organisms (*Table 8*), but also against the mixed cultures from six of our cases (*Table 9*).

### PROCEDURE WITH COMPOUND NO. 280

Compound No. 280, prepared in a 1½ per cent borax solution, was used in testing for antibacterial activity. Test tubes containing 10 per cent horse serum in pork infusion broth were set up. Dilutions of Compound No. 280, ranging from 1-200 to 1-12,800, were made and inoculated with a 4 mm. loop of a twenty-four hour culture of the test organism. Both gram-positive and gram-negative bacteria were tested against Compound No. 280. The Staphylococcus aureus 209 is the Department of Agriculture's standard test strain. Streptococcus C-203 has been used in testing penicillin and the sulfonamides. Ps. aeruginosa (pyocyaneus), Shigella dysentery (Shiga), and all the other organisms tested, other than those in the mixed cultures from this study, were taken from isolations in the autopsy laboratory at the Johns Hopkins Hospital. All tubes were incubated twenty-four hours at 37° C and then read. Cup-plates prepared according to the standard F.D.A. technic were set up to show the millimeters of inhibition against the mixed cultures taken from the soldiers' ears.

### TREATMENT

Depending upon the result of cultures, the treatment was instituted as follows:

1. Chronic suppurative otitis media with culture of predominantly gram-positive organisms were given 20,000 units of penicillin intramuscularly every three hours until the gram-positive organism disappeared and the gram-negative organisms, especially Ps. aeruginosa, flourished. These ears were cleaned and irrigated with a 1 per cent solution of Compound No. 280 once or twice daily, followed by insufflation of the Compound No. 280 powder. The powder consisted of the following:

| | |
|---|---|
| Compound No. 280 | 2% |
| Borax | 3% |
| Talc | 95% |

2. Treatment was instituted in otitis externa cases immediately following initial culture. All of these cases, regardless of the type, were cleaned as thoroughly as possible, depending upon the amount of cellulitis present. In the cases of otitis externa diffusa, a wick of ¼" gauze saturated with 2 per cent Compound No. 280 in carbowax was introduced into the canal. This can remain from twenty-four to seventy-two hours and another wick inserted. Occasionally the patient will complain of a burning sensation which may persist for as long as three hours. This procedure may be repeated, depending upon the clinical appearance. Often one wick is sufficient in these cases. In the eczematous type of external otitis, treatment consisted either of insertion of a wick or simple local application for twenty-four hours, followed by cleansing with dry wipes and an insufflation of the Compound No. 280 powder. In those cases where there may be simply slight weeping, insufflation will suffice. The powder may be insufflated at daily to weekly intervals, depending upon the appearance of the lesions. In this study 1 case of sensitivity to Compound No. 280 was found. The reaction was that of a complicating contact dermatitis. In subsequent observa-

tions not included in this group, only 1 other case of sensitivity was encountered.

Of the 71 cases of external otitis studied (*Tables 1, 2* and *3*), 42 cases revealed the presence of Ps. aeruginosa, and, in the remainder, gram-negative organisms were usually present. The exceptions were the 2 cases of otomycosis. Only 21 of the 71 cases were under three weeks' duration and these responded readily to treatment either with the original Monobromsaligenin or with the Compound No. 280. Even without the initial cleansing of the canal, response to the treatment proved satisfactory. These cases for the most part resolved in less than one week. Relief of symptoms often occurred in less than twenty-four hours.

Fifty of the cases of external otitis were chronic, many being of the recurrent type and having lasted up to twenty years. Thirty resolved in one week or less and the remainder required two to four weeks' treatment. Two cases failed to return, and 1 case was seen at infrequent intervals and was classified as a failure. Ten cases were checked bacteriologically after thirty days and found to be negative. Only 3 cases were seen after six months, although many were followed up to three months. With the exception of 1 case of seborrheic dermatitis, no recurrences were seen. In this case only the local auricular lesion was treated and responded promptly.

In the treatment of otitis externa, Compound No. 280 caused a more rapid alleviation of symptoms and final resolution than the original Monobromsaligenin.

This study included 28 cases of otitis media (*Tables 4* and *5*). With little regard to underlying foci or extent of lesion, these cases were treated systemically or locally, as indicated in *Tables 4* and 5. It is again interesting to note that of these 28 cases, 17 revealed the presence of Ps. aeruginosa and only 4 cases suggested that we were dealing with a gram-positive organism as an etiologic agent. In many instances, however, penicillin or sulfadiazine had been used before a culture was obtained. Twelve of the treated ears became dry under two weeks and six of these had Compound No. 280 insufflation alone. Two radical cavities cleared up in less than two weeks. In 1 case of chronic otitis media of four years' duration, complicated by external otitis, complete resolution with a healed drum occurred. The remaining cases required up to two and a half months' treatment. Eight of these did not become dry and these individuals were separated from the army.

## SUMMARY

1. In the temperate zone otogenic infections are essentially of two types: winter type infections due to gram-positive organisms and summer type infections due to gram-negative organisms.
2. Infections by gram-positive type of bacteria can be treated adequately with the sulfa drugs and other antibiotics. The gram-negative organisms do not respond to these drugs.
3. One hundred cases with 160 cultures were studied bacteriologically and clinically.
4. It was found that the resistant etiological agent in chronic suppurative otitis media and external otitis was predominantly Ps. aeruginosa.
5. A new compound, dibromsalicylaldehyde, proved to be especially effective against gram-negative organisms, including Ps. aeruginosa, and also against fungi.
6. Dibromsalicylaldehyde can be utilized in a powder, ointment and solution.
7. Of 71 cases of external otitis, relief of symptoms occurred within 24 hours. Thirty resolved in one week

or less and the rest required two to four weeks' treatment. Three cases were classified as failures because they failed to return for treatment. Only one case of seborrheic dermatitis returned with a recurrence, although follow-up was inadequate.

8. Of 28 cases of otitis media, 12 became dry in two to six weeks; the remainder required up to two and a half months' treatment. Only 8 cases failed to become dry.
9. In our cases, dibromsalicylaldehyde proved to be an effective agent in the treatment of gram-negative and fungus infections of the ear.

**Table 1**

**OTITIS EXTERNA**

**(Cases treated initially by other preparations followed by Monobromsaligenin*)**

| Case No. | Duration | Clinical Type | Bacteriologic Findings | Length of Treatment | Comment |
|---|---|---|---|---|---|
| 1 | 5 days | Furuncle | Salmonella morgani | 12 days | Resolved |
| 2 | 6 mos. | Diffusa | Staph., strep., diphtheroids | 26 days | Resolved |
| 3 | 6 mos. | Diffusa | Staph., diphtheroids | 30 days | Resolved |
| 4 | 2 mos. | Diffusa | Yeast, E. coli, staph., diphtheroids | 14 days | Resolved |
| 5 | 2 yrs. Recurrent | Diffusa | Ps. aeruginosa, staph. | 15 days | Resolved |
| 6 | 4 mos. | Diffusa | Ps. aeruginosa, E. coli, staph., diphtheroids | 23 days | Resolved |
| 7 | 2 yrs. in Persia | Eczematous | E. coli, beta strep., hemo. staph., diphtheroids | 30 days | Resolved |
| 8 | 4 days | Diffusa | Aerobacter aerogenes, S. morgani, staph., diphtheroids | 8 days | Resolved |
| 9 | $5\frac{1}{2}$ yrs. | Diffusa | Ps. aeruginosa, staph., diphtheroids | 37 days | Resolved |
| 10 | 2 mos. | Diffusa | A. aerogenes, proteus, staph., searcina | 60 days | Resolved |
| 11 | 6 wks. | Diffusa | Ps. aeruginosa, E. coli, staph. | 2 weeks | Resolved |
| 12 | 1 mo. | Diffusa | Ps. aeruginosa, hemo. Staph. aureus | 19 days | Resolved |
| 13 | 4 yrs. | Diffusa | Ps. aeruginosa, E. coli, staph., gram-pos. rods | 6 days | Resolved |
| 14 | 1 wk. | Diffusa | Ps. aeruginosa, paracolon bacillus, diphtheroids | 21 days | Resolved |
| 15 | 2 mos. | Diffusa | Ps. aeruginosa, E. coli, beta strep., staph., diphtheroids, spore-former | 3 days | Resolved |
| 16 | 4 mos. | Eczematous | Ps. aeruginosa, beta strep. | 3 days | Resolved |
| 17 | 2 wks. | Diffusa | Ps. aeruginosa, beta strep., Staph. albus, diphtheroids | 8 days | Resolved |
| 18 | 1 wk. | Diffusa | Both ears: Ps. aeruginosa, staph., diphtheroids | 7 days | Resolved |
| 19 | 4 yrs. | Diffusa | Ps. aeruginosa, alpha strep. | 8 days | Resolved |

*Bromsalizol.

**Table 2**

**OTITIS EXTERNA**

**(Cases treated by Bromsaligenin alone*)**

| Case No. | Duration | Clinical Type | Bacteriologic Findings | Length of Treatment | Comment |
|---|---|---|---|---|---|
| 1 | 20 yrs. | Diffusa | Beta strep., diphtheroids, proteus, yeast | 14 days | Resolved |
| 2 | 10 yrs. Recurrent | Eczematous | Staph., Ps. aeruginosa | 14 days | Resolved |
| 3 | 6 mos. | Diffusa | Ps. aeruginosa, aerogenes-coli group, staph., diphtheroids | 14 days | Resolved |
| 4 | 2 wks. | Diffusa | Ps. aeruginosa, staph. | 14 days | Resolved |
| 5 | 1 wk. | Furuncle | Ps. aeruginosa, staph. | 7 days | Resolved |
| 6 | 9 mos. | Eczematous | A. aerogenes, staph. | 1 day | No return |
| 7 | 1 mo. | Diffusa | Ps. aeruginosa, gram-pos. rods, staph., strep. | 2 wks. | Resolved |
| 8 | 2 wks. | Diffusa | Hemo. staph., strep., proteus | 8 days | Resolved |
| 9 | 1 mo. | Diffusa | Ps. aeruginosa, beta strep. | 6 days | Resolved |
| 10 | 2 mos. | Diffusa | Ps. aeruginosa, hemo. Staph. aureus | 9 days | Resolved |
| 11 | 2 yrs. | Diffusa | Ps. aeruginosa, strep., diphtheroids, Staph. aureus | 2 days | Resolved |
| 12 | 2 yrs. | Diffusa | Ps. aeruginosa, beta strep., Staph. aureus | 7 days | Resolved |
| 13 | 1 mo. | Diffusa | Paracolon bacillus, staph. | 2 days | Resolved |
| 14 | 22 mos. | Eczematous | Ps. aeruginosa, E. coli, staph., strep., diphtheroids | 2 days | Resolved |
| 15 | 2 wks. | Diffusa | Non-lactose fermenter, beta strep., hemo. staph., diphtheroids | 5 days | Resolved |

*Bromsalizol.

**Table 3**

**OTITIS EXTERNA**

**(Cases treated with Compound No. 280*)**

| Case No. | Duration | Clinical Type | Bacteriologic Findings | Length of Treatment | Comment |
|---|---|---|---|---|---|
| 1 | 2 wks. | Diffusa | Ps. aeruginosa, beta strep., staph. | 7 days | Resolved |
| 2 | 2 yrs. Recurrent | Otomycosis | Fungi, staph. | 16 days | Resolved |
| 3 | 6 wks. | Diffusa with excoriation | Ps. aeruginosa, A. aerogenes, gram-pos. rods in chains, staph., strep. | 21 days | Resolved |
| 4 | 6 mos. | Diffusa | Ps. aeruginosa, staph. | 7 days | Resolved |
| 5 | 4 mos. | Otomycosis | E. coli, Aspergillus niger | 7 days | Resolved |
| 6 | 2 wks. | Diffusa | Ps. aeruginosa, beta strep., hemo. Staph. aureus | 7 days | Resolved |
| 7 | 3 yrs. | Diffusa Acute Cellulitis 2 wks. | Ps. aeruginosa, beta strep., hemo. Staph. albus, diphtheroids | 30 days | Resolved |
| 8 | 1 wk. | Excoriative Severe Cellulitis (Aspergillus) | Aspergillus niger, Staph. albus, beta strep. | 14 days | Resolved |
| 9 | 8 wks. | Exfol. Derm. | Ps. aeruginosa, beta strep., alpha strep., short gram-pos. bacilli in pairs, hemo. Staph. aureus | 7 days | Resolved |
| 10 | 2 wks. | Diffusa (Green pus) | Ps. aeruginosa | 3 days | Resolved |

**Table 3—Continued**

**OTITIS EXTERNA**

**(Cases treatedwith Compound No. 280*)**

| Case No. | Duration | Clinical Type | Bacteriologic Findings | Length of Treatment | Comment |
|---|---|---|---|---|---|
| 11 | 1 wk. | Diffusa | Ps. aeruginosa, A. aerogenes, alpha strep. | | Resolved |
| 12 | 1 mo. | Diffusa | Coli-aerogenes group, hemo. staph., alpha and beta strep. | 7 days | Resolved |
| 13 | 6 mos. Recurrent | Diffusa | Ps. aeruginosa | 3 days | Resolved |
| 14 | | Diffusa | Bacterium bronchisepticum | 9 days | Resolved |
| 15 | 2 yrs. Recurrent | Diffusa | Hemo. Staph. aureus | 14 days | Resolved |
| 16 | 7 wks. | Diffusa | Ps. aeruginosa, Coli-aerogenes group, Staph. albus, alpha strep. | 7 days | Resolved |
| 17 | 7 wks. | Diffusa | Hemo. Staph. aureus, beta strep., diphtheroids | 1 day | Resolved |
| 18 | 4 yrs. Recurrent | Diffusa | A. aerogenes, hemo. Staph. albus | 3 days | Resolved |
| 19 | 3 wks. | Eczematous Severe excoriation | E. coli, hemo. Staph. albus, alpha strep. | 12 days | Resolved |
| 20 | 6 mos. | Eczematous | Non-lactose fermenter, beta strep., Staph. albus, diphtheroids | 2 days | Resolved |
| 21 | 4 mos. Recurrent P.W. | Diffusa | Aspergillus (white), beta strep., Staph. albus, Ps. aeruginosa | 6 wks. Periodic | Ps. aeruginosa persisted; pt. did not return |
| 22 | 3 mos. | Diffusa | A. aerogenes, hemo. aerobic gram-pos. rods | 2 days | Resolved |
| 23 | 6 wks. | Diffusa | Ps. aeruginosa, A. aerogenes, beta strep., diphtheroids | 1 day | Resolved |
| 24 | 2 wks. | Eczematous | Ps. aeruginosa, A. aerogenes, strep., staph. | 3 days | Resolved |
| 25 | 4 wks. | Diffusa | Gram-pos. aerobic spore-forming bacillus, beta and alpha strep. | 2 days | Resolved |
| 26 | 2 mos. Recurrent in summer 2x | Diffusa | Ps. aeruginosa | 3 days | Resolved |
| 27 | 1 mo. | Diffusa | Ps. aeruginosa | 7 days | Resolved |
| 28 | 30 days | Diffusa | Alcaligenes faecalis, hemo. Staph. aureus | 14 days | Resolved |
| 29 | 3 days | Furuncle | Ps. aeruginosa, beta strep., hemo. Staph. albus | 7 days | Resolved |
| 30 | 3 yrs. | Eczematoid | Ps. aeruginosa, fungus-penicillium, A. aerogenes, staph., diphtheroids | 30 days | Resolved |
| 31 | 1 wk. | Furuncle | Proteus, gram-pos. rods in chains (seen in thioglyc. broth), beta strep., diphtheroids | 4 days | Resolved |
| 32 | 3 wks. | Diffusa | Ps. aeruginosa, A. aerogenes, long thin gram-pos. rods, staph. | 6 days | Resolved |
| 33 | 3 days | Furuncle | Hemo. Staph. aureus, alpha strep. | 8 days | Resolved |
| 34 | 8 days | Diffusa | Coli-aerogenes group organism, diphtheroids | 7 days | Resolved |
| 35 | 14 days | Diffusa | Hemo. Staph. aureus, diphtheroids | 4 days | Resolved |
| 36 | 6 mos. Recurrent | Eczematous | Ps. aeruginosa, alpha strep., staph. | 6 days | Resolved |
| 37 | 4 mos. | Furuncle | Ps. aeruginosa | 6 days | |

*Dibromsalicylaldehyde.

**Table 4-A**
**CHRONIC OTITIS MEDIA**
**(Treated with Sulfadiazine or Penicillin followed by insufflation of Compound No. 280 powder*)**

| Case No. | Duration | Clinical Type | Bacteriologic Findings | Length of Treatment | Comment |
|---|---|---|---|---|---|
| 1 | 15 yrs. | | Ps. aeruginosa, staph., strep., diphtheroids | 16 days | Dry |
| 2 | 8 yrs. Recurrent Present attack 1 mo. | | Paratyphoid "B." S. morgani, staph. | 5 days | Dry |
| 3 | 33 yrs. | | Ps. aeruginosa, hemo. Staph. aureus, beta strep. | 40 days | Dry |
| 4 | 10 yrs. | | Hemo. Staph. aureus, diphtheroids | 2 wks. | Dry |
| 5 | 5 mos. | | Proteus, E. coli, Ps. aeruginosa, beta and alpha strep. | 30 days | Dry |

*Dibromsalicylaldehyde.

**Table 4-B**
**CHRONIC OTITIS MEDIA**
**(Treated with Sulfadiazine and Penicillin)**

| Case No. | Duration | Clinical Type | Bacteriologic Findings | Length of Treatment | Comment |
|---|---|---|---|---|---|
| 1 | 20 yrs. | | Coli-aerogenes group organism, staph., gram-pos. spore-former | 30 days | Improved |
| 2 | 15 yrs. | | Ps. aeruginosa, staph. | 30 days | Improved |
| 3 | 20 yrs. | | E. coli, proteus, beta and alpha strep., staph., diphtheroids | 30 days | Improved |

**Table 4-C**
**CHRONIC OTITIS MEDIA**
**(Treated with insufflation of Compound No. 280* alone)**

| Case No. | Duration | Clinical Type | Bacteriologic Findings | Length of Treatment | Comment |
|---|---|---|---|---|---|
| 1 | 12 mos. | | Ps. aeruginosa, beta strep., staph. | 1 wk. | Dry |
| 2 | 1 yr. | | Gram-neg. (salmonella), beta strep., diphtheroids | 4 days | Dry |
| 3 | 8 mos. P.W. | | Ps. aeruginosa, A. aerogenes, staph., strep. | 5 days | Dry |
| 4 | 6 yrs. | Radical Cav. | Ps. aeruginosa | 14 days | Dry |
| 5 | 4 yrs. | Radical Cav. | Ps. aeruginosa, alpha strep. | 4 days | Dry |

*Dibromsalicylaldehyde.

**Table 5-A**
**CHRONIC OTITIS MEDIA COMPLICATED BY EXTERNAL OTITIS**
**(Treated with Penicillin and or Sulfadiazine followed by Compound No. 280*)**

| Case No. | Duration | Clinical Type | Bacteriologic Findings | Length of Treatment | Comment |
|---|---|---|---|---|---|
| 1 | 4 yrs. | | Hemo. staph. | 28 days | Improved |
| 2 | 16 yrs. | | Ps. aeruginosa, staph., beta strep. | 4 mos. | Dry |
| 3 | 35 yrs. | | Ps. aeruginosa, A. aerogenes, beta strep., staph. | 14 days | Canal improved but ear not dry |
| 4 | 6 mos. | | Ps. aeruginosa, Staph. aureus, strep., E. coli | 75 days | Dry |
| 5 | 20 yrs. | | Ps. aeruginosa, staph. | 20 days | Improved |
| 6 | 4 yrs. | | Ps. aeruginosa, alpha and beta strep., staph. | 30 days | Drum healed and canal clear. |
| 7 | 20 yrs. | | Ps. aeruginosa, A. aerogenes, staph., diphtheroids. | 30 days | Dry |
| 8 | 25 yrs. | | Ps. aeruginosa, hemo. staph., strep. | 30 days | Improved |
| 9 | 26 yrs. | | Ps. aeruginosa, Staph. aureus, alpha strep. | 14 days | Dry |
| 10 | 20 days | | Staph., diphtheroids | 16 days | Dry |

*Dibromsalicylaldehyde.

**Table 5-B**
**CHRONIC OTITIS MEDIA COMPLICATED BY EXTERNAL OTITIS**
**(Treated with Compound No. 280* alone)**

| Case No. | Duration | Clinical Type | Bacteriologic Findings | Length of Treatment | Comment |
|---|---|---|---|---|---|
| 1 | 8 yrs. | | Ps. aeruginosa, gram-pos. aerobic spore-former, staph. | 15 days | Dry |
| 2 | 20 yrs. | | Ps. aeruginosa, staph., beta strep. | 4 mos. | Dry |
| 3 | 4 yrs. | | Ps. aeruginosa, staph. | 10 days | Dry |
| 4 | 2 yrs. | | S. morgani, staph. | 5 days | Not followed |
| 5 | 19 yrs. | | Hemo. Staph. aureus, diphtheroids | 5 days | Dry |

*Dibromsalicylaldehyde.

**Table 6**
**NORMAL CASES**

| Case No. | Duration | Type | Bacteriologic Findings |
|---|---|---|---|
| 1 | | Normal | Hemo. staph., beta strep. |
| 2 | | Normal | Gram-pos. cocci. |
| 3 | | Normal | Hemo. Staph. aureus. |
| 4 | | Normal | Para-colon bacillus, staph. |

Table 7
CUP-PLATE TEST
Five Million Spores Trichophytonmentagrophytes.
Results in Millimeter Inhibition.

| Compound | | Dilution | | | |
|---|---|---|---|---|---|
| | | 1:200 | 1:2000 | 1:20,000 | 1:200,000 |
| No. 280 | 1:20 Complete | 23 mm. | 10 mm. | 8 mm. | — |
| No. 280* | 1:50 Complete | 28 mm. | 11 mm. | trace | — |
| Zinc Undecylenate and Undecylenic Acid | 1:18 15 mm. | 12 mm. | 6 mm. | trace | — |
| Propionic Acid and Sodium Propionate | 1:5 20 mm. | | | | — |
| B. S. | 20 mm. | 2 mm. | | | — |

Technic as described by Emmonds, A.J.P.H., Vol. 35, No. 8, August, 1945, p. 844.
*Bisulfite form.

Table 8
Antibiotic Activity of Compound No. 280.
Shown in Dilutions of (10% Horse Serum) Infusion Broth.

| Name of gram-positive organisms tested | Dilutions of Compound No. 280 | | | | | | |
|---|---|---|---|---|---|---|---|
| | 1000 | 2000 | 4000 | 8000 | 16,000 | 32,000 | 64,000 |
| Staphylococcus aureus (209) | — | — | — | + | ++++ | ++++ | ++++ |
| Streptococcus (C-203) | — | — | — | + | ++++ | ++++ | ++++ |

| Name of gram-negative organisms | Dilutions of Compound No. 280 | | | | |
|---|---|---|---|---|---|
| | 800 | 1600 | 3200 | 6400 | 12,800 |
| Escherichia coli | — | — | — | — | + |
| Proteus | — | — | — | + | +++ |
| Aerobacter aerogenes | — | + | + | ++ | +++ |
| Pseudomonas aeruginosa (pyocyaneus) | — | pcl | + | ++ | +++ |
| Shigella dys. (Shiga) | — | — | — | + | + |
| Friedlander's bacillus | — | + | ++ | +++ | ++++ |

Table 9
Antibiotic Activity of Compound No. 280 Tested in 10% Horse Serum Infusion Broth Against Mixed Cultures of Six Cases

| Case | No. 280 ..... 1%<br>Duponol ..... 1%<br>Borax ..... 1½% | | | | | | Cup-plate No. 280—1% |
|---|---|---|---|---|---|---|---|
| | 200 | 400 | 800 | 1600 | 3200 | 6400 | |
| No. 21 (Table 3) | — | — | pel. ++ | ++++ | ++++ | ++++ | Ring shadow of 5 mm. in 24 hrs. No true inhibition |
| No. 28 (Table 3) | — | — | pel. + | ++ | ++++ | ++++ | 5 mm. complete inhibition |
| No. 29 (Table 3) | — | — | + | pel. +++ | ++++ | ++++ | |
| No. 2 (Table 6) | — | — | + | pel. +++ | ++++ | ++++ | Less than 1 mm. complete inhibition |
| No. 30 (Table 3) | — | — | ++ | pel. ++++ | ++++ | ++++ | Slight trace of inhibition |
| No. 3 (Table 6) | — | — | ++ | +++ | flocculent sediment ++++ | ++++ | |

## CONCLUSIONS

This study was begun in 1941, before attention had been drawn to the fact that Ps. aeruginosa is an important etiologic factor in chronic ear infections. It was this observation which led the authors to search for a more effective chemical agent against this type of persistent infection. In a carefully controlled bacteriologic and clinical study of 100 cases of otitic infections, a new chemical agent, dibromsalicylaldehyde, has been found more satisfactory than other agents suggested thus far. Due to the unavailability of streptomycin, no opportunity has been afforded to study the efficacy of this antibiotic in these types of ear infections. Dibromsalicylaldehyde is a readily available, inexpensive chemical agent, which is effective against both bacteria and fungi.

## ACKNOWLEDGMENTS

The authors express their appreciation and thanks to Dr. Lloyd C. Felton, who prepared the compounds and various solutions employed in this study, and to Miss Beatrice Day for her assistance in isolating the organisms from the pathologic ear canals. Particular appreciation is expressed to Colonel Harry A. Clark, M.C., U. S. Army, under whose command most of the clinical work was conducted, and to Sergeant Thomas Pilon, whose assistance was of great aid in the technical management of the study.

## REFERENCES

1. Abraham, E. P., and Chain, E.: Enzyme from bacteria able to destroy penicillin, Nature, 146:837 (Dec. 28) 1940.
2. Daggett, W. I.: Desquamative otitis externa in Malta, J. Laryng. & Otol., 57:427-446 (Oct.) 1942.
3. Davis, E. L.: Mycotic ear infections at an advanced allied base, M. J. Australia, 2:437-438 (Nov. 27) 1943.
4. Friedman, H. S., and Hinkel, C. L.: External otitis: a study of the comparative merits of medical and roentgen therapy, Arch. Otolaryng., 33:749-757 (May) 1941.
5. Gill, W. D.: Mycotic infections in otolaryngology, South. M. J., 31:678-685 (June) 1938.
6. Gill, W. D.: Otitis externa, Ann. Otol. Rhin. & Laryng., 51:370-377 (June) 1942.
7. Kummel, W.: in Otologic surgery, Kopetzky, Hoeber, Ed. 2, 1929, p. 6.
8. Minchew, B. H., Collins, B. E. and Harris, M. M.: External ear disease with

special reference to the fungous type, South. M. J., 33:1345-1348 (Dec.) 1940.
9. Morley, G.: Otitis externa: "hot weather ear" investigation of 100 cases and method of treatment, Brit. M. J., 1:373-377 (Feb. 19) 1938.
10. Nelson, Roy F.: External otitis in the South Pacific, Ann. Otol., Rhin. & Laryng., 54:367-372 (June) 1945.
11. Quayle, A. F.: Otitis externus in New Guinea, M. J. Australia, 2:228-231 (Sept. 2) 1944.
12. Salvin, S. B. and Lewis, M. L.: External otitis, with additional studies on the genus pseudomonas, J. Bact., 51:495-506, 1946.
13. Senturia, Ben H.: Etiology of external otitis, Laryngoscope, 55:277-293 (June) 1945.
14. Syverton, J. T. et al: Otitis externa, Arch. Otolaryng., 43:213-225, 1946.
15. Williams, H. L., Montgomery, H. and Powell, W. N.: Dermatitis of the ear, J. A. M. A., 113:641-646 (Aug. 19) 1939.
16. Woodruff, H. B. and Foster, J. W.: Microbiological aspects of penicillin; bacterial penicillinase, J. Bact., 49:7-17 (Jan.) 1945.
17. Woodward, Fletcher: in Diseases of the nose, throat and ear, Jackson & Jackson, Saunders Co., Philadelphia, 1945, p. 282.

## DISCUSSION

BERNARD J. MCMAHON, M.D., St. Louis, Mo.: Dr. Hayes and Dr. Hall are to be congratulated upon the presentation of a very excellent paper. They have advanced a rather original hypothesis relative to the variations in bacterial etiology of infections of the ear according to seasons. We have always been under the impression that the usual organisms found in infections of the upper respiratory tract could be responsible for an otitis media, a mastoiditis or an otitis externa at any time during the year, not only as primary etiologic factors but also as secondary invaders irrespective of their reactions of the Gram stain. The only variations in etiology of which we have been cognizant over the course of years have been those which have occured during an endemic or epidemic series of infections of the middle ear or mastoid, in which the staphylococcus, streptococcus or pneumococcus proved to be the etiologic agents. Bacillus pyocyaneus has occurred in approximately two to three per cent of the cases. However, many of these were acute conditions.

The idea of gram-positive and gram-negative organisms having a specific vulnerability to certain drugs is not entirely new. After the introduction of mercurochrome 220 soluble, the statement was made that this chemical was bactericidal for gram-negative organisms. The theory was later advanced that gram-positive organisms were susceptible to the action of gentian violet.

In 1926, when there occurred in St. Louis large numbers of infants suffering from the diarrhea-vomiting complex, in which there was present an otitis media with or without a mastoiditis, and in which the diarrhea and vomiting were relieved by paracentesis or mastoidectomy, the discharge frequently persisted despite the relief of symptoms. Gentian violet was used in one mastoid cavity and middle ear and mercurochrome in the other in the same patient in certain cases without appreciable specific effect upon the presence or growth of either the gram-positive or the gram-negative organisms. It is interesting to note, therefore, that the speaker has been able to obtain such spectacular results with this new compound.

Bacillus pyocyaneus occurs as a secondary invader of otitis media and mastoiditis in patients whose resistance has been vitiated by prolonged infection or under-nourishment. The actual pathogenicity and toxicity of the organisms for humans is said to be practically negligible.

The bactericidal action of a drug, however, is not the only nor the most important consideration in the use of such drug. We must consider pre-eminently the effect which it may have upon the cells with which it comes in contact. Newer drugs should not be used empirically but should be subjected to more thorough analysis and tested for toxicity against cells growing in tissue culture, for the amount of irritation caused to the conjunctiva of the eye, for their capacity to destroy muscle on injection, to cause the peritoneal adhesions, and to influence the rate of wound healing, as stated by E. L. Howes. This investigator has applied these criteria to streptomycin, penicillin calcium and tyrothricin in determining the efficacy of these drugs upon the bacteriology of wounds in his studies of their rate of healing.

The tissue reaction to the contact of bactericidal drugs is actually far more important than the bactericidal effects, for what does it profit the human economy to remove evidence of infection, if in so doing we destroy the function of the part? Infection, irritation, overstimulation by medication cause the ingress of connective tissue cells into a tissue, and these, as we know, are not only phagocytic but the forerunners of fibrosis, scarring, strictures and the ankylosis of movable parts.

The immediate results obtained in the treatment of otitis externa by this drug appear to

be most satisfactory. The length of time of the cure and the matter of recurrence were not clearly mentioned, however. We all know that these conditions have a great tendency to recur, and it is important to so instruct the patient so that he may realize the importance of submitting himself to continued observation at stated intervals. There is always the possibility of an overlooked allergic dermatitis as well.

We realize the impracticability of the individual physician applying all of these more exact criteria to each new drug which is presented to him by the manufacturer, but it is within his province and it is also his responsibility to carefully inquire into the claims made for and the experimental background of such claims before adopting any new procedure in an extensive way.

REA E. ASHLEY, M.D., San Francisco, Calif.: Dr. Hayes has given us an excellent report on his bacteriologic studies made from external otitis and chronic middle ear infections. He has also presented new drugs for the treatment of these troublesome conditions. Both Dr. Hayes and his co-author are to be congratulated on the way in which they have organized their data and have presented it.

The fact that the number of remedies recommended as efficacious for the treatment of externa otitis and chronic suppurative ear infections is legion indicates the refractiveness of these pathologic conditions.

EXTERNAL OTITIS: In cases of external otitis it is well to remember that the external auditory canal and the tympanic membrane are covered with epithelium and therefore are potentially subject to any pathologic condition of the skin occurring elsewhere in the body.

Gill published an excellent article on this subject in the "Transactions of the Laryngological, Rhinological and Otological Society" in 1942, in which he classified the various causes of external otitis and outlined treatment.

Many ear canals are narrow, moist and warm, and frequently the accumulation of cerumen produces anaerobic conditions in the canals, making them ideal incubators for the growth of all types of bacteria, fungi and molds. The moisture and the narrowness of the canal also produce favorable conditions for epithelial maceration with its attendant lack of resistance to infection.

Allergies often play a part in the initial pathology as do chemicals, hair dyes, rinses, irritating scalp and skin soaps, etc.

Diabetes also should not be overlooked as an etiologic factor.

Unless these causative factors are eliminated or treated, the return of secondary infection is a foregone conclusion.

In the treatment of the pyrogenic types, the local preparations found most useful in our hands have been preliminary use of Burow's solution (aluminum acetate), applied to the canal on wool yarn as suggested by Shea. The wool is saturated with Burow's solution every two hours, and on removal after twenty-four hours the swelling of the canal is usually found to be much reduced. This solution is used even in cases of chronic middle ear suppuration when marked swelling of the canal is present.

The local treatment from this point on depends on the type of infection present. Cresatin packed into the canals has proved most valuable. Sulfathiazole lightly dusted into the canals cures many. Saltzberger powder dusted into the canal is nonirritating and frequently works well. A combination of equal parts of alcohol 70 per cent and glycerine, containing 1,000 units of penicillin per cc. on wool yarn packed into the external canal is also of great value in some cases.

In our experience dyes have been of little value. Ammoniated mercury ointment 5 per cent, Dimozon, coal tar ointment, and Furacin have all been useful, but ointments have on occasion been most irritating, causing exacerbation of the symptoms.

As stated by Dr. Hayes, careful cleansing and careful drying of the canals before applying the medicaments has seemed to hasten the patient's recovery.

CHRONIC OTITIS MEDIA PURULENTA: Time does not permit even a most superficial discussion of this subject. However, the following procedures have been found of value:

1. The instillation of a saturated solution of urea. This digests dead protein material and leaves the infected area clean so that antiseptics may come in better contact with the infection. Offensive odors of the discharge quickly disappear. Urea is only slightly antiseptic, however, and instillation of equal parts of alcohol 70 per cent and glycerin to which 1,000 units of penicillin per cc. have been added has been found very useful. After careful drying of the canal, the insufflation of boric-iodine powder or sulfathiazole powder have also given good results. Care must be taken to allow only the lightest dusting of these latter drugs, however, or caking and interference with drainage will result.

Regardless of the type of infection or the origin of the infection, the following rules are applicable:

1. Control of pain by sedation.
2. Continue treatment for some time after symptoms have subsided to prevent recurrence.
3. Be on the lookout for any general debilities, which may be contributing factors.

Since the drugs presented by Dr. Hayes are entirely new, or at least new to me, I have had no opportunity to use them or to judge their efficacy, but if they are as good as they appear to be from this report, they are, indeed, valuable additions to our long list of preparations used in the treatment of these chronic conditions.

MATTHEW S. ERSNER, M.D., Philadelphia, Pa.: I should like to say that I had the pleasure of reading Drs. Hayes and Hall's paper. I also had the opportunity of discussing the subject with them. Unfortunately, due to lack of time allotted to them, several important factors had to be omitted from the text when presenting the paper.

This work was done under complete control because most of the work was done while Dr. Hayes was in the service. All patients were under complete control and Dr. Hayes was able to follow the treatment with the exception of a few patients who were discharged from the army.

In civilian life, the authors are now carrying on some of the work on the toxic factors. I believe that they will in a short time be able to report the minimal toxicity of this therapy. There is no doubt that they have brought something new to us and I am sure that everyone appreciates the fact that the problem of external otitis is one that has been a boomerang, more or less to us, insofar as the treatment is concerned.

I might also add that in conversation with the authors, I learned that the soldiers did not benefit by the employment of the standard drugs and only began to show improvement and cure when this new therapy was instituted. Therefore, it seems that from the number of cases that have been reported, we have something new on the horizon which we will be able to employ in the near future.

It is interesting to note that the pyocyaneus organism which at one time was not considered of any importance, and was eliminted particularly in the preparation of vaccines, has now become a factor. Heretofore, we always thought that the pyocyaneus did not belong to this type of infection. Today, we know that it is part and parcel of the irritation of the dermatitis as well as the infection.

Dr. Hayes states that he tried 2 per cent acetic acid in a great many cases, as the recent literature recommended this therapy for this type of infection. Many of the patients also had the benefit of the acetic acid therapy as well as the salicylate drugs. But, they did not show any improvement.

With this newer management of otogenic infection, let us hope that the problem of external otitis will be solved.

MERRILL B. HAYES, M.D., Chester, Pa.: I want to thank the discussers for their very gracious remarks.

Some pharmacology has been done on this dye. A lot more is being carried out. We hope that you will avail yourselves of it and try it out. We put it in your hands. We want you to use it and on you will rest the final decision as to how efficacious the substance is.

Thank you very much.

# CACOSMIA AS A SYMPTOM IN CHRONIC SPHENOIDITIS

G. Edward Tremble, M.D.
Montreal, Canada

This symptom has been mentioned as occurring during infections in the sphenoid, but its importance has not been stressed.

Most of the standard textbooks describe cacosmia as the perception of an unpleasant odor by the patient. This varies from a perverted sense of smell (parosmia), which sometimes follows influenza, to a total loss of smell (anosmia), which occurs occasionally in ethmoiditis or when atrophic changes have taken place in the nose.

In speaking of chronic infections of the sphenoid sinus, Tilley[8] mentioned in the last edition of his book that anosmia is sometimes present and "cacosmia, or a sensation of evil odor is a symptom in other cases." Skillern[7] was more definite. He states, "Cacosmia is a frequent symptom, owing to the stagnation and putrefaction of secretion in the immediate proximity of the olfactory fissure. This symptom is more strongly marked during expiration through the nose than on inspiration."

Although most writers mention it as a casual symptom, as in Logan Turner's[9] book, "A disagreeable odor may be observed, but on the other hand, owing to anosmia, this symptom may be in abeyance." Peele and LeJeune[3] in a more recent article on the sphenoid sinus in 75 cases studied, stated that "Cacosmia was characteristic."

The writer's attention was brought to this subject by a number of cases of chronic infection in the sphenoid sinuses in which this was the predominating symptom. In the absence of discharge, pus or crusting in the nose when cacosmia is present a thorough investigation of the sphenoid sinuses is indicated.

In the literature there seems to be a marked divergence of opinion as to the relative frequency as well as the symptoms of sphenoiditis. Fraser[2] quoted Harke as finding 4½ per cent of all cases of sinus infection to be due to the sphenoid. Oppikofer in 165 cases of chronic sinusitis reported the antrum to be involved in about 67 per cent, the frontal in 18 per cent, the ethmoids in 7 per cent, and the sphenoid sinus in only 2 per cent. Fraser himself found definite evidence of infection in the sphenoid in 6 per cent of all cases of sinusitis.

Symptoms. The subjective symptoms of empyema of the sphenoid are also extremely variable and inconstant. No doubt this is the reason why many cases of mild chronic inflammation in this region are overlooked. It is generally agreed that the most frequent symptoms are headache, disturbances due to postnasal discharge, cacosmia or interference with the sense of smell and vertigo. Perhaps the most common complaint is pain in and behind the eye, over the parietal region and back to the occiput. This single symptom is mentioned because it is typical, but also because it may be entirely absent in well-marked cases of sphenoidal sinusitis.

Postnasal discharge and the results in the pharynx and nasopharynx are the most frequent objective symptoms. Discharge along the edge of the vomer, in the sphenoidal recess or in the fossa of Rosenmüller are strongly suggestive of

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

sphenoiditis. Chronic nasopharyngitis, enlarged red lymph follicles, inflamed and thickened lateral bands are evidence of constant or intermittent drainage posteriorly.

In this connection, Yates[10] using indigo-carmine and lamp-black attempted to estimate the ciliary action and the flow of mucus and pus in normal and infected sinuses. His method in brief was to inject the various sinuses with a small quantity of the dye in solution and to map out the pathways down to the entrance of the esophagus.

It is necessary to describe the track from the antrum in order to understand those from the posterior group of sinuses. "On injecting 10 cc. of this fluid (0.4 per cent indigo-carmine solution in saline) into the antrum, if the sinus was normal the indigo-carmine appeared in the nasopharynx in about two minutes.

"From the ostium of the sinus it appeared to follow a definite path, as follows:

"It passed along the upper part of the middle meatus, and reaching the posterior part of this, turned outward and then downwards, and came to form a definite track in front of the Eustachian tube. From this situation, it passed downwards between the attachment of the soft palate and the posterior wall of the pharynx, and, continuing, extended as a definite stream which lay just behind the posterior pillar of the fauces; from this part of the stream some of the fluid was directed in all cases anterior to the posterior pillar of the fauces, and found its way into the recess between the tonsil and the posterior pillar.

"At the level of the dorsum of the tongue the main stream divided into a larger and a smaller portion.

"The smaller portion was conveyed across the dorsum of the tongue in the position of the lingual tonsil, and from thence in some cases it reached the glosso-epiglottidean pouch, and in other cases was swallowed directly.

"The larger portion continued outwards along the posterior margin of the posterior pillar of the fauces, and thus reached the sinus pyriformis and continued down this to the posterior aspect of the arytenoids, finding its way into the interarytenoid space.

"If a small quantity of the 0.4 per cent indigo-carmine solution was placed in the posterior ethmoidal sinus by means of a special curved cannula, the fluid passed in a direction as follows:

"It was first seen by examination of the nasopharynx in the superior meatus, and passing backwards it escaped from this into the nasopharynx and was conveyed outwards for a short distance until it occupied a position at the upper part of the nasopharynx, immediately in front of the uppermost portion of the Eustachian tube. Here it divided into two streams, a larger which extended downwards and joined the position of the stream from the antrum, and a smaller which passed over the Eustachian tube and downwards through the fossa of Rosenmüller, and curving forwards joined the antral stream at the level of the lower border of the soft palate. The further course of these streams was the same as that described in connection with the stream from the antrum. If the fluid was injected into the sphenoidal sinus it passed from the opening of the sinus in a downward direction until it reached the highest point of the choanae. Here it turned backwards and spreading out like a fan upon the vault of the nasopharynx was conveyed in a series of streams, the outer of which made its way into the lower part of the fossa of Rosenmüller.

"The inner streams passed in an outward direction until they joined together in the position of the antral stream, about half an inch below the lower margin of the soft palate, and the further

course of the fluid was the same as that already described in connection with the antrum.

"The ciliary action in cases of sinusitis is somewhat different.

"When mucus or pus is present in any of the sinuses, the rapidity of the flow of the indigo-carmine solution may be greatly modified. In cases where the contents of the sinus are mucus only, the rate of flow is little altered. In those where thick gelatinous pus is present in the sinus, there is some slowing of the stream, but this is slight. . . .

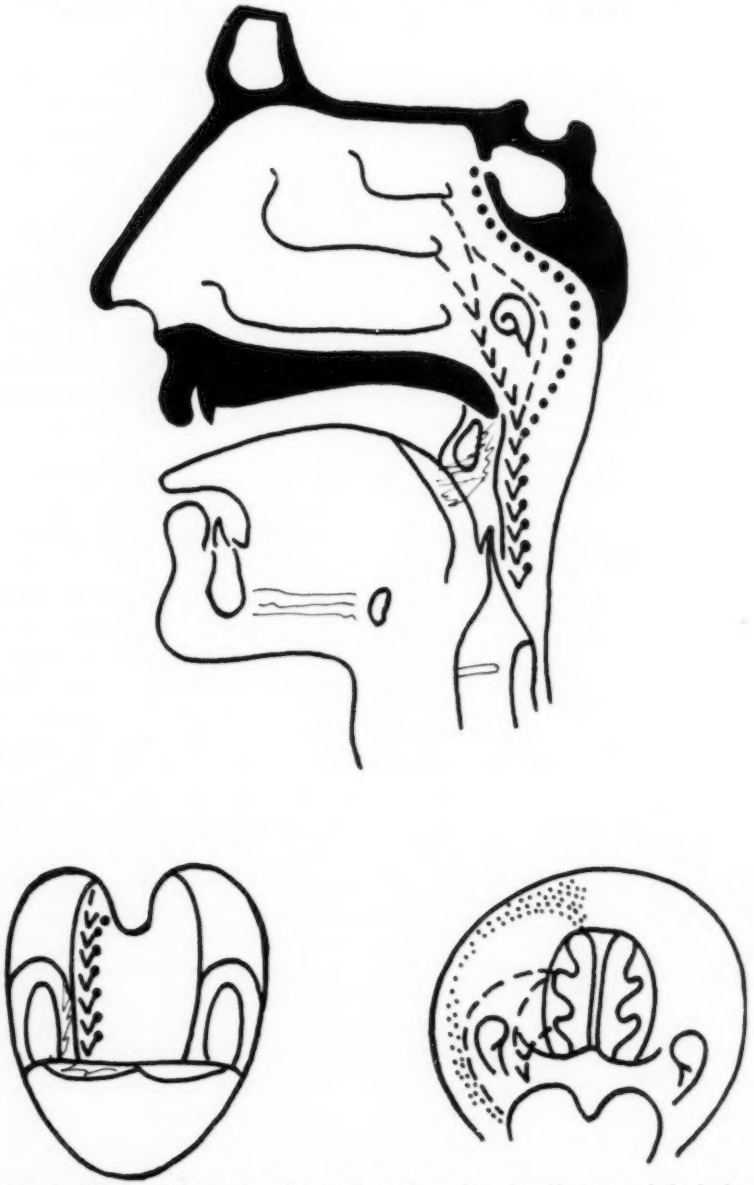


FIG. 1—The dots indicate the ciliary pathway from the sphenoid sinus and the broken lines the tracks from the other sinuses (after Yates).

In sinusitis of the atrophic type, "the fluid is conveyed away very sluggishly and there may be complete stasis of the fluid within the sinus, while the part of the track nearest to the sinus may appear to be deeply stained, the stain remaining in position for some hours unless washed away by douching."

From the foregoing statements, it would seem to be a fair assumption that the movement of the colored fluid is due to the action of the ciliated epithelium and that stasis of the fluid is due to a "paralysis" or partial destruction of the cilia. However, in cases of severe sinusitis where the cavities are bathed in pus, normal cilia have been found to beat vigorously.[6] Whether or not over long periods of time the cilia are permanently incapacitated by the toxins of the infecting organisms remains to be proven.

It should be mentioned that infection in any of the sinuses might cause cacosmia. However, the antrum and sphenoid are more often responsible as they are larger cells and contain more air. Also the ostium, in each case near the roof, although poorly placed for drainage favors retention and putrefaction of infected material. The highly placed ostia of the antrum and the sphenoid aid in the exchange of air from the nose to the sinuses and from the sinuses to the nose. This air exchange occurs more freely from the antrum and sphenoid than from the frontal sinus or ethmoid cells. Anatomically we have retention and stagnation with intermittent exchange of air from the sinus depending on the amount of swelling or closure of the ostium of the particular sinus.

The common organisms found in the nose during an acute or chronic sinusitis are streptococcus, staphylococcus, pneumococcus, and B. influenza. All these aerobic bacteria form pus but it has no odor. However, anerobic streptococci and bacteriodes which are also anerobes produce foul smelling pus, particularly when associated with aerobes. These organisms and B. coli (aerobe) are frequently found in the nose. In cases of patients complaining of a bad odor, anaerobic cultures are absolutely mandatory if the correct explanation is to be found. It is not unimportant in this respect to remember also that a significant number of ordinary hemolytic streptococci will not grow in primary culture except under anerobic conditions.

The frequent association of anaerobes with aerobic bacteria must often lead to erroneous interpretations of the significance of the aerobes unless the presence of anaerobes has been definitely ruled out. A trivial aerobic growth may often mask completely a much heavier anaerobic flora.

These anaerobes because they are deprived of the use of normal oxidation derive the requisite energy for growth from the anerobic metabolism of a very large amount of food stuff, usually in the form of protein—containing exudates and pus. The sulfur-containing amino-acids in these latter are broken down into mercaptans which explain the foul smell. B. coli on the other hand will frequently produce indole and skatole and in that way give rise to a bad odor. However, certain strains of B. coli do not bring about this chemical change and may grow freely in the sinuses without the sensation of cacosmia.

It should be mentioned that cacosmia from a sinus infection is always intermittent. If a patient is conscious of a constant odor, the condition is definitely a neurologic one and he should be referred to a neurologist. Actual smell perceptions constantly applied soon result in fatigue and the patient would not experience them for any length of time.

In an article on the microscopic findings of the sphenoid ostium Proetz[4] brought out a number of valuable points. In part, he said, "The sphenoid mucosa

is characteristically thin, its tunica composed chiefly of parallel elastic elements laid closely together and bound firmly to the underlying bone. It contains very few glands. The nasal mucosa on the other hand, is many times thicker and is permeated with vascular channels and glandular elements. The thin mucous membrane of the sinus and thick nasal mucosa follow up both sides of the thin anterior wall of the sphenoid and keep their respective identity until they reach the edge of the ostium above the underlying sharp edge of the bone. The ciliated columnar epithelium is the only layer which passes uninterruptedly through the ostium. The submucosa of the sinus terminates abruptly a little short of the margin of the ostium. By this arrangement the ostium is formed by nasal mucosa with two or three small folds at the opening made up of loose connective tissue with numerous mucous glands and blood spaces."

A practical point which may be deduced from these findings suggests itself according to Proetz, "Since the ostium is composed chiefly of nasal, and not sinus elements, and is supplied chiefly by nasal arteries, application of constrictors, such as ephedrine, to the neighborhood of the ostium should suffice to open it." This means that in an acute infection in the nose with inflammation and swelling of the nasal mucosa surrounding the ostium, the opening could be intermittently narrowed or even closed.

In reference to this point, the inspired and expired air through the nose should be considered. A satisfactory method of studying air currents is to use a human adult head split sagitally just to one side of the septum. This was described many years ago by Franke[1] and more recently by other writers.[5,11] A piece of window glass replaces the septum and is sealed around the edges. A rubber tube about the size of the trachea is fixed in the postnasal space, and a small cardboard box is fitted tightly into the anterior naris or connected by a short piece of narrow tubing, about the caliber of the nostril. This is a slight modification of the previous methods. The glass surface and rubber connections must all be hermetically sealed. After warming the glass over a spirit lamp, the mouth is filled with cigarette smoke and blown into the cardboard box or through the specimen. Now the smoke is inhaled through the tube in the nasopharynx imitating the rate at which air normally enters the nose in quiet breathing, i.e., sixteen to twenty times a minute.

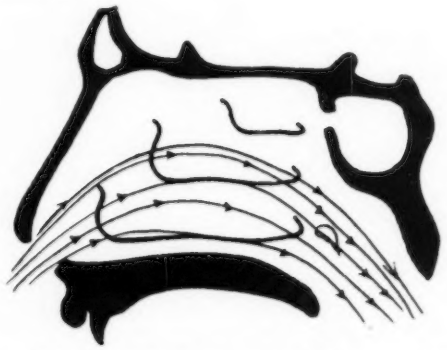


FIG. 2. The inspired air curves high up into the nose and down into the nasopharynx but does not enter the meatuses.

The currents of smoke form a definite pattern which is difficult to depict on paper because it is impossible to represent the fact that a portion of the stream which has, say a square millimeter section at the narrow anterior naris, fans out to ten times that section as it enters the large cavity of the nose; moreover the currents which run close to the glass take a different course than those influenced by friction against the complicated outer wall. Certain points, however, come out clearly. The main inspiratory stream curves in a wide arc high up in the nose and down to the nasopharynx. Its direction is controlled by the relatively small anterior naris,

the vault of the nose and the larger posterior choana. As the direction of the main current is upward, very little passes through the lower part of the nose. On the other hand, even on deep inspiration, only small amounts of smoke appear to reach the olfactory area. The main stream passes over the lateral wall reaching about the level of the attachment of the middle turbinate but does not enter the meatuses. In this way, a large porportion of the stream hugs, so to speak, the medial surface of the inferior and the lower border of the middle turbinates. These, of course, are precisely the parts which are supplied with cavernous tissue, which warms and moistens the air and prepares it for the unspecialized parts of the respiratory tract below.

Expiration is very similar to inspiration. Here the expired air passes high up in the nose before emerging through the smaller opening at the nostril. Its direction is influenced by the lower border of the sphenoid which seems pushed into the nasal cavity, the flange-like posterior ends of the turbinates and the narrow exit at the anterior naris. On exhaling smoke through the sectioned head, the stream fans out into the various fossae. Owing to the small anterior opening the expired air cannot all pass directly through the nostril so that a large circular eddy forms which enters the meatuses. Thick currents also pass through the inferior, middle and superior meatuses and a smaller one passes up into the olfactory cleft.

This latter stream is a much more prominent feature than that which reaches the olfactory fissure on inspiration. It impinges directly upon the olfactory nerve both on the lateral wall above the superior turbinate and on the corresponding area of the septum. This fact explains why a very slight cold will temporarily cause anosmia, but it requires a severe inflammation and

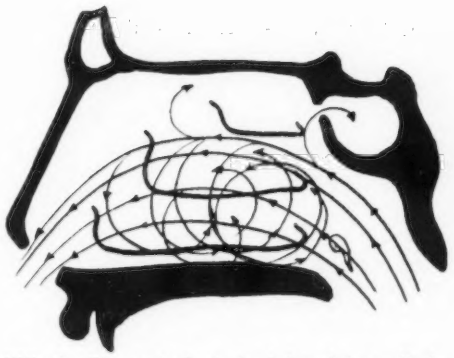


FIG. 3. The expired air is deflected into all the meatuses and also into the olfactory cleft.

swelling to block the back of the nose and to impair the appreciation of flavors, which are also carried through the olfactory nerve. It is interesting to note in a sectioned head that sometimes a thin coil of smoke enters the sphenoidal sinus. When this occurs, it cannot be stirred by any amount of violent exhalation, but is easily diluted by rapid inhalation.

If left alone this smoke tends gradually to seep out through the ostium until it all disappears. In the presence of pus in a sphenoid sinus which has remained for some time, it seems reasonable that a patient experiences a bad odor. The inspired air which is imprisoned in the sphenoid comes away gradually, helped by the act of swallowing, and the suction of the soft palate against the posterior pharyngeal wall. From time to time this air passes the olfactory area, hence the intermittent cacosmia.

By referring to figure 1, we see how postnasal discharge, particularly from the posterior ethmoids and sphenoid, lies directly in the path of expired air as it fans out into each meatus and above the superior turbinate.

With this in mind and acting on the suggestion of Proetz that constricting agents in the neighborhood of the ostium should open it, the writer has used this procedure as a routine in all cases of

suspected sphenoiditis. The ostium is cocainized well, ephedrine applied, and the patient then asked to swallow and exhale a number of times. It is sometimes more effective if the patient holds the nostrils closed on swallowing. On questioning as to whether or not a bad odor is experienced, it is surprising to find how often it is present, provided, of course, that the sense of smell has not been entirely destroyed. If cacosmia is present and the antrum has been ruled out, it seems reasonable to presume that infection probably exists in the posterior ethmoid cells or the sphenoidal sinus. In each of the cases mentioned in which cocosmia was the predominant symptom, the usual nasal examination was negative. This included the nasopharyngoscope and x-ray.

In summarizing, cocosmia as a symptom is very important. Any patient who complains of this should have the sphenoid sinuses irrigated and cultured. It is only by routine examination of the sphenoids when cacosmia alone is present that many cases of chronic low-grade sphenoiditis are found and treated successfully.

## REFERENCES


1. Franke, G.: Arch. f. Laryng. u. Rhin., No. 2, 1:230-249, 1893.
2. Fraser, J. S.: The frequency of sphenoidal sinus operations, J. Laryng. & Otol., 39:393-396, (July) 1924.
3. Peele, J. C. and LeJeune, F. E.: A clinical survey of the sphenoid sinus with special reference to the direct method of lavage and injection of opaque medium, Laryngoscope, 52:423-452 (June) 1942.
4. Proetz, A. W.: The minute anatomy of the sphenoid ostium, Tr. Am. Laryng. A., 55:125-127, 1933.
5. Proetz, A. W.: Some intimate studies of nasal function: their bearing on diagnosis and treatment, Ann. Otol., Rhin. and Laryng., 41:125-140 (March) 1932.
6. Proetz, A. W.: Studies of nasal cilia in the living mammal, Ann. Otol., Rhin. and Laryng., 42:778-788 (Sept.) 1933.
7. Skillern, R. H.: The catarrhal and suppurative diseases of the accessory sinuses of the nose, J. B. Lippincott & Co., Philadelphia, p. 384, 1923.
8. Tilley, H.: Diseases of the nose and throat, H. K. Lewis & Co., Ltd., London, 1919.
9. Turner, A. Logan: Diseases of the nose, throat and ear, John Wright & Sons, Ltd., Bristol, 1930.
10. Yates, A. Lowndes: Methods of estimating the activity of the ciliary epithelium within the sinuses, J. Laryng. & Otol., 39:554-560 (Oct.) 1924.
11. Waggett, E. B.: Diseases of the nose, Oxford Med. Pubs., London, p. 34, 1907.


## DISCUSSION

FRANK R. SPENCER, M.D., Boulder, Colo.: Disease in one or both sphenoidal sinuses is more frequently overlooked than in any of the other sinuses, because the sphenoidal sinuses are less accessible for examination and treatment than any of the others. Posterior ethmoidal cells may be just as inaccessible. Those of you who have looked over Dr. Mosher's specimens, as I had an opportunity to do when I took his course, will appreciate this because some posterior ethmoidal cells can be extremely inaccessible.

So far as my own personal experience goes, I have found cacosmia from one or more infected posterior ethmoidal cells more frequently than from the sphenoidal sinuses. The difficulty of draining these cells has been greater than draining the sphenoidal sinus or sinuses.

The late Ross Hal Skillern had in his textbooks elaborate and carefully planned diagrams on the skull, showing the pain area for each sinus. These diagrams are very helpful, but many patients with cacosmia from sinus discharge do not have headache or, in fact, any pain. The cacosmia may be the only symptom complained about.

I agree with the author that anaerobic organisms are more likely to produce cacosmia than aerobic. Cultures from the sinuses are easily contaminated. Some germs grow more readily than others on artificial media. The real offenders may not grow at all. He is quite right in saying aerobic and anaerobic cultures should be made.

The varnished or glazed appearance of the posterior pharyngeal wall is a valuable objective symptom in patients with chronic discharge from the posterior group of sinuses. It is not always present. When present it should lead to a thorough study of the posterior group of sinuses.

The author's diagrams of air currents in the nasal fossae and nasopharynx show why cacosmia can be such a prominent symptom. However, the application of adrenalin, ephedrin and particularly cocaine make it more difficult in my experience to elicit the cacosmia. Dr. Tremble's irrigation of the sphenoidal sinuses as a diagnostic and therapeutic measure is certainly justified. After a diagnosis of sphenoidal sinusitis has been made, enlarging the natural ostium will usually relieve the cacosmia. A small opening will likely close. Too large an opening will often chill the nasal ganglion, during cold winter weather, and produce sphenopalatine ganglion neuralgia.

I have tried in recent years to strike a happy medium, as I know the rest of you have, by having an opening not so small it will close nor one so large the patient will have neuralgia.

Each winter I try to find time to study the anatomy of the head and neck. Anatomy is the easiest thing I know of to forget and I try to review it, but I am fearful I don't review the physiology as often as I should. Dr. Tremble emphasizes some very important points in the physiology of air currents and the physiology of respiration. He tells me that Dr. Proetz urged him to do this. I am glad it has been brought out. Dr. Proetz has made all of us more conscious of nasal physiology, and we will be better diagnosticians, better surgeons, and better rhinologists because of this knowledge.

I have enjoyed the privilege of discussing this paper. I am glad Dr. Tremble has called this important subject to our attention.

FRED W. DIXON, M.D., Cleveland, Ohio: The term cacosmia is used by rhinologists and neurologists rather loosely. For clarity of discussion, disagreeable odors may be central or parosmias and peripheral or cacosmias. To the patient these are the same. To the diagnostician the origin is entirely different. Irritation of the olfactory fibers in the uncinate gyrus, or irritation of the mesial border of the temporal lobe, or hemorrhage into the floor of the anterior cranial fossa may produce parosmia or an illusion of a bad odor.

When the origin of the offending odor is peripheral it may be respiratory, toxic, inflammatory or of sinus origin. Cacosmia of sinus origin is common. In my experience it is the maxillary sinus which is the greatest offender. This is due to the fact that it is most frequently the reservoir of putrefaction, and that the infection is often of foul smelling dental origin. I do not believe you can diagnose any particular sinus by odor. Dr. Tremble does not so state. He brings to our attention a rather forgotten fact that it can be a diagnostic finding and that when corroborated by other findings sphenoidal infection is to be considered and irrigation performed.

Irrigation of the sphenoid can be done in 70 per cent of patients. The failures will be where the ethmo-septal angle is less than 30 degrees. That is the angle made by the turbinates and the septum. In measuring several hundred skulls I found the sphenoid ostium to be a little over 8 millimeters from the roof and 5 millimeters from the septum with two exceptions. If the ethmo-sphenoidal recess is large, the ostium will be more laterally placed, or if the posterior ethmoid cell has driven in above the sphenoid, the ostium will be placed up in the corner approximately 2.4 mm. from the roof and 2.4 mm. from the septum. With these conditions kept in mind, I believe that the sphenoid in the great majority of cases can be irrigated.

I wish to thank Dr Tremble for the privilege of reviewing his paper beforehand and discussing it here today.

ROBERT F. RIDPATH, M.D., Philadelphia, Pa.: One feels that a more distinctive and comprehensive definition of cacosmia might be, "Cacosmia—the perception of an unpleasant fetid odor by the patient. This odor may also be perceptible to others in close proximity to the patient, the odor coming either from the nose or throat or both."

Cacosmia may be present whenever we have a stagnation or putrefaction of secretion within the nasal cavities or pharynx. It is a distinct symptom as Dr. Tremble has pointed out, of sphenoidal involvement. This may be of subacute or chronic variety. Dr. Tremble has quoted several authorities as to the frequency of sphenoidal infections. My personal experience tells me these are all too low. It is a regrettable fact that so many rhinologists are, shall we say, not as careful in their examination of the sphenoidal area as they might be. The examination for a sphenoidal infection requires considerable time, and unless this is freely given, many cases will be overlooked.

The essayist is quite correct in saying that the symptoms of empyema of the sphenoid are extremely variable and inconstant. We find that headache associated with this condition is mostly occipital, radiating in a fan shape to the vertex. Perhaps the most frequent symptom of sphenoidal empyema is vertigo. This vertigo is nearly always present in one of its various types and can become extremely pronounced. The pain back of the eye, seemingly pushing the eye forward is also a frequent symptom

as well as forgetfulness, lack of initiative, and malaise.

I was very glad to hear mentioned enlarged lymph follicles and nasopharyngitis, as these are always noticed when irritation from post-nasal drainage is present. Sphenoidal discharge is more likely to be seen in the olfactory fissure, this due to capillary drainage, than along the inferior borders of the turbinate.

Essentially, in sinusitis of the atrophic type, the discharge is likely to lie for some time on the thinned and pathologically changed mucous membrane, with resultant loss of ciliary action. This necessarily gives a fetid odor.

We feel that among the various bacteria associated with upper respiratory tract infections one should not overlook Friedlander's bacilli. This in itself gives a distinctive foul odor.

We should like to compliment Dr. Tremble for bringing forward the association and significance of the anaerobic and aerobic bacteria. This is of extreme importance, and, as stated, cultures with this in view should always be made.

Although the olfactory filaments and cells are soon tired or fatigued, I cannot perceive that cacosmia is intermittent. Perception of the offending odor may be lessened or absent to the patient, but it is still present and perceptible to others.

We thoroughly agree that any patient who is cognizant of a fetid or cacosmic odor should have a most careful examination not only of the sphenoid, but of the whole upper and lower respiratory tract. We must remember the little cul-de-sac or blind spot in the epipharyngeal space, where we are exceedingly prone to harbor tenaceous and mucilaginous drainage, with resultant odor. We must also remember that lower respiratory involvement as malignancies, actinomycosis, cancer, cavities, gangrene, etc., all may and frequently do have distinctive disagreeable odors of a cacosmic or similar type, and these odors will be carried by exhalation to the upper respiratory tract and unless considered, might result in wrong diagnosing of our case.

G. EDWARD TREMBLE, M.D., Montreal, Canada: In summing up there is very little that I can add to the discussion. I was very pleased in hearing Dr. Spencer's remark on his interest in anatomy following his course with Dr. Mosher. As a matter of fact, my experience was very similar and has been a great stimulation to me ever since.

Dr. Dixon also mentioned that the antrum is more often involved. Without doubt this is quite true as infections in this sinus occur much more frequently but the antrum is so easy to explore that we can rule it out very quickly.

Dr. Ridpath mentioned that not enough men are familiar with the sphenoid and that we should explore this sinus more often. It is surprising on questioning rhinologists in various parts of the country to find how few investigate the sphenoid. My one thought to leave with you is that although we should not unduly center on the sphenoid, we should be able to recognize and treat these cases whenever they occur.

# THE DISPLACEMENT OF PENICILLIN SOLUTION FROM THE NASAL CHAMBERS INTO THE PARANASAL SINUSES BY MEANS OF SELF-INDUCED INTRANASAL NEGATIVE PRESSURE

CHARLES HYMES, M.D.
MINNEAPOLIS, MINN.

PENICILLIN has been reported to be effective in treating acute and subacute paranasal sinusitis when given in solution by the intramuscular or intravenous route and by the inhalation of penicillin aerosol. A method whereby the patient is able to instill solutions of penicillin directly into the paranasal sinuses by means of self-created intranasal negative pressure is reported in this paper. In the treatment of sinusitis, Barach and his associates report good results from the use of a solution of penicillin in aerosol introduced into the sinuses by negative pressure created by suction applied to the nasal chambers. A vapor with a high concentration of penicillin is employed. The patient is instructed to inhale this vapor five or six times after which suction is applied to the anterior nares while the patient sounds the letter K. The penicillin vapor is then forced into the nasal chambers under positive pressure. This whole process is repeated a number of times during the course of a single treatment. Barach stated that the penicillin vapor penetrates into the sinus cavities.

When one considers the physical principles involved in this procedure, one recognizes that the negative pressure within the nasal chambers and sinuses is instantaneously lost the moment positive pressure is exerted within the nose. At that moment, the soft palate falls away from the K-position and the vacuum ceases to exist. It seems probable that little if any penicillin vapor can reach the sinus cavities. On the other hand, penicillin may reach the sinuses by way of the blood stream after being absorbed from the lungs, for as pointed out above, the method involves the breathing of highly concentrated penicillin vapor before negative pressure is applied. This method of treatment requires frequent visits to the doctor or the purchase of costly equipment if the patient is to administer the treatment to himself.

The Proetz displacement method serves adequately for introducing solutions into the sinuses. Effective treatment by this method, however, requires the patient to visit the rhinologist many times a day in order that a sufficient quantity of penicillin solution may be kept in contact with the diseased sinus membranes long enough to prove effective. Most patients can afford neither the cost nor the time for such frequent treatments. The same objection holds for the intramuscular or intravenous administration of penicillin as this type of treatment requires hospitalization.

Negative pressure may be induced in the nasal chambers and paranasal sinuses by applying suction at the external nares with the soft palate in the K-position. It occurred to the writer that similar intranasal negative pressure might be produced by closing the nostrils with the thumb and index finger and making inspiratory efforts with the mouth closed.

Presented as *A New Technic* at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

Department of Ophthalmology and Otolaryngology, University of Minnesota.

In order to demonstrate the degree of negative pressure which may be produced intranasally in this manner, the writer attached one arm of a mercury manometer U-tube to his nose while the other arm was attached to a rotating tambour. Both nostrils were then closed around the tube and repeated inspiratory efforts were made in the manner described above.

Figure 1 indicates that upon each inspiratory effort, a negative pressure of 50 to 80 mm. of mercury was induced intranasally, with an average sustained negative pressure of 60 mm. of mercury.

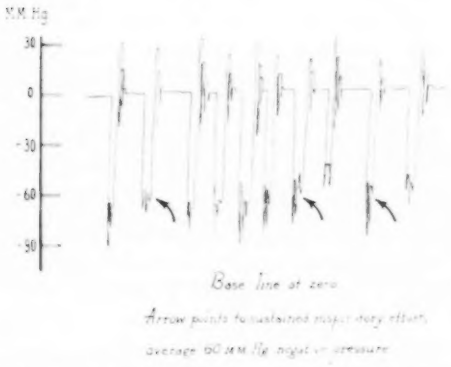



FIG. 1 — Reproduction of tambour tracing of negative intranasal pressure. Oscillations below arrow points and above zero line are due to momentum of mercury column. Average negative pressure obtained, 60 mm. of mercury.

Figure 2 is an illustration of the lateral wall of the right nasal chamber. If a horizontal line is drawn from the lowermost point of the sphenoid projection to the inner wall of the nose anteriorly, it can be seen that all the sinus openings, that is, the middle and superior meati as well as the spheno-ethmoidal recess lie above this line. With the head extended backward, well over the edge of a table or bed, the nose pointing upward, this area forms a concavity into which as much as 8 cc. of fluid may be placed without spilling into the nasopharynx. While placing the solution in the nasal cavity, the patient must breathe through the mouth. With the soft palate in the contracted or K-position, considerably more than 8 cc. of fluid may be instilled into the nasal chamber.

FIG. 2. — A view of the lateral nasal wall taken from Pratt's *Intranasal Surgery*. All of sinus ostii lie above a horizontal line drawn from the lowermost point of projection of sphenoid bone to the nose anteriorly. The opening of the eustachian tube is well below this line.

After the fluid is instilled into the nose, the patient is instructed to close his nostrils with the thumb and index finger and to make inspiratory efforts through his nose against the closed anterior nares. About five or six such inspiratory efforts are sufficient to cause the solution in the nasal chamber to be displaced into the sinuses. If 4 to 6 cc. of fluid are placed into one or both nasal chambers, practically all of the fluid will be displaced into the paranasal sinuses. The process may be repeated and more of the solution displaced into the sinuses if it is desired. It should be pointed out also that with the head in the extended supine position, no fluid will be displaced into the eustachian tube or middle ear as the orifice of the eustachian tube will then be well above the level assumed by the fluid in the nasal chamber.

It is possible to displace the penicillin solution into the sinuses on one side or both as the occasion requires, with the head in the extended supine position. However, if the chin is turned toward the shoulder, the filling of the anterior group of sinuses on the dependent side of the head including the maxillary,

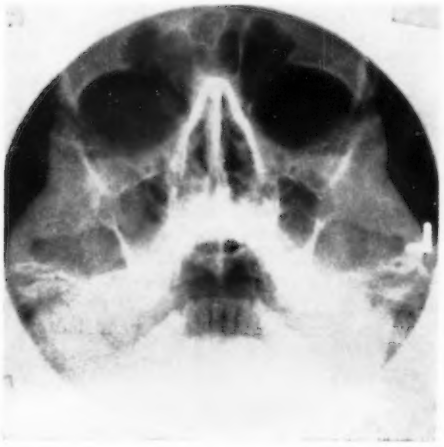


FIG. 3a. — Case 1 — Before instillation — polyp or mucocele in right maxillary sinus with thickening of membrane.

FIG. 4a — Case 2 — Before instillation—sinuses are clear.

FIG. 3b — Case 1 — After instillation—arrow points to fluid levels in left frontal, left maxillary and sphenoid sinuses.

FIG. 4b — Case 2 — After instillation—arrow points to definite fluid level in right maxillary, and less definite level in left maxillary sinus. A fluid level in the sphenoid is also apparent.

frontal, and anterior ethmoid air cells is facilitated.

That sufficient negative pressure can be induced in the nasal chambers to cause displacement of solutions into the sinuses by this method, is evidenced by the following series of x-ray films, taken in each instance before and after the instillation of saline solution into the nasal chambers. These films were read and reported by Dr. L. G. Rigler of the Department of Radiology of the University of Minnesota.

Case 1. A postero-anterior view of the paranasal sinuses in the upright position reveals a large polyp or mucocele and marked thickening of the mucous membrane throughout the right maxillary sinus. The remaining sinuses are fairly clear. Because of the presence of this polyp or mucocele, unilateral filling was tried. The film made after the introduction of fluid in the nasal orifice indicates a definite fluid level in the left maxillary sinus suggesting that it con-

tains several cubic centimeters at least. There is a fluid level in the left frontal sinus and a distinct fluid level in both sphenoids. No fluid can be made out in the right maxillary sinus.

Case 2. The roentgenogram made before instillation of saline solution shows all sinuses to be well developed and clear. A roentgenogram made after introduction of fluid shows a definite fluid level in the base of the right maxillary sinus and a less definite level in the left maxillary sinus. A fluid level in the sphenoid is also clearly apparent.

Case 3. A preliminary roentgenogram shows all sinuses well defined and clear. A similar view made after introduction of fluid into the nasal orifices followed by induction of negative pressure in the manner described shows a fluid level in the base of both maxillary sinuses, probably a little more being present on the right than on the left. There is also evidence of a small amount of fluid in the sphenoid sinuses.

Case 4. A roentgenogram made before the introduction of fluid shows all the sinuses to be well developed and clear. After the introduction of fluid the roentgenogram shows that fluid levels can be made out at the base of both maxillary sinuses. A level indicating a

FIG. 5a — Case 3 — Before instillation of fluid, all sinuses clear.

FIG. 6a — Case 4 — Before instillation, all sinuses clear.

FIG. 5b — Case 3 — After instillation, very definite fluid levels can be made out in both maxillary sinuses. There is also evidence of a small amount of fluid in sphenoids.

FIG. 6b — Case 4 — After instillation, fluid levels are to be seen in both maxillary sinuses and a small amount of fluid in sphenoid.

small quantity of fluid can be observed in the sphenoid sinuses.

Case 5. A preliminary roentgenogram shows all the sinuses fairly well delineated, the frontals showing marked lack of development. A film of the sinuses made after instillation of fluid on the right side shows a definite fluid level at the base of the right maxillary sinus, the measurements of which would indicate that approximately 4 and 6 cc. of fluid had entered the sinus.

FIG. 7a.—Case 5.—Before instillation all sinuses are clear. There is a lack of development of the frontals.

FIG. 7b.—Case 5.—After instillation, definite fluid level in right maxillary sinus indicating about 4 to 5 cc. of fluid. Sphenoid is not shown.

It should be noted that ethmoid filling is not reported in any case. The roentgenograms were taken only in the antero-posterior position and because of the superimposition of bone and tissues and because contrast media were not employed, it is difficult to draw any conclusions regarding the ethmoids. However, there can be little question that the ethmoid sinuses were filled in the same manner as the others.

In the treatment of patients with acute and subacute sinusitis, 2,000 units of penicillin per cc. of saline was used. The patient was instructed to instill 4 to 6 cc. of the solution into each nasal chamber, make six inspiratory efforts with the nostrils clamped and to repeat the treatment three to four times daily. It must be demonstrated very definitely to the patient how the treatment is to be carried out. Occasionally a solution of sodium penicillin is found to be irritating and causes a burning sensation in the nose and eyes. This is no doubt due to an impure product, as the purity of sodium penicillin has not yet been standardized. Calcium penicillin, however, even in much higher concentrations has not been found to cause any irritation, probably because of its greater purity.

The number of patients treated in this manner up to the present time has been too small to allow for exact conclusions. However, the results in most of these cases have appeared to be so favorable that I have high hopes that it will prove to be a worthy addition to our therapeutic armamentarium.

The results of the treatment of thirty cases may be summarized briefly as follows: Three patients with recurrent suppuration of the middle ear associated with acute maxillary and ethmoidal sinusitis appeared to recover with unusual rapidity. The suppuration from the ear and sinuses in the first patient ceased within forty-eight hours. In the second patient, the suppuration from the middle ear ceased in seventy-two hours, while the third case was well within five days.

The remainder of the patients who had acute or subacute suppurative sinusitis appeared to be well in two to four days after the beginning of this treatment with the exception of two. One patient in whom an antrum window had been made before I began using the instillation of penicillin solution as I have described, developed an acute recurrence of sinusitis in the antrum on which the surgery had been performed. A large patent antrum window permitted the instillation of penicillin solution directly into the maxillary sinus without employing negative pressure. The patient instilled the solution into the sinus while lying with the head tilted to the side. Ten days were required before recovery seemed manifest.

The second patient with acute maxillary sinusitis in whom this method of treatment did not effect a rapid cure was one who failed to carry out instructions properly. This patient required two sinus irrigations with instillation of penicillin solution through the trocar after the irrigation to effect a cure.

It should be pointed out that this method is not to be employed during the acute phase of a coryza, but only after symptoms suggesting a complicating sinusitis have become manifest. Nature by this time has had an opportunity to build up its defenses and there is no danger of carrying the infection into sinuses that are not involved. Since patients as a rule do not present themselves for treatment during the early phase of a coryza, the possibility that one would be tempted to treat a case too soon would not be likely to occur very often. Most patients have had sufficient experience with the common cold to know that for the most part it is self-limited and usually subsides quickly. It is only when the common cold becomes complicated that patients present themselves for treatment and it is precisely at this time that the treatment described can be most effective.

In summary, it may be said that evidence has been presented to demonstrate that negative intranasal and intrasinus pressure may be self-induced by the patient by means of making inspiratory efforts through the nose against the closed anterior nares. This negative pressure is sufficient to permit intranasal solutions to be displaced into the sinuses. This method is effective in treating locally the complications of the common cold with penicillin solution. It is inexpensive and easily carried out by the patient.

## REFERENCES

1. Barach, A. L. et al: Penicillin aerosol and negative pressure in the treatment of sinusitis, Am. J. Med., 1:268 (Sept.) 1946.
2. Proetz, A. W.: The displacement method of sinus diagnosis and treatment, Annals, St. Louis, 1931.

# TESTS FOR HETEROPHORIA

## Reliability of Tests, Comparisons Between Tests, and Effect of Changing Testing Conditions

RICHARD G. SCOBEE, CAPT., M.C., AUS

*and*

EARL L. GREEN, CAPT., A.C., AUS

BY INVITATION

RANDOLPH FIELD, TEXAS

MUSCLE balance is a subject in which the student of ocular functions almost invariably takes an early interest. The discovery of several points soon lessens this interest. Among these is the clinical observation that relatively large amounts of heterophoria may be present in an individual and yet that individual may be perfectly comfortable. Another of these is the statement found in the literature on ocular muscle balance that heterophoria varies not only from day to day but also from moment to moment in the same individual.[2,7] These two observations lead many students to the conclusion that it matters little what test of heterophoria is used or how carefully it is performed since the results will probably be inaccurate and of dubious significance.

Nevertheless, in any thorough survey of ocular functions, heterophoria cannot be neglected. Heterophoria is a deviation of the eyes from the position of fixation when fusion is prevented.[7] Measurements of heterophoria therefore reveal how much of a motor task fusion has to perform in bringing images upon corresponding retinal points. Certainly the individual with little or no heterophoria, all other things being equal, will secure binocular single vision with much less effort than the individual with marked heterophoria. The average clinician is not particularly concerned with whether his patient has 4 prism diopters of exophoria or 6 prism diopters of exophoria, unless that exophoria is indicative of symptom-producing strain. Neither is he particularly concerned when a patient tested on one day shows 4 prism diopters of exophoria and when tested again, on a subsequent visit, shows 6 prism diopters of exophoria.

When it becomes necessary, however, to select large numbers of men in accordance with certain arbitrary standards of heterophoria, as in the selection of pilots for the Army Air Forces, the amount of the heterophoria and the reproducibility of the determinations become important. To illustrate the point, the exophoria limit for pilots for Class I in the Army Air Forces has been chosen as 5 prism diopters. An applicant for pilot training who has more than this amount will receive no further consideration. If, after attaining his coveted "wings," a pilot is found to manifest

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

From the Departments of Ophthalmology and Statistics of the Army Air Forces School of Aviation Medicine. The authors are deeply indebted to Herbert L. Moss for assistance in testing for heterophoria, and to Walter I. Shimer and Leter G. Mertz for assistance with the statistical analysis.

On leave from Oscar Johnson Institute, Department of Ophthalmology, Washington University School of Medicine, St. Louis, Mo.

On leave from the Department of Zoology, Ohio State University, Columbus, Ohio.

more than the limit of 5 prism diopters of exophoria at any time, he will be "grounded," perhaps for a time, perhaps permanently.

At least three sources of error may be recognized in the routine performance of tests now available. First, an individual may have inconstant amounts of manifest heterophoria.[8] This variation may be due either to a variation in the amount of manifest heterophoria of the subject or to inaccuracies in the testing technic. The two sources of variation are inseparable and may be considered together as individual variation. In evaluating tests for heterophoria, those which show little individual variation in comparison with the total range in the sample of tested subjects will be preferred over those which show large individual variation. This is the same as saying that the test must have high reproducibility or *reliability*.

Second, examiners may not all perform the test for heterophoria in exactly the same way. Particularly if a large number of individuals must be examined at many different places in short periods of time, as in time of war, the multitude of examiners will not have equal degrees of competence in the administration of the test.

Third, different tests or conditions of testing may not be equally effective in converting similar amounts of the total heterophoria into manifest heterophoria which can be measured. Despite the number of tests available, a comparison of the tests by modern statistical methods has seldom been made. Cridland's work[3] stands almost alone in this field.

The present study is directed toward answering some of the questions about the available tests for heterophoria. First, we shall estimate the reliability of the screen-Maddox rod test, and in the same experiment look for systematic differences between examiners which may contribute to unreliability. The screen-Maddox rod test was chosen because this test, described by Dolman,[4] has been in use throughout the Army Air Forces. Second, we shall make comparisons between several available tests, including among these the screen and parallax test which is universally regarded as being the most accurate one. These comparisons were made at two different testing distances, 20 feet for distant fixation and 13 inches for near fixation. Third, we shall attempt to estimate the effect of varying the testing conditions upon the Maddox rod test. The alternative conditions studied were a light *vs.* a dark room, the use of screening *vs.* not screening, the placing of the Maddox rod over the dominant *vs.* the non-dominant eye, and the use of a red *vs.* a white Maddox rod.

## DESCRIPTION OF TESTS

All of the tests used in this study are familiar to students of ocular muscle balance. With one exception, only brief descriptions will be given.

*Screen and parallax test.* A 1 cm. light was used at 20 feet and an ophthalmoscope with the head removed was used at 13 inches. A cover was shifted from one eye to the other, square prisms being changed behind the cover until any objective movement of the eye and any subjective movement of the light had ceased.

*von Graefe's prism diplopia test.* At a distance of 20 feet, a 20/100 size Snellen letter E was employed as a test object, while at 13 inches a similar letter E 2 mm. in height was used. The test object was adequately illuminated at both distances and an insuperable diplopia was created with a vertically placed 5 diopter prism before one eye. The examinee was asked to adjust the two images (by means of a Risley rotary prism) so that they were in the same vertical line.

*Screen-Maddox rod test.* Because the study to be reported concerns the technic of the screen-Maddox rod test, the test is described in full. At 20 feet, the test object was a muscle light 1 cm. in diameter; at 13 inches, it was an ophthalmoscope with the head removed. All lights in the immediate vicinity of the muscle light were turned out but the room was not entirely darkened. With the Maddox rod placed before the non-dominant eye, a cover is also held before the same eye and removed regularly for a period of 1 second at about 3 second intervals, meanwhile allowing the dominant eye to maintain fixation constantly. This very brief uncovering or "flashing" of the non-dominant eye has for its purpose a further weakening of the fusion control. For the measurement of heterophoria, a Risley rotary prism is rotated into position (also before the non-dominant eye) and set between 3 and 4 diopters "off" of the zero position; this forces the examinee to make some adjustment even though orthophoria is present. The examinee grasps the handle of the rotary prism and, while the muscle light is being "flashed" before the eye behind the Maddox rod, he is instructed to adjust the line so that it bisects the light.

*Maddox rod test.* This test is the same as the screen-Maddox rod test just described except that screening is omitted.

*Maddox wing test.* Used at 13 inches, this is a well-known test and needs no particular elaboration other than to mention that an arrow is seen by the right eye only, while calibrated lateral and vertical scales are seen by the left eye only.

*Thorington test.* This is also a well-known test and employs a Maddox rod. Linear scales, calibrated in units of 2 prism diopters, are drawn on the face of a card which is mounted on the head of a flashlight, a pin hole being located at the exact center of the card which is also the point of intersection of the lateral and vertical scales. The flashlight bulb is visible only through the hole in the card's center. The testing distance is 13 inches. A Maddox rod is placed before one of the examinee's eyes and he is asked to state at what point the line of light (as seen through the Maddox rod) intersects the calibrated scales.

## RELIABILITY OF THE SCREEN-MADDOX ROD TEST

The problem of reliability of the screen-Maddox rod test was approached from the point of view that if the test were not reproducible under relatively ideal conditions, then it was not likely to be reproducible under the routine testing conditions necessary for the physical examination for flying used in the Army Air Forces. The experiment was therefore planned to yield a maximum reliability of the screen-Maddox rod test.

*Plan of study.* Two examiners, who had watched each other work and whose testing technics were as nearly identical as possible, performed the tests. Both examiners tested each subject on each of two different days. No subject remained seated in the examination room for consecutive tests by both examiners; instead, one examiner examined the entire group of subjects for the day before turning them over to the second examiner. The test-retest interval varied from one to five days. Both lateral and vertical heterophoria measurements were made at each testing period. All subjects were tested at the 20 foot distance; none wore glasses and there was no correction of refractive error. The screen-Maddox rod test technic as outlined in the previous section was utilized throughout.

*Limitations of the study.* All subjects were aviation cadets who, theoretically,

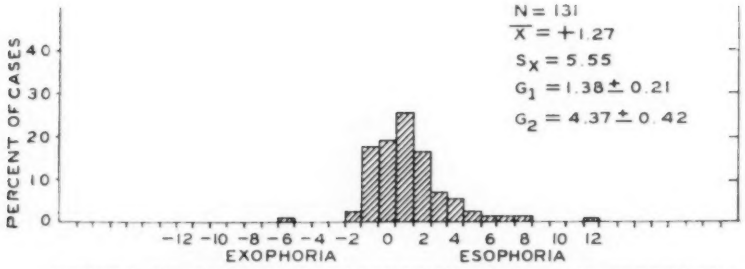



FIGURE 1. DISTRIBUTION OF 131 DETERMINATIONS OF HETEROPHORIA

had a visual acuity of at least 20/20 in each eye, a normal amount of accommodation and convergence, and heterophoria of not more than 1 prism diopter of hyperphoria, 10 prism diopters of esophoria, or 5 prism diopters of exophoria. The population available for study therefore represents a special selection from the general population with the consequence that the heterophoria readings extend over a limited portion of the entire range. Another limitation arises from the frequency distribution of heterophoria which was not normal in form. A distribution for 131 subjects, shown in figure 1, tails out to the right (the statistic of skewness, $g_1 = 1.38$, is 6.5 times its own standard error; the statistic of kurtosis, $g_2 = 4.37$, is 10.4 times its own standard error). To test the normality of distributions still further, data were borrowed from Cridland[3] for the purpose. In one of these (the red-green test) there is significant skewness to the right and in the other (the Maddox rod test) there is significant skewness to the left. The inconsistency of these findings suggests no general way of transforming the heterophoria scale to normality at this time.

*Results.* The results of testing 50 subjects for lateral heterophoria and 49 subjects for vertical heterophoria are summarized in table 1. Examiner A recorded an average of 1.10 prism diopters of esophoria and examiner B, an average of 1.30. This difference is not significant ($F = 3.92$ where $F_{.01} = 6.90$) and consequently there is no evidence of general disagreement between examiners. Furthermore the examiners agreed within the limits of experimental error for all subjects. (The interaction S×E is not significant.) Similar statements may be made with respect to the records of vertical heterophoria. Under the relatively ideal conditions of this study, therefore, it was found possible to achieve a high agreement between examiners, and the results are similar to those which may be expected if a single examiner performed all the testing.

The measurements of both lateral and vertical heterophoria are highly reliable when made with the screen-Maddox rod test. The coefficients of reliability are +0.95 and +0.92 for the two types of measurements, respectively. These coefficients are based upon the internal consistency of the four records taken on each subject and are computed by a method suggested by Hoyt.[6] In practical terms, these coefficients mean that any careful examiner can make heterophoria measurements with sufficient accuracy to discriminate between subjects. A notion of the absolute accuracy with which heterophoria may be measured may be obtained from the standard errors of single determinations. For lateral heterophoria, the standard error is 0.73 which means approximately that a lateral heterophoria measurement may be in error by as much as 1.5 prism diopters

TABLE 1

Average heterophorias obtained by two examiners using the screen-Maddox rod test, analysis of variance applied to mean differences, and reliability coefficients.

| Examiner | Lateral, n = 50 | | | Vertical, n = 49 | | |
|---|---|---|---|---|---|---|
| | 1st test | 2nd test | Average | 1st test | 2nd test | Average |
| A. | +1.02 | +1.18 | +1.10 | —0.06 | —0.11 | —0.09 |
| B. | +1.39 | +1.22 | +1.30 | —0.10 | —0.08 | —0.09 |
| | | | +1.20 | | | —0.09 |

| Variation (due to) | df. | Mean Square | F | df. | Mean Square | F | $F_{.01}$ |
|---|---|---|---|---|---|---|---|
| Subjects, S | 49 | 11.27 | 21.03 | 48 | 0.43 | 12.56 | 1.73 |
| Examiners, E | 1 | 2.10 | 3.92 | 1 | 0.001 | —— | 6.90 |
| S x E | 49 | 0.24 | —— | 48 | 0.04 | 1.12 | 1.73 |
| Error | 100 | 0.54 | | 98 | 0.03 | | |
| Standard error of a single determination | | 0.73 | | | 0.18 | | |
| Coefficient of (reliability) | | 0.95 | | | 0.92 | | |

Note: For lateral heterophoria, + means esophoria, — means exophoria; for vertical heterophoria + means right hyperphoria, — means left hyperphoria.

($2\times0.73$). For vertical heterophoria, the error may be as much as 0.4 prism diopters ($2\times0.18$).

### COMPARISONS BETWEEN TESTS

Many factors, both anatomic and innervational, determine the amount of heterophoria elicited by any one test at any one time. If it were possible to eliminate entirely all these innervational factors, then the eyes would assume the position variously referred to as the position of rest, the fusion-free position, or the fusion-frustrated position. Bielschowsky[2] has pointed out that any determination of the absolute position of rest is practically impossible because the totality of nervous influences cannot be eliminated in any way.

Various tests of heterophoria yield varying results in the same individual because varying numbers of innervational factors are eliminated by each of the different tests. In choosing a heterophoria test for use in testing large numbers of men, as in time of war, it is necessary to have an accurate test that can be administered in a minimum of time. Of all heterophoria tests, the screen and parallax test eliminates a greater number of innervational factors than any other test and thus allows the closest possible approach to the position of absolute rest. The screen and parallax test is considered somewhat tedious by the novice, however, and requires a fair degree of skill for proper performance. Hence it appears desirable to know how other tests of heterophoria compare with the screen and parallax test.

*Plan of study.* Fifty aviation cadets were tested by a single examiner for lateral heterophoria on eight tests, three with a testing distance of 20 feet, and five with a testing distance of 13 inches. At 20 feet, the screen and parallax test, von Graefe's prism diplopia test, and

TABLE 2

Average lateral heterophorias, standard deviations, and coefficients of correlation between tests for heterophoria.

| | 20 feet | | | 13 inches | | | | |
|---|---|---|---|---|---|---|---|---|
| | Screen and parallax | von Graefe prism diplopia | Screen-Maddox rod | Screen and parallax | von Graefe prism diplopia | Maddox wing | Thorington | Screen-Maddox rod |
| Mean $\bar{x}$ | +0.5 | +1.1 | +1.4 | —8.2 | —3.0 | —3.0 | —2.8 | —5.9 |
| Standard deviation $s_x$ | 2.2 | 1.9 | 2.8 | 7.4 | 5.2 | 3.4 | 4.3 | 5.8 |

Correlation coefficients, r

| 20 feet | | | | 13 inches | | | | |
|---|---|---|---|---|---|---|---|---|
| von Graefe prism diplopia | +0.84 | —— | —— | von Graefe prism diplopia | +0.72 | —— | —— | —— | —— |
| Screen-Maddox rod | +0.81 | +0.85 | —— | Maddox wing | +0.68 | +0.76 | —— | —— | —— |
| | | | | Thorington | +0.77 | +0.88 | +0.70 | —— | —— |
| | | | | Screen-Maddox rod | +0.48 | +0.41 | +0.29 | +0.35 | —— |

Note: For lateral heterophoria, + means esophoria, — means exophoria.
To be significant, any r should exceed $r_{.01} = +0.36$.

the screen-Maddox rod test were used. At 13 inches, the screen and parallax test, von Graefe's prism diplopia test, the Maddox wing test, the Thorington test, and the screen-Maddox rod test were used.

*Results.* It is obvious from the summary of results in table 2 that the tests which use a testing distance of 20 feet do not agree precisely with each other. Yet the level of agreement is reasonably high considering the diverse nature of the tests. All three tests yielded about the same average heterophoria and about the same spread around the averages. The coefficients of correlation are equal within the limits of random sampling. There is no clear choice among these three tests based upon statistical evidence alone. From the standpoint of ease of performance and time required, the screen-Maddox rod test may be preferred by some to either von Graefe's prism diplopia test or the screen and parallax test.

When the heterophoria tests which use a testing distance of 13 inches are considered, the screen-Maddox rod test is apparently no longer the satisfactory test that it was at 20 feet (see table 2)

The screen and parallax test, as expected, uncovered the highest average exophoria, 8.2 prism diopters. The screen-Maddox rod test uncovered an average of 5.9 prism diopters of exophoria. The other three tests were about equivalent. They uncovered an average of 3.0 prism diopters of exophoria in the sample of 50 subjects. The variabilities about the averages, as judged by the

standard deviations, are quite different. Consequently on the basis of the means and standard deviations alone, the five tests at 13 inches cannot be regarded as equivalent. The coefficients of correlation reveal that four of the tests would rank the subjects in approximately the same order of degree of heterophoria. The exception is the screen-Maddox rod test which is not sufficiently correlated with the other four tests to be used as a replacement for any one of those tests. The coefficient of correlation of the Thorington test with the screen and parallax test is 0.77, while that of von Graefe's prism diplopia test is 0.72, and that of the Maddox wing test is 0.68. The screen-Maddox rod test trails all the others with a correlation coefficient of only 0.48.

Although the screen-Maddox rod test is apparently in a class by itself at a testing distance of 13 inches, this may in part be due to the fact that the sample is preselected on the basis of the screen-Maddox rod test. Removal of this restriction might yield a higher correlation with the screen and parallax test. However, in another sample of similar subjects tested in similar fashion, the screen-Maddox rod test agreed well with the screen and parallax test. This sample is discussed in a later section.

It must be realized that the screen-Maddox rod test at 13 inches, although apparently in a class by itself, may possibly be the best instead of the worst test of heterophoria and be measuring something that the other tests are not. It is unfortunate that the present state of our knowledge is insufficient to allow a decision to be made.

### CONDITIONS OF THE MADDOX ROD TEST

The screen-Maddox rod test has been found to have a satisfactory self-consistency, at least under ideal conditions, and a satisfactory agreement with the screen and parallax test when the testing distance is 20 feet. These facts, plus its relative ease of administration, make it a desirable test for use for routine measurements of heterophoria on large numbers of subjects, as in time of war.

The screen-Maddox rod test was chosen as the standard test for the Physical Examination for Flying (WD AGO Form No. 64). Certain variations in the testing conditions appeared during the war, and their effect upon the heterophoria measurements was not known, at least not in quantitative terms. Among the departures from uniform conditions, the following alternatives were regarded as important enough to merit special study: a light *vs.* a dark room, the use of screening *vs.* not screening, the placing of the rod over the dominant *vs.* the non-dominant eye, and the use of a red *vs.* a white Maddox rod.

The effect of illumination in the testing room was investigated in a separate experiment. The other three alternatives were incorporated into a single experiment.

*Effect of Illumination.* There are conflicting arguments in the literature on heterophoria about the amount of illumination required or permitted in the testing room. Those who prefer testing in darkness contend that, if only a spot of light is visible in an otherwise dark room, any planes of reference which would serve to fix accommodation will be lost in the darkness and therefore eliminated as variables. More heterophoria would be uncovered in this manner and hence the test made more accurate. The assumption behind this argument is, of course, that the test uncovering the most heterophoria is the best.

In contrast, those who prefer testing in an illuminated room maintain that the subject in darkness is in an abnormal situation in no way comparable to conditions requiring visual efficiency which

TABLE 3

Comparison of heterophorias determined in light and in dark. n = 60 subjects.

| | Lateral | | Vertical | |
|---|---|---|---|---|
| | Light | Dark | Light | Dark |
| Mean, $\bar{x}$ | +1.17 | +1.02 | —0.03 | —0.02 |
| S.D., $s_x$ | 2.20 | 2.37 | 0.24 | 0.25 |
| Correlation, r | +0.89 | | +0.90 | |
| Mean difference | 0.150 | | —0.008 | |
| Standard error of mean difference | ±0.137 | | ±0.015 | |
| Ratio, t* | 1.10 | | 0.55 | |

*For the mean difference to be significant, t should exceed 2.66.

For lateral heterophoria, + means esophoria, — means exophoria;
for vertical heterophoria, + means right hyperphoria, — means left hyperphoria.

might reasonably be affected by heterophoria.[4]

*Methods.* In an experiment designed to resolve this difference of opinion, lateral and vertical heterophoria was determined under two alternative conditions at a testing distance of 20 feet upon 60 subjects. All the subjects were aviation cadets and thus were a select group. The two conditions of testing were:

1. *Dark.* The testing room was in complete darkness except for a muscle light 1 cm. in diameter located 20 feet from the subject. The walls and ceiling of the room were painted black.

2. *Light.* The same room was used except that a 60-watt, frosted Mazda bulb was burning directly above the subject's head. Illumination measured 5 foot candles at head-height directly beneath the bulb (9 foot ceiling) and 0.5 foot candles at the end of the room (20 feet away) where the muscle light was located. In this illumination, all objects in the room could be seen fairly distinctly.

There was no appreciable lapse of time between tests, the light room and dark room determinations being made at the same examination on each subject, although the order of the two tests was reversed in every other subject.

*Results.* The results of the tests for heterophoria using the screen-Maddox rod test under the two conditions of illumination are summarized in table 3. For this group of 60 subjects, the following assertions may be made:

1. The average heterophorias, determined in light and in dark, are not significantly different. This is true for both lateral and vertical heterophoria.

2. The variability about the average, measured in terms of the standard deviation, is essentially the same for the two conditions of illumination, for both lateral and vertical heterophoria.

3. There is a high correlation between the determinations of heterophoria in light and in dark, for both lateral and vertical heterophoria.

There is no evidence, therefore, from this experiment to support the claim that amounts of illumination up to that used in this study will significantly affect the measurement of heterophoria. Cridland[3] reached a similar conclusion using the Maddox rod test (without screening).

TABLE 4

Average heterophorias at 20 feet and at 13 inches. n = 100 subjects.

| | 20 feet | | | 13 inches | | |
|---|---|---|---|---|---|---|
| | With screening | Without screening | Average | With screening | Without screening | Average |
| Dominant eye | (1) | (2) | | (1) | (2) | |
| Red rod | +1.80 | +1.49 | +1.65 | —4.97 | —5.33 | —5.15 |
| | (3) | (4) | | (3) | (4) | |
| White rod | +1.58 | +1.11 | +1.35 | —4.44 | —5.33 | —4.89 |
| Average | +1.69 | +1.30 | +1.50 | —4.71 | —5.33 | —5.02 |
| Non-dominant eye | (5) | (6) | | (5) | (6) | |
| Red rod | +1.89 | +1.55 | +1.72 | —4.99 | —5.04 | —5.02 |
| | (7) | (8) | | (7) | (8) | |
| | +1.70 | +1.33 | +1.52 | —4.70 | —4.99 | —4.85 |
| Average | +1.80 | +1.44 | +1.62 | —4.85 | —5.02 | —4.92 |
| Average | | | | | | |
| Red rod | +1.85 | +1.52 | +1.68 | —4.98 | —5.19 | —5.08 |
| White rod | +1.64 | +1.22 | +1.43 | —4.57 | —5.16 | —4.87 |
| Average | +1.74 | +1.37 | +1.56 | —4.78 | —5.17 | —4.98 |

+ means esophoria
— means exophoria

*Effect of Screening, Eye Dominance, and Rod Color*

*Methods.* One hundred subjects, all aviation cadets, were tested for heterophoria for near and distant fixation in a refracting alley 20 feet in length whose walls were painted black. The room was illuminated as described in the previous section on Maddox rod determinations in a "light" room. The muscle light used at the 20 foot distance was a spot of light 1 cm. in diameter. At the 13 inch distance, the light was an ophthalmoscope with the head removed. Each subject was seated comfortably in a chair and determination of the dominant eye made by asking the subject to sight the muscle light across the room through a 1 cm. hole in a 10″×10″ board which was held at arm's length with both hands, both eyes being held open. Heterophoria measurements were first made by the method of screen and parallax using the testing distances of 20 feet and 13 inches and employing a Beren's plastic prism bar. The calibrations on the bar had been checked and found to be correct at a previous time.

After this measurement, a phorometer was accurately adjusted before the subject and testing was begun with the Maddox rod. Both red and white Maddox rods were used in testing before each eye separately, with and without the use of screening. This was done at testing distances of both 20 feet and 13 inches. All possible combinations of the four variables just mentioned were tested on each subject. This necessitated 16 separate measurements on each subject, the measurements being taken in random order.

The heterophorias determined at 20 feet are different from the heterophorias determined at 13 inches in several respects. In the present series of deter-

TABLE 5

Standard deviations of heterophoria distributions at 20 feet and at 13 inches

n = 100 subjects

| | 20 feet | | 13 inches | |
|---|---|---|---|---|
| | With screening | Without screening | With screening | Without screening |
| Dominant eye | (1) | (2) | (1) | (2) |
| Red rod | 2.65 | 2.67 | 5.29 | 5.52 |
| | (3) | (4) | (3) | (4) |
| White rod | 2.67 | 2.40 | 5.24 | 5.53 |
| Non-dominant eye | (5) | (6) | (5) | (6) |
| Red rod | 2.98 | 3.07 | 5.66 | 6.07 |
| | (7) | (8) | (7) | (8) |
| White rod | 2.90 | 2.38 | 5.75 | 5.78 |

Test for homogeneity of variances:

$X^2 = 12.15$ $X^2 = 2.79$
$P = .10 - .05$ $P = .95 - .90$

minations upon 100 subjects, the average at 20 feet was about 1.5 prism diopters of esophoria, whereas the average at 13 inches was about 5.0 prism diopters of exophoria (table 4). The variation about the averages of the determinations at 20 feet is quite unlike that of the determinations at 13 inches (table 5). These facts make it necessary to preserve a separation of the data collected at the two different distances, and to look for the effects of screening, the dominance of the eye, and the colors of the rod separately in the two bodies of data. Further, the variabilities in the eight distributions of heterophoria for the eight experimental conditions with a testing distance of 20 feet were found to be homogeneous when tested by the method of Bartlett.[1] It appears, therefore, that whatever change is introduced into the heterophoria determination at 20 feet by a change in the experimental conditions, the change affects the average only and does not affect the distribution about the average. A similar assertion may be made for the changes introduced in the 13 inch test.

*Screening vs. not screening at 20 feet.* When a Maddox rod is used in heterophoria determinations, it is placed before one of the examinee's eyes. The eye behind the rod perceives a spot of light as a line of light while the other eye sees the spot as it really is. This distortion or dissimilarity of images disrupts fusion to a certain extent, thus allowing the eye behind the rod to deviate toward its dissociated position. The extent of this deviation may be measured exactly by the utilization of prisms. Dissimilarity between the two images of the same object as perceived by the two eyes does *not* completely abolish the fusion impulse.[2,9]

The cover test, i.e., alternate occlusion of each eye separately, breaks up fusion as completely as is possible without actually occluding both eyes simultaneously. The covering or screening principle is the basis of the screen and parallax test.

The addition of intermittent screening of the eye behind the Maddox rod in testing heterophoria allows this test to approximate more closely the screen

and parallax test, since the same basic principle is common to both. Omission of screening from the screen-Maddox rod test would seem to alter the test.

When the screen was used at 20 feet the average heterophoria was found to be 1.74 prism diopters of esophoria; when the screen was not used, the average was 1.37 prism diopters of esophoria. The difference of 0.37 prism diopters is statistically significant, and indicates that on the average the use of the screen adds about 0.3 or 0.4 prism diopters to the heterophoria reading.

This difference due to screening appears to exist regardless of the color of the Maddox rod or of the dominance of the eye. When a red rod was used, the average was 1.85 prism diopters of esophoria with screening and 1.52 prism diopters of esophoria without screening, giving a difference of 0.33 prism diopters. When a white rod was used, the averages were 1.64 and 1.22 prism diopters of esophoria with and without screening respectively, giving a difference of 0.42 prism diopters. With the rod before the dominant eye, the difference was 0.39 prism diopters (1.69 with screening minus 1.30 without screening), and before the non-dominant eye, the difference was 0.36 prism diopters (1.80 minus 1.44). Table 4 contains a summary of the means for all combinations of the experimental conditions.

*Screening vs. not screening at 13 inches.* At 13 inches, effects comparable to those at 20 feet were found. With screening, the average heterophoria was 4.78 prism diopters of exophoria; whereas, without screening, the average was 5.17 prism diopters of exophoria, or an increase of 0.39 prism diopters. This difference is statistically significant, and is the same in magnitude and direction as the shift observed at 20 feet when screening was omitted. (An increase in exophoria is the same in direction as a decrease in esophoria.)

The effect of screening is present with the rod before either the dominant or the non-dominant eye, and with both colors of rods. When the rod is before the dominant eye, the change is 0.62 prism diopters, i.e., from 4.71 with screening to 5.33 without screening, and when the rod is before the non-dominant eye, the change is 0.17 prism diopters, i.e., from 4.85 to 5.02. With the red and white rods, the changes are 0.21 and 0.59 prism diopters respectively (table 4).

It is necessary to specify, therefore, that screening be done or omitted throughout the routine testing of heterophoria, whether at 20 feet or at 13 inches.

*Dominant vs. non-dominant eye at 20 feet.* The question of whether the Maddox rod should be placed before the dominant or the non-dominant eye during heterophoria testing is one of long standing. Dolman[4] suggested placing the rod before the non-dominant eye on the basis of observations of 100 cases, 81 per cent of which revealed different heterophoria measurements when each eye was tested separately. He observed that 66 per cent exhibited more heterophoria when the rod was placed before the non-dominant eye, 7 per cent had more with the rod before the dominant eye, and 17 per cent gave identical figures regardless of which eye was behind the Maddox rod. Dolman made conflicting interpretations of these observations when he said, ". . . it is evident . . . that the unequal degrees of deviation depend upon the fact that the role of fixation is given to each eye in turn. The stimulus received by the retina of the eye fixing the test object (fixation reflex) determines the direction of both visual axes when the other eye is screened."

When Dolman's data are subjected to statistical analysis, it becomes obvious that his conclusions, drawn on analysis of percentages only, are erroneous. Actually there is in Dolman's data no significant difference between results of testing with the Maddox rod before first the dominant eye and then the non-dominant eye. Clinical testing for heterophoria varies in this respect. Placement of the Maddox rod before the dominant eye is recommended by many clinicians on the assumption that occlusion of the habitually fixing eye more completely disrupts fusion. Others routinely place the Maddox rod before the right eye, which means that the Maddox rod is before the dominant eye about 75 per cent of the time.

No significant difference was discovered between heterophoria measures with the Maddox rod placed separately before either the dominant or the non-dominant eye under any condition tested in this study. The average with the rod before the dominant eye was 1.50 prism diopters of esophoria, and before the non-dominant eye was 1.62 prism diopters of esophoria. This similarity exists with or without screening, and with the red or with the white Maddox rod.

*Dominant vs. non-dominant eye at 13 inches.* There was no significant difference between heterophoria measured with the Maddox rod placed separately before either the non-dominant or the dominant eye at 13 inches. When the rod was before the dominant eye the average was 5.02 prism diopters of exophoria, and when the rod was before the non-dominant eye the average was 4.92 prism diopters of exophoria. This similarity in heterophoria reading is present with or without screening, and with a red or with a white rod.

It does not appear necessary to designate whether the dominant or the non-dominant eye shall be used in the routine testing for heterophoria, at 20 feet or at 13 inches. This conclusion is consistent with Dolman's[4] data.

*Red vs. white Maddox rod at 20 feet.* The color of the Maddox rod used to measure heterophoria may be important in testing because the human eye is approximately 0.5 diopter hypermetropic for the color red.[5] Thus the eye should accommodate 0.5 diopter more in clearly imaging a line seen through a red Maddox rod than one seen through a white Maddox rod. This increased accommodation should create an increased convergence tendency which may manifest itself as esophoria. Thus if a red rod is used instead of a white one, we may logically expect exophoria to decrease and esophoria to increase. A white rod should theoretically yield a more accurate measure for this reason. Another argument for the use of the white Maddox rod instead of the red one is that the white rod is usually far more optically accurate than the red by virtue of the production methods utilized for the two rods.[4]

In this study, changing from a red to a white rod was, on the average, equivalent to subtracting 0.25 prism diopters of esophoria from the reading. With the red rod, the average reading was found to be 1.68 prism diopters of esophoria; with the white, it was 1.43 prism diopters of esophoria. The difference is statistically significant.

This difference due to color of the rod was found to exist when the rod was before either the dominant or the non-dominant eye. With the rod before the dominant eye, the change from a red to a white rod produced 0.30 prism diopters less of esophoria; with the rod before the non-dominant eye, the change produced 0.20 prism diopters less of esophoria. Likewise the difference introduced by the color of the rod persisted in the presence or absence of screening.

TABLE 6

Correlation coefficients and regression coefficients for Maddox rod test. n = 100 subjects.

| Variables i | Variables j | 20 feet Correlation $r_{ij}$ | 20 feet Regressions $b_{ij}$ | 20 feet Regressions $b_{ji}$ | 13 inches Correlation $r_{ij}$ | 13 inches Regressions $b_{ij}$ | 13 inches Regressions $b_{ji}$ |
|---|---|---|---|---|---|---|---|
| A. With screening *vs.* without screening | | | | | | | |
| 1 | 2 | 0.88 | 0.87 | 0.89 | 0.87 | 0.83 | 0.91 |
| 3 | 4 | 0.90 | 1.00 | 0.81 | 0.89 | 0.84 | 0.94 |
| 5 | 6 | 0.95 | 0.92 | 0.98 | 0.84 | 0.78 | 0.90 |
| 7 | 8 | 0.88 | 1.07 | 0.72 | 0.83 | 0.83 | 0.83 |
| B. Red rod *vs.* white rod | | | | | | | |
| 1 | 3 | 0.93 | 0.92 | 0.94 | 0.88 | 0.89 | 0.87 |
| 2 | 4 | 0.93 | 1.04 | 0.84 | 0.89 | 0.89 | 0.89 |
| 5 | 7 | 0.93 | 0.96 | 0.90 | 0.91 | 0.90 | 0.92 |
| 6 | 8 | 0.91 | 1.17 | 0.71 | 0.92 | 0.97 | 0.88 |
| C. Dominant eye *vs.* non-dominant eye | | | | | | | |
| 1 | 5 | 0.80 | 0.71 | 0.90 | 0.90 | 0.84 | 0.96 |
| 2 | 6 | 0.78 | 0.68 | 0.90 | 0.80 | 0.73 | 0.88 |
| 3 | 7 | 0.88 | 0.81 | 0.96 | 0.89 | 0.81 | 0.98 |
| 4 | 8 | 0.83 | 0.84 | 0.82 | 0.80 | 0.77 | 0.84 |

The numeral used to designate the variables may be identified on the table of means (table 4).

$b_{ij}$ is the regression of i on j.

$b_{ji}$ is the regression of j on i.

With screening, the white rod gave 0.21 prism diopters less of esophoria than the red rod; without screening, the white rod gave 0.30 prism diopters less. The difference between the red and the white rod is a little less than the 0.50 prism diopters expected on theoretical grounds.

For routine determinations of lateral heterophoria, it appears to be important to specify the color of the rod which is to be used in the testing at 20 feet.

*Red vs. white Maddox rod at 13 inches.* Changing the color of the rod did not significantly change the heterophoria reading at 13 inches. With a red rod, the average was 5.08 prism diopters of exophoria, and with a white rod, 4.87 prism diopters of exophoria. This similarity was found both in the presence and absence of screening, and with either the rod before the dominant or the non-dominant eye. For determinations of heterophoria at 13 inches, therefore, it does not appear to be necessary to designate the color of the Maddox rod.

The fact that the color of the Maddox rod makes a significant difference at a testing distance of 20 feet but none at 13 inches can be explained on logical grounds. At 20 feet, the patient is using a minimum of accommodation and the increase of 0.5 diopter necessary to clearly image the red rod's line is sufficient to become manifest as an increase in esophoria. At 13 inches, however, a minimum of 3 diopters of accommodation is being exerted and the added 0.5 diopter necessitated by changing from a white to a red Maddox rod is not sufficient to become manifest as more esophoria.

*Correlation between measurements of heterophoria at 20 feet.* While an average increase of 0.37 prism diopters was

introduced by the use of the screening, this amount of change did not occur exactly for each subject under each of the combinations of testing conditions. The high uniformity of the change, however, may be seen from product moment coefficients of correlation in table 6. The correlation coefficients between the screening and not screening measurements average about +0.90, which is equivalent to saying that knowledge of heterophoria determined without screening may be used to predict heterophoria with screening with a high degree of accuracy. Similar correlation coefficients for the red rod versus the white rod tests, and for the rod before the dominant eye versus the rod before the non-dominant eye likewise show high relationships for these testing conditions.

To facilitate transformation of heterophoria readings taken at 20 feet with screening into readings without screening, linear regression coefficients are included in table 6. To illustrate the use of the regression coefficients, assume that a measurement of heterophoria was made on a single individual with a light at 20 feet and a red rod before the non-dominant eye using screening. Assume further that it is desired to transform this measurement into that expected if screening had not been used. The regression of not screening on screening ($b_{ns}$ in table 6) is 0.98 for these conditions, which says that for each diopter change in heterophoria measured with screening, there is on the average 0.98 prism diopter change in the same direction without screening. Substitution in the following formula will accomplish the desired transformation.

| Predicted heterophoria without screening | = | Average heterophoria without screening | + | Regression of screening -uou uo screening | Observed heterophoria with screening | — | Average heterophoria with screening |
|---|---|---|---|---|---|---|---|

Taking the appropriate values for the conditions specified (red rod before non-dominant eye), gives

Predicted=1.55 + 0.98 (Observed — 1.89)

Assume that 4 prism diopters of esophoria were observed in a subject with screening. The predicted heterophoria for this subject without screening is 3.6 prism diopters of esophoria.

*Correlation between measurements at 13 inches.* The correlation coefficients for the intensity of the relationship between measurements at 13 inches are sufficiently high to enable accurate prediction from a measurement taken under one circumstance to a measurement under another (table 6).

*Correlation of Maddox rod tests with the screen and parallax tests at 20 feet.* The screen and parallax test using a light source at 20 feet from the subjects gave an average of 0.64 prism diopters of esophoria (table 7). This is about one diopter less than the average of the eight variations of the Maddox rod tests. The measurements of heterophoria by the eight variations of the Maddox rod test correlate equally well with measurements made by the screen and parallax test. At first glance, there appears to be higher correlations between the screen and parallax test and all variations of the Maddox rod test when the rod is before the non-dominant eye than when the rod is before the dominant eye. The difference between the corresponding correlations was found to be non-significant (by z transformation). There is consequently no obvious advantage of testing with the non-dominant eye. Further, there is no general advantage of

TABLE 7

Correlation of screen and parallax test with eight variations of the Maddox rod test. n = 100 subjects.

| | 20 feet | 13 inches |
|---|---|---|
| Screen and parallax test | | |
| Mean | +0.64 diopters (eso.) | —8.07 diopters (exo.) |
| Stan. Dev. | 1.70 | 5.99 |
| Correlation coefficients of screen and parallax test with Maddox tests. | | |
| D-R-S | +0.56 | +0.60 |
| D-R-O | +0.60 | +0.59 |
| D-W-S | +0.73 | +0.64 |
| D-W-O | +0.65 | +0.60 |
| N-R-S | +0.82 | +0.77 |
| N-R-O | +0.77 | +0.70 |
| N-W-S | +0.76 | +0.73 |
| N-W-O | +0.77 | +0.79 |

D = rod before dominant eye R = red rod S = with screening
N = rod before non-dominant eye W = white rod O = without screening

the screen-Maddox rod test over the Maddox rod test alone (without screening) so far as the correlation with the screen and parallax test is concerned.

At this testing distance, the results in this experiment agree with those for a previous experiment summarized in table 2, where the correlation between the screen-Maddox rod test and the screen and parallax test was found to be 0.81.

*Correlation of the Maddox rod tests with the screen and parallax test at 13 inches.* With respect to correlations with the screen and parallax test there is no obvious choice among the variations of the Maddox rod test at a testing distance of 13 inches (table 7). However, in this sample, the correlations are all higher than the correlation found in the previous experiment (table 2). No obvious explanation for this discrepancy has occurred to us.

*Note on statistical method.* These two experiments, at near and distant fixation, are factorial in type with three factors being tested at each of two levels. When it was found that the variances of heterophoria at 20 feet constituted a homogeneous set which was definitely different from the variances at 13 inches, the data for the two distances were kept separate. This made it possible to use the method of analysis of variance to detect the main effects of screening, eye dominance, and rod color, as well as the interactions of these factors. The results of the statistical analysis are summarized in table 8, in which evidence is contained for each of the assertions about significance or non-significance of the difference made in the preceding sections.

The design of this experiment also makes possible an estimate of reliability of the measurement of heterophoria using the methods of internal consistency proposed by Hoyt.[6] For heterophoria measured with a testing distance of 20 feet, the reliability coefficient was 0.976, and with a distance of 13 inches the coefficient was 0.975.

TABLE 8

Analysis of variance for heterophorias at 20 feet and at 13 inches; 100 subjects tested 16 times, i.e., at two distances, with and without screening, with red and white rods, and with rod before the dominant and the non-dominant eyes.

| Source | df. | 20 feet Mean square | F | 13 inches Mean square | F | $F_{.01}$ |
|---|---|---|---|---|---|---|
| Screening (with-without) K | 1 | 27.75 | 54.41 | 31.60 | 11.05 | 6.70 |
| Lighting (red-white) M | 1 | 12.75 | 25.00 | 9.46 | 3.31 | 6.70 |
| Eyes (dom., non-dom.) O | 1 | 3.00 | 5.88 | 1.53 | ..... | 6.70 |
| Subjects, N | 99 | $s^2_n$ = 50.98 | 99.96 | $s^2_n$ = 215.40 | 75.31 | 1.42 |
| K x M | 1 | 0.45 | ..... | 7.41 | 2.59 | 6.70 |
| K x O | 1 | 0.06 | ..... | 10.35 | 3.62 | 6.70 |
| K x N | 99 | 1.55 | 3.04 | 9.63 | 3.37 | 1.42 |
| M x O | 1 | 0.45 | ..... | 0.45 | ..... | |
| M x N | 99 | 0.92 | 1.80 | 6.10 | 2.13 | 1.42 |
| O x N | 99 | 4.00 | 7.84 | 10.37 | 3.63 | 1.42 |
| Remainder | 397 | $s^2_e$ = 0.51 | | $s^2_e$ = 2.86 | | |
| All interactions | 697 | $s^2_g$ = 1.21 | | $s^2_g$ = 5.36 | | |
| Estimate of reliability, based on $s^2_n$ and $s^2_g$ $r_{tt}$ | | = 0.976 | | = 0.975 | | |


SUMMARY

1. The reproducibility or reliability of measurements of heterophoria when taken by two examiners using the screen-Maddox rod test was found in one sample of aviation cadets to be satisfactorily high. The coefficients of reliability were +0.95 for lateral heterophoria and +0.92 for vertical heterophoria at a testing distance of 20 feet. In another sample, after removal of systematic effects due to the use of screening and to changing the color of the rod, the Maddox rod test both at 20 feet and at 13 inches had a reliability coefficient of +0.97 for lateral heterophoria.

2. There is no significant difference in results obtained by two different examiners in administering the screen-Maddox rod test.

3. Several tests for heterophoria were compared with the screen and parallax test, which is considered the most accurate test. At a testing distance of 20 feet, a high correlation was found with von Graefe's prism diplopia test and with the screen-Maddox rod test. At a testing distance of 13 inches, a high correlation was found with von Graefe's prism diplopia test, the Maddox wing test, and the Thorington test. The correlation with the screen-Maddox rod test was somewhat lower in one experiment, but high in another.

4. The effect of varying the testing conditions upon the Maddox rod test was determined. The amount of illumination had no significant effect. It makes no difference whether the Maddox rod is placed over the dominant or the non-dominant eye. At a testing distance of 20 feet, a red Maddox rod uncovers more esophoria than does a white one, but there is no difference between the effect of the red and white rods at 13 inches. With screening the Maddox rod test uncovers more esophoria or less exophoria than without screening.

5. The Maddox rod test for all possible combinations of four alternative testing conditions was found to be high-

ly correlated with the screen and parallax test.

6. The following conclusions may be drawn from the results.

a. Heterophoria measurements may be performed with sufficient accuracy to discriminate between individuals.

b. The Maddox rod test gives results sufficiently like those of the screen and parallax test to be used as a replacement for it for purposes of routine testing.

c. It is recommended that the Maddox rod test be performed with a white rod, without screening, and without regard to placing the rod before the dominant or non-dominant eye for purposes of routine testing.

## REFERENCES

1. Bartlett, M. S.: Properties of sufficiency and statistical tests, Proc. Roy. Soc., s. A, 160: 268-282, 1937.
2. Bielschowsky, A.: Lectures on motor anomalies, Hanover, N. H., Dartmouth College Publications, 1940.
3. Cridland, Nigel: Measurement of heterophoria, Brit. J. Ophth., 25:141-167 (April) 1941.
4. Dolman, P.: Relation of the sighting eye to the measurement of heterophoria, Am. J. Ophth., 3:258 (April) 1920.
5. Duke-Elder, W. S.: Practice of refraction, Philadelphia, P. Blakiston's Son & Co., Inc., 1928, p. 66.
6. Hoyt, C.: Test reliability estimated by analysis of variance, Psychometrika, 6:153, 1941.
7. Lancaster, W. B.: Fifty years' experience in ocular motility. Am. J. Ophth., 24:485 (May); 619 (June); 741 (July); 1941.
8. Scobee, R. G.: Heterophoria, Air Surgeon's Bull., 2:38-39 (Feb.) 1945.
9. White, J. W.: Hyperphoria; diagnosis and treatment, Arch. Ophth., 7:739-747 (May) 1932.

## DISCUSSION

JOHN H. DUNNINGTON, M.D., New York, N. Y.: The findings in this careful statistical survey of tests for heterophoria furnish much food for thought. The meticulous manner in which the tests were done makes their results all the more valuable. They have demonstrated that two careful examiners can reproduce more or less identical results with the various tests at our command. They have compared the commonly employed methods and pointed out in a most convincing manner the fluctuations one can expect to find in normal individuals with each test. Their observations on the unimportance of (1) the amount of illumination, (2) the eye before which the Maddox rod is placed will do much to clarify the use of this test. Their findings that "with screening the Maddox rod test uncovers more esophoria or less exophoria than without screening" is in direct contrast to the generally accepted idea viz: with the Maddox rod one usually finds a higher degree of esophoria or less exophoria than with the screen and parallax test. My only explanation for this discrepancy is that these tests were made on normal individuals in whom all variations are reduced to a minimum. I believe if similar tests were made on pathologic subjects these findings would be reversed. At least in my experience such has been the case. A similar objection can be raised to their conclusions "The Maddox rod test gives results sufficiently like those of the screen and parallax test to be used as a replacement for it for purposes of routine testing." Since tests for heterophoria are dependent upon suspension of fusion and the Maddox rod admittedly "does not completely abolish the fusion impulse" it seems to me that routine adoption of this test will necessarily lead to erroneous findings. These examinations were made upon subjects with muscle findings well within the normal limits so one should expect to find a minimal variation in this group whereas if it is used as the routine method much greater discrepancies will become manifest. The use of the screen with the Maddox rod was advocated by Dolman because during World War I we found this situation to exist. Several cases of high exophoria were detected by the screen and parallax test which had previously passed the Maddox rod test. In routine office practice the Maddox rod is particularly useful for the detection of small amounts of hyperphoria and is a valuable adjunct to our muscle armamentarium, but I for one am not ready to adopt it routinely to the exclusion of the screen and parallax test.

Such thorough analyses are so rare that I must congratulate the authors upon their painstaking efforts and excellent presentation from which all of us have learned a lot. I only hope that similar investigations upon a group of routine office patients will soon be forthcoming.

GLEN G. GIBSON, M.D., Philadelphia, Pa.: Dr. Scobee and Dr. Green are to be congratulated for making this scientific contribution to

ophthalmology while under the pressing circumstances of wartime military service. In my opinion this is an important piece of work because it focuses attention on a problem which is to become progressively more important, namely, the visual qualifications for aviation. Since the objective of all is to learn the truth about this subject, it is probably fortunate that the military ophthalmologists are primarily concerned with the best of the population in the field of muscle balance, while the clinical ophthalmologist is necessarily more interested in the other extreme. Approaching this problem from these two extremes one would anticipate greater differences of opinion than actually exist.

The authors have carefully studied an adequate group of subjects and correctly concluded, with due regard for the limitations of their study, that two men can on this selected group, make measurements with the screen-Maddox rod test, which are repeatable and statistically reliable. I feel that in general this is quite true, as day to day measurements of heterophoria probably do not vary much if the conditions of the test are repeated. Statements to the contrary in the literature, for the most part, refer to the more pathologic cases than to normal heterophoria. If the screening adds only .3 to .4 prism diopters in the maddox-rod test to the reading without screening, I do not see the necessity for screening in the test in which the clinical error is easily 1 diopter, as the authors prove. It is interesting and instructive that the authors demonstrate such an agreement in the screen-parallax test and the screen-Maddox rod test. In clinical work I have not been able to routinely demonstrate such a close parallelism, particularly in the very low and in the higher amounts of heterophoria.

One wonders if the incentive for this work was a recognition by the authors that present testing technics and standards were excluding good men from military service. I appreciate that it is not within the province of this paper to justify the Air Corps standards, but for one who is unfamiliar with military standards, I find it difficult to understand why 5 prism diopters of exophoria is set as the dividing line between acceptable and non-acceptable men, and I should like to ask Dr. Scobee if he would care to answer whether this is a fair end point for the discrimination between individuals.

The authors have permanently settled the fact that total darkness is not necessary, and that it is not necessary to place the rod before the non-dominant eye. It is reassuring to have statistical confirmation of these aspects of our testing technic.

This paper manifests the necessity for more precise definitions, classifications, and interpretations in the field of muscle balance based on a better understanding of the reflex mechanisms involved. Most textbooks consider heterophoria as a subdivision of motor anomalies when the problem is primarily a sensory one. What we are measuring is the motor compensation for sensory conditions. Most cases of heterophoria are instances of imperfection but they are not anomalies. There is need for more precise definitions between normal heterophoria and clinically significant heterophoria.

Fusion does not perform a motor task as the authors state. Fusion is a purely sensory process which occurs after the fusion-vergence reflexes have produced the motor adjustment necessary to bring the eyes from the disassociated to the associated position. The fact that this is an insignificant task in the great majority of cases should be appreciated in setting standards for this subject. I feel that many of the points the authors have made will stand the test of time and prove to be of permanent value, and it is hoped that they will make a similar survey on cases with clinically significant heterophoria in the near future.

I wish to express my sincere thanks to Dr. Scobee for this most instructive paper.

WILLIAM F. BONNER, M.D., Temple, Texas: In a paper presented before the Philadelphia College of Physicians in December, 1923, on "The Relation of the Weakness of the Extraocular Muscles and Depth Perception," based on two groups, one with premature presbyopia and one with normal near vision, I said, "In patients with premature presbyopia: (1) Exophoria combined with good depth perception; (2) Esophoria combined with poor depth perception; (3) Orthophoria combined with good depth perception, except in cases of high latent hyperopia, where the depth perception was poor.

"In patients with normal near vision, as a rule exophoria combined with good depth perception; low degrees of esophoria combined with good depth perception; high degrees of esophoria combined with poor depth perception; right hyperphoria combined with poor depth perception, but hyperphoria and depth perception were improved by correcting lens and prism."

RICHARD G. SCOBEE, M.D., St. Louis, Mo.: May I express my appreciation to the discussers of the paper for their very kind handling of it?

The problem of repeating the test on "abnormals" is, of course, very obvious. We have already tested some 100 patients in clinical practice, and we are beginning work on a separate group who are definitely abnormal, to see

if the test is reproducible there, as it obviously seems to be on normals. Certainly that is an indicated step.

In answer to Dr. Dunnington, it was not intended that the screen-Maddox rod test should replace the screen and parallax test in any sense of the word. The idea was to show that if one takes ten patients and ranks them in order from most to least heterophoria, from 1 up to 10, or vice versa and if one tests those same patients with the screen and parallax test, nine of those patients, roughly speaking, will rank in the same order of degree of heterophoria, and the parallax test will uncover more exophoria than will the Maddox-rod test. Certainly, I would be the last one to detract from the screen and parallax test, which I fully agree with Dr. Dunnington is the most accurate test we have of heterophoria. It was not a substitute recommendation but simply one of comparison, because if some one sends a patient to you and he says, "This patient has so much heterophoria," and you say, "Oh, no, he has so much," you are both perfectly sincere and you are both perfectly right but you are using different tests of heterophoria.

Dr. Gibson made the same point.

The question was raised as to how this work began. We wanted to know why the original limits were selected in the Army Air Forces, as they were, of 5 diopters of exophoria and 10 diopters of esophoria for Class I pilots. There seemed to be no good reason, and certainly I cannot suggest any answer to that. They must have been pulled out of a hat. It would not be surprising to find 6 7/8 as one limit. We hope by a study of the normal to be able then to predicate what might be considered abnormal.

Thank you again for your very kind attention.

# MARGINAL CORNEAL INFILTRATES AND ULCERS

PHILLIPS THYGESON, M.D.
SAN JOSE, CALIF.

CORNEAL ulcers are of two general types, one showing a decided affinity for the margin of the cornea, the other an equally decided affinity for the central or pupillary area. Marginal ulcers occur in relation to conjunctival disease and, in the case of ring ulcers and infiltrates, to systemic disease. The three types, simple catarrhal, ring, and chronic serpiginous (Mooren's), arise from a wide variety of causes and it is often difficult to make an etiologic diagnosis, particularly of the endogenous cases. Central ulcers, on the other hand, are with rare exceptions exogenous, are unrelated to either conjunctival or systemic disease, and are caused by a limited number of readily identifiable microorganisms.

Marginal ulceration is by far the most common corneal disease seen in this country. Of the three types, the ring and chronic serpiginous (Mooren's) ulcers are always potentially dangerous, actual destruction of the cornea not infrequently resulting from them. Simple catarrhal ulcers, on the other hand, are characteristically benign although even in this type severe recurrent lesions are sometimes seen and may cause permanent visual damage due to an extension of cicatrization into the pupillary area or to the development of irregular astigmatism.

The present study was undertaken in an effort to extend the knowledge of the etiology, prophylaxis, and treatment of this common condition. A series of 200 consecutive cases were observed over a period of eight years at the Vanderbilt Clinic, New York, in Army hospitals, and in consultation.

*Methods of study.* A thorough etiologic survey was made of each case. This included careful clinical examination to determine the presence or absence of conjunctivitis or blepharitis and, if present, the clinical type of the related conjunctival or lid margin infection. With only a few exceptions it included exhaustive bacteriologic analysis, not only of the ulcer itself but of any associated conjunctivitis or blepharitis. In suspected endogenous cases a search was made for evidence of systemic infection. Scrapings from the ulcer, and from the conjunctiva and lid margins when these structures were involved, were also subjected to cytologic analysis. Cell studies proved to be of particular value in diagnosing allergic cases. Therapeutic studies consisted primarily in the determination of the effect of sulfonamide and penicillin therapy.

*Clinical Observations.* Simple catarrhal ulcers and infiltrates, usually single and with only two exceptions unilateral, constituted by far the largest group of marginal ulcers in this series. They were almost always associated with a catarrhal conjunctivitis, usually chronic in type but occasionally acute. In this connection it is interesting that in this series only two etiologic types of acute catarrhal conjunctivitis, namely, staphylococcic and Koch-Weeks conjunctivitis, had associated catarrhal ulcers. In the cases associated with chronic staphylococcic conjunctivitis there was usually also a highly characteristic epithelial

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

keratitis exhibiting minute fluorescein-staining punctate erosions and involving especially the lower half of the cornea. In a high proportion of all the simple catarrhal cases there was an associated blepharitis. The ulcers themselves showed a marked tendency to recur but were usually benign. There were two very severe cases, however, with recurrent multiple lesions, which produced permanent visual damage.

The second of the important clinical types of marginal ulcer observed in this series was the ring ulcer and infiltrate. In this type, which was far less benign than the simple catarrhal type, the infiltrate, and eventually the ulcer, formed a continuous ring in the periphery of the cornea. The ring was usually continuous from onset but sometimes developed as a coalescence of discrete infiltrates. In general the conjunctival reaction, except for the circumcorneal injection associated with the corneal reaction, was minimal and the relationship of the ulcer to conjunctivitis, found to be so characteristic of the catarrhal ulcer, was lacking. The two exceptions were ring ulcers which formed in cases of severe acute catarrhal conjunctivitis from a coalescence of numerous marginal ulcers. In contrast to its lack of association with conjunctivitis, the ring ulcer's relation to general disease was striking. Its endogenous origin was obvious in most of the cases of this series.

The third and least common of the clinical types of marginal ulcer seen during the course of this study was the chronic serpiginous (Mooren's) ulcer. Its diagnosis was based entirely upon its clinical appearance since laboratory findings were consistently negative. The 6 cases in this series, which appeared to be typical, were characterized by their chronic course, the undermining of their central border, and the relative absence of conjunctival inflammation. They differed sharply from ring ulcers in their much more chronic course, several months being required for the surrounding of the cornea.

Excluded from this series were 6 cases of marginal ulceration in which the presence of limbal phlyctenules made possible a diagnosis of phlyctenular conjunctivitis. In the absence of limbal phlyctenules a diagnosis of corneal phlyctenulosis cannot be made, however, and it is within the realm of possibility that one or more cases of phlyctenulosis lacking this distinguishing feature may have been included unwittingly in the series. Excluded also were the few cases of ulceration seen in trachoma, the so-called "pannus ulcers." In the writer's experience these have become increasingly rare since the sulfonamide treatment of trachoma has come into general use.

Table I.

BACTERIOLOGIC FINDINGS IN 200 CASES OF MARGINAL ULCERS AND INFILTRATES

| | |
|---|---|
| Catarrhal ulcers: | |
| Normal flora | 16 |
| Pathogenic staphylococci | 133 |
| Diplobacilli | 11 |
| Koch-Weeks bacilli | 8 |
| Coliform bacilli | 3 |
| Pathogenic staphylococci and coliform bacilli | 2 |
| Beta hemolytic streptococci | 2 |
| Streptococcus viridans and coliform bacilli | 2 |
| Non-hemolytic streptococci and coliform bacilli | 2 |
| Pneumococci | 1 |
| Ring ulcers and infiltrates: | |
| Normal flora | 12 |
| Pathogenic staphylococci | 2 |
| Chronic serpiginous (Mooren's) ulcers: | |
| Normal flora | 5 |
| Coliform bacilli | 1 |
| | 200 |

Table II.

CLINICAL TYPES OF MARGINAL ULCERS
ANALYSIS OF 200 CASES

| | |
|---|---|
| Simple catarrhal ulcers: | |
| Secondary to chronic catarrhal conjunctivitis | 156 |
| Secondary to acute catarrhal conjunctivitis | 12 |
| Secondary to endogenous conjunctivitis | 4 |
| Without associated conjunctivitis | 8 |
| Ring ulcers or infiltrates | 14 |
| Chronic serpiginous (Mooren's) ulcers | 6 |
| | 200 |

ETIOLOGY

*Bacteriology of simple catarrhal ulcers.* Table I summarizes the bacteriologic findings in this type of benign marginal ulceration. It will be noted that coagulase-positive staphylococci were by far the most numerous pathogenic organisms found, outnumbering many times over the next most frequent organism, the diplobacillus of Morax-Axenfeld. Next in order of frequency was the Koch-Weeks bacillus with other such pathogenic organisms as coliform bacilli, streptococci, etc., occurring only rarely.

All these organisms could be presumed to be the cause of the chronic conjunctivitis and secondarily of the marginal ulcerations, but actual proof of the relationship could of course not be established in every case. Pathogenic bacteria are occasionally found in small numbers on the normal conjunctiva but in the absence of other causes it seems justifiable to assume that the pathogenic bacteria found in these cases had etiologic significance.

Beta hemolytic streptococci were isolated from 2 cases and pneumococci from one. All 3 were cases of typical chronic catarrhal conjunctivitis with marginal ulceration but were notably lacking in the blepharitis so typical of the staphylococcic and diplobacillary cases. As has already been mentioned, no corneal ulceration or infiltration has been noted in any of the hundreds of cases of acute catarrhal conjunctivitis due to pneumococci which have been observed by the writer.

The origin of the marginal ulcers in cases with a normal flora is difficult to explain. Faulty bacteriologic technic could have been responsible for some of the negative findings but certainly could not have accounted for all of them since repeated cultures were made in most of the bacteriologically negative cases. The possibility that some of the cases may have been phlyctenulosis, with the phlyctenules limited to the cornea only, is worthy of consideration. Endogenous origin is of course a further possibility. It is noteworthy that most of these normal flora cases were in elderly people with arcus senilis and a minimal amount of conjunctival inflammation. One patient, a young woman 25 years of age, had severe seborrheic blepharitis and mild secondary conjunctivitis when the marginal ulceration developed. Blood agar cultures from the conjunctiva and lid margins showed only diphtheroids and white staphylococci but there were numerous budding yeast-like forms identical with Pityrosporum ovale in scrapings from the lid margins. The ulcer healed within a week after treatment of the blepharitis had been inaugurated. As the writer[10] has previously reported in a study of marginal blepharitis, however, catarrhal ulcers are not a characteristic feature of pure seborrheic blepharitis.

Six of the catarrhal ulcer cases were associated with acne rosacea. The ulcers differed in no way from ulcers occurring in pure staphylococcic conjunctivitis and pathogenic staphylococci were recovered from each. The writer agrees with Wise[11] that marginal ulcers in acne rosacea are probably largely staphylococcic in origin.

*Bacteriology of ring ulcers and infiltrates.* As may be seen in Table II, pathogenic bacteria were found in only 2 of the 14 cases of ring ulcer. In both of these the organisms were coagulase-positive Staphylococcus aureus, obviously exogenous since in both cases there was an associated acute conjunctivitis. This finding was sharply in contrast to the general lack of conjunctival reaction in the other 12 cases. Not included in this series was a case seen in consultation which developed ring infiltration after an operation for pterygium. The lesion responded well to penicillin but although it was undoubtedly bacterial in origin culture studies were unfortunately not made prior to the institution of chemotherapy.

The probable systemic origin of the ring ulcer or infiltrate was indicated in at least 11 of the 14 cases although in only one was it possible to demonstrate the relationship beyond doubt. This was the case of a woman 35 years old who had been doing secretarial work in Puerto Rico for several years. On her way to the United States on vacation she developed on shipboard a very severe bacillary dysentery which necessitated hospitalization when she arrived in New York. One week after the onset of the infection bilateral ring infiltrates developed. The infiltration progressed to ulceration in one eye. On sulfonamide therapy the lesions in both eyes disappeared coincidentally with the healing of the dysentery. Five months later there was a recurrence of the dysentery with simultaneous recurrence of the ring infiltrates.

In another patient an interesting relationship between ring ulcers and conjunctival and systemic allergy was demonstrated. The patient was a woman 55 years of age who for many years had had bronchial asthma, hay fever, and various other allergic manifestations. For some months prior to my first observation of her she had had a chronic conjunctivitis and when first seen by me had developed a bilateral ring ulcer, more marked in the left eye. Bacteriologic studies were consistently negative but there was a conjunctival eosinophilia and a constant blood eosinophilia. Over a period of several months the ulcers encroached on the cornea, more rapidly in the left eye, until only a small button of clear cornea remained. During this time the patient's physical condition became gradually worse until death ensued. At autopsy a periarteritis nodosa was found but unfortunately permission for examination of the eyes could not be obtained. Since recent investigations seem to indicate that periarteritis nodosa is allergic in nature, it would seem probable that both the conjunctival and corneal lesions in this case were allergic.

Table III.

ASSOCIATION OF RING ULCERS AND INFILTRATES WITH SYSTEMIC DISEASES

| | |
|---|---|
| Bacillary dysentery | 3 |
| Influenza | 2 |
| Periarteritis nodosa | 2 |
| Scleroderma | 1 |
| Arthritis deformans | 2 |
| Lupus erythematosus | 1 |
| Negative findings | 3 |
| | 14 |

*Bacteriology of chronic serpiginous (Mooren's) ulcer.* The 6 cases of Mooren's ulcer occurring in this series were studied exhaustively for a causal microbe but without success. A few scattered colonies of coliform bacilli were isolated from 1 case on one examination but after a week's treatment with sulfathiazole they could no longer be recovered although the progress of the ulcer had been in no way retarded. In addition to repeated culture studies on various media, under both aerobic and anaerobic conditions, scrapings from the ulcer were subjected to careful microscopic

scrutiny. No micro-organisms of possible etiologic significance were found in either culture or scrapings. In 3 cases an effort was made to determine the presence or absence of a filtrable virus, scrapings being used for inoculation of rabbit corneas, the corneas of rhesus monkeys, the brains of white mice, and the yolk sacs and chorioallantoic membranes of developing chick embryos. Scrapings were also used for intradermal inoculation of the skin in human volunteers and for a microscopic search for the presence of virus inclusion bodies. Unfortunately it was not possible to obtain infection in laboratory animals with ulcer material and all the special virus studies made had to be considered negative.

THERAPY

*Treatment of simple catarrhal ulcers.* With two exceptions the catarrhal ulcers in this series were all benign and healed readily without local cauterization. With the same two exceptions it was unnecessary to atropinize any eyes. Treatment was directed toward the conjunctivitis and blepharitis when present and was varied somewhat according to etiology. For example, in pure diplobacillary infection zinc sulfate solution ¼ per cent was applied to the conjunctiva three times daily and ½ per cent zinc sulfate ointment applied to the conjunctiva and lid margins at night. Since diplobacillary infection almost always induces a blepharitis, the importance of treating the lid margins is apparent. All cases did very well on this regime and there were no recurrences during the period of observation. It should be noted, however, that mixed infection with pathogenic staphylococci occurs not infrequently and that such cases are totally resistant to zinc preparations. Frequent application of sulfathiazole in ointment form was found to be the most satisfactory method of treatment in these mixed cases, penicillin apparently being totally inactive against diplobacillary infection.

Pure staphylococcic marginal ulcers usually responded promptly to treatment of the associated conjunctivitis and usually associated blepharitis. Of the various medications employed three appeared to be better than any others. These were (1) 5 per cent sulfathiazole ointment, (2) penicillin ointment (1000 U/cm.), and (3) a combination of 1:5,000 oxycyanide of mercury solution instilled three or four times daily, with application of a mercurial ointment, such as 2 per cent ammoniated mercury ointment, to the lid margins at night. When it proved impossible to eliminate the staphylococci by local treatment, due in most cases to meibomian gland involvement, staphylococcus toxoid seemed to be of value in preventing recurrences. In this connection the general statement can be made that recurrences were preventable even though the staphylococci could not be eliminated from the lid margins. For example, C. D., a chauffeur, aged 42, had had recurrent marginal ulcers, developing every four or five months, over a period of eight years. Each attack lasted from a week to two weeks and usually incapacitated him from driving. The ulcers had on most occasions been treated by cauterization with tincture of iodine, by antiseptic collyria, and by hot applications. When first observed by me he was suffering from typical staphylococcic blepharoconjunctivitis with a single marginal ulcer in the right eye. Intensive treatment with sulfathiazole and later with mercurial ointments, to which was added a course of staphylococcus toxoid, produced substantial clinical improvement in the conjunctivitis and blepharitis. However, due probably to a chronic meibomitis, it was impossible to eradicate the infection. The patient was seen at six months intervals over a period of three years and each

time was given a "booster" injection of 0.5 cc. staphylococcus toxoid. There were no recurrences of the marginal ulcers during this three year period.

Catarrhal ulcers due to other causes responded well to treatment of the primary conjunctivitis involved and recurrences were not a problem. This was no doubt due to the absence of blepharitis and meibomitis in these cases. Ordinary methods of treatment sufficed, 5 per cent sulfathiazole ointment being particularly valuable, especially in treating Koch-Weeks infections.

Catarrhal ulcers, like phlyctenules, heal spontaneously in from four to fourteen days. *I was unable to see that topical applications to the ulcer shortened its course but appreciable shortening seemed to result from treatment of the conjunctiva and lid margins with the sulfonamides or, in the case of staphylococcic infection, with penicillin.* On the strength of Johnson's and Eckardt's report[6] on the efficacy of riboflavin in the treatment of marginal ulcers in rosacea, this vitamin was used orally in the recommended dosage in the treatment of 18 consecutive cases (9 of them with associated rosacea), but it was impossible to conclude that healing had been noticeably accelerated. Application of vitamin A ointment and large doses of vitamin A by mouth failed also to affect the clinical course of the keratitis. There was no evidence to indicate, as has been suggested, that riboflavin or other vitamin deficiency in patients with catarrhal conjunctivitis predisposed to marginal ulceration.

*Treatment of ring infiltrates and ulcers.* With the exception of the two ring ulcers occurring secondarily to severe staphylococcic conjunctivitis, which responded dramatically to penicillin ointment, local antiseptic treatment did not appear to affect this type of marginal ulceration. Penicillin and sulfathiazole ointments were tried in all the other cases without any evidence of benefit. Oral sulfonamide therapy, however, was strikingly beneficial in the one case of ring ulcer, previously described, which was due to bacillary dysentery, and was highly effective in another case of unknown etiology.

Paracentesis, combined with intravenous typhoid vaccine, was used in the treatment of the 3 cases in which no systemic disease could be demonstrated. The therapeutic effect of this procedure was difficult to evaluate but is believed to have been beneficial, resolution occurring in all 3 cases in periods varying from one to four weeks. Delimiting keratotomy, as advocated by Gifford,[4] was not used in any of the ring ulcer cases. Cauterization of the ulcers was employed in 2 cases but served only to aggravate the condition.

*Treatment of chronic serpiginous (Mooren's) ulcer.* All 6 cases of chronic serpiginous ulcer were given a thorough trial with oral sulfonamide therapy, 2 receiving sulfanilamide, 1 sulfathiazole, and 3 sulfadiazine. In no case was there any clinical improvement. Penicillin was tried in only 1 case, a course of 800,000 Oxford units being administered over an eight day period without effect. Topical sufathiazole and penicillin therapy was also unsuccessful. In view of the poor results with cauterization which have been reported in the literature, this procedure was not tried. At least partial success was obtained by the use of delimiting keratotomy, combined with repeated paracenteses, as recommended by Gifford.[4] As a result of this procedure the ulcers were apparently arrested in 3 cases but in the remaining 3 they progressed centrally and jumped the keratotomy incisions. The keratotomy was performed ahead of the advancing border or borders, according to the technic outlined by Gifford, and the

anterior chamber emptied daily for ten or more days by pressure with a spatula on one edge of the wound.

## DISCUSSION

The overwhelming importance of pathogenic staphylococci as a cause of catarrhal ulcers and infiltrates of the cornea is clearly demonstrated in this series. It is interesting to speculate on the reasons for the consistently benign nature of the catarrhal ulcer. There is some evidence to indicate that this type of ulcer may be caused by toxins rather than by an invasion of organisms as is the case with the central ulcers, and that its toxic origin is in turn responsible for the ulcer's benign character. The susceptibility of rabbit and human corneas to instillations of staphylococcus toxin is well known and was particularly well demonstrated in the studies of Allen.[2] It is of course not possible to state with absolute certainty that invasion of the cornea with staphylococci and other bacteria did not occur in the marginal ulceration of this series as no eyes were removed for section and biopsy was not a feasible procedure. Scrapings consistently showed few or no bacteria, however, in sharp contrast to scrapings from the invasive central ulcers in which numerous bacteria were always demonstrable. This is an important point and should be studied further since the rationale for the cauterization of catarrhal ulcers as commonly practiced is based on the presence of actual bacteria in the ulcers. Gifford[4] was a strong advocate of cauterization but the procedure seems to me to be undesirable in most cases. The recent report by Allen[1] in which staphylococcic ulcers are said to have responded well to antitoxin is of interest in this connection.

The reaction of the cornea to the pneumococcus has a bearing on this problem. The pneumococcus can be extremely destructive to the cornea as the cause of a serpiginous ulcer but when it involves the conjunctiva to produce an acute catarrhal conjunctivitis catarrhal ulcers never result. The writer has never seen a catarrhal ulcer associated with any one of many hundreds of cases of acute conjunctivitis caused by this organism, and there are no reports in the literature to indicate that it has ever been considered a cause of simple marginal ulcers. Although proliferation of pneumococci on corneal epithelium has been demonstrated by Howard,[5] it is known that the cornea is usually not involved in pneumococcic conjunctivitis in contrast to its often serious involvement in staphylococcic conjunctivitis. No exotoxin comparable to staphylococcus toxin has been demonstrated for the pneumococcus and it is entirely possible that this lack of toxin is responsible for the relative inability of the pneumococcus to induce catarrhal ulcers.

From this series it would seem that the other causes of catarrhal ulcer are few in number, the diplobacillus of Morax-Axenfeld being next in importance to the staphylococcus. In the writer's experience the distribution of the diplobacillus is notably spotty in the United States. Found commonly in the southwestern states it is apparently rare in other parts of the country.

Although there were 2 cases of marginal ulcer associated with vernal catarrh in this series, it cannot be said that the ulcers characteristic of this disease are typically marginal, the writer having observed 4 which were central in type. Even these central ulcers were benign in character, however, and entirely unlike typical central ulcers due to the pneumococcus or to the other four principal central ulcer bacteria. Since many cases of vernal catarrh are secondarily infected, the question immediately arises as to whether or not ulcers associated with this condition may be due to secondary

infection rather than to the vernal catarrh itself. The 2 cases of vernal catarrh with marginal ulcers in this series, however, were not secondarily infected and it therefore seems logical to suppose that the ulcers were caused by the vernal catarrh itself. In two central ulcers in vernal catarrh seen recently there was heavy secondary infection with pathogenic staphylococci but the ulcers were extremely superficial and did not behave like infectious ulcers.

In this series there was only one marginal ulcer due to allergy other than the two found in vernal catarrh. This occurred in a case of sensitivity to pontocaine. The ulcer was typically catarrhal in type and healed very shortly after discontinuance of the offending drug. Ulcers and infiltrates secondary to drug allergies are not always marginal, however, as was well demonstrated by a patient with a shallow central ulcer which appeared in the course of an allergic reaction to butyn and healed immediately on withdrawal of the drug. It is of interest that in spite of the numerous cases of conjunctival allergy observed during the time of compiling this series, no other corneal ulcers or infiltrates were noted which could possibly be considered allergic. The reports of O'Brien and Allen[7] and of Parlato[9] indicate, however, that allergy is definitely a factor to be reckoned with in marginal ulceration.

The possible role of bacterial allergy in the production of marginal ulcers is worthy of consideration since it is well known that corneal phlyctenules may simulate catarrhal ulcers and even be mistaken for them in certain instances in which limbal phlyctenules are absent. Since bacterial allergy does not induce a conjunctival eosinophilia, and has no specific diagnostic signs other than the production of conjunctival and limbal phlyctenules, the problem is a difficult one. In favor of an etiologic role for bacterial allergy is the fact that marginal ulcers usually do not appear early in the course of a staphylococcic or diplobacillary conjunctivitis but only after the inflammation has been present some time or after repeated exacerbations. They are extremely rare in children, moreover, and become progressively more common with increasing age. Skin tests with staphylococcic antigens have not proved significant in the writer's hands but the subject of bacterial allergy in staphylococcic infections should certainly be explored more fully. It is worthy of note that in an epidemic of Koch-Weeks conjunctivitis observed by the writer in Florida there were no marginal ulcers among the 30 children seen whereas 3 were observed among the 12 adult cases.

This series would seem to indicate that simple marginal ulcers are usually exogenous but typical catarrhal lesions occurred transiently in 2 cases of endogenous gonorrheal conjunctivitis. Exogenous gonococcal ulcers are usually central but one marginal ulcer occurred in a physician with a severe, accidentally acquired exogenous infection. Curiously enough this ulcer was not typically catarrhal, the ulceration extending directly from the limbus without the customary "lucid" interval of the catarrhal ulcer. In his monograph on endogenous gonorrheal affections, Byers[3] noted that some of the ulcers and infiltrations of endogenous gonorrhea were typically marginal. In the literature there are reports[8] of marginal ulcers occurring in the course of such systemic infections as lymphogranuloma venereum.

The writer has never observed a case of marginal ulceration due to Zur Nedden's bacillus. The distribution of this organism would seem to be extremely limited. So far as could be determined it has never been reported in the United States.

## SUMMARY AND CONCLUSIONS

1. Simple catarrhal ulcers and infiltrates were the most common type of corneal disease observed in this study of 200 cases of marginal ulcers. Although ordinarily benign they were sometimes severe and recurrent, leading to cicatrization extending into the pupillary area or to irregular astigmatism. They were almost always associated with chronic catarrhal conjunctivitis, usually staphylococcic but in a few cases caused by such other agents as the diplobacillus of Morax-Axenfeld, streptococcus nonhemolyticus, and coliform bacilli. Two cases were associated with vernal catarrh and one was an allergic reaction to a local anesthetic. Coincidental blepharitis was an almost constant feature of the staphylococcic and diplobacillary cases. Marginal catarrhal ulcers were also associated with acute catarrhal conjunctivitis caused by Staphylococcus aureus and by the Koch-Weeks bacillus (H. conjunctivitidis). Four cases occurred coincidentally with gonorrheal arthritis and were presumably secondary to metastatic gonorrheal conjunctivitis. Eight cases were secondary to a chronic conjunctivitis for which the causes could not be found.

2. There were 14 examples of ring ulcers and infiltrates in the series and with 2 exceptions they appeared to be endogenous in origin, occurring in association with gonorrheal arthritis, bacillary dysentery, influenza, periarteritis nodosa, and lupus erythematosus. The 2 exceptions were cases of severe staphylococcic conjunctivitis with multiple marginal ulcers which coalesced to form ring ulcers.

3. There were 6 examples of chronic serpiginous (Mooren's) ulcer, all typical in clinical appearance and course. There was no associated conjunctivitis in these cases and, so far as could be determined, no relationship to any systemic infection. Careful laboratory studies of smears, scrapings, and cultures failed to reveal any significant micro-organisms and animal inoculation experiments were all negative.

4. The prophylaxis of recurrent marginal ulcers of the catarrhal type is believed to be entirely a matter of controlling the chronic conjunctivitis. This in turn is largely dependent upon control of the chronic blepharitis, either staphylococcic or diplobacillary, which in most instances was found to be the primary focus for both the conjunctivitis and the keratitis. No information of prophylactic value was obtained in connection with either ring ulcers or chronic serpiginous (Mooren's) ulcers.

5. Treatment of catarrhal marginal infiltrates and ulcers appeared to be entirely a matter of treating the primary conjunctivitis and blepharitis. Except in rare instances local treatment of the ulcers was unnecessary. Staphylococcus toxoid proved to be of value in preventing recurrences of catarrhal ulcers even when cure of the primary conjunctivitis was not obtained. Treatment of ring ulcers and infiltrates was also directed toward the primary cause which was almost always a systemic disease. Local treatment was found to have little value and cauterization of the ulcers seemed only to aggravate them. In the treatment of chronic serpiginous (Mooren's) ulcer the sulfonamides and penicillin were ineffective. Delimiting keratotomy and repeated paracenteses were apparently successful in arresting progression in 3 of the 6 cases.

6. No evidence was obtained to indicate that riboflavin deficiency contributed to the development of marginal ulcers or that treatment with riboflavin was curative.

7. No true example of marginal keratitis due to acne rosacea was seen in this series. There were 9 cases of rosacea with typical catarrhal marginal ulcers but these were believed to be due to secondary staphylococcic infection.

## REFERENCES


1. Allen, J. H.: Antitoxin treatment of staphylococcic corneal ulceration, Tr. Am. Ophth. Soc., 1946. To be published.
2. Allen, J. H.: Staphylococcic conjunctivitis. Experimental reproduction with staphylococcus toxin, Am. J. Ophth., 20:1218, 1939.
3. Byers, W. G. M.: A study of the ocular manifestations of systemic gonorrhoea with reports of cases of this nature, Studies from the Royal Victoria Hospital, Montreal, Vol. 2, No. 2.
4. Gifford, S. R.: Ring ulcer of the cornea, Arch. Ophth., 27:231-241 (Feb.) 1942.
5. Howard, H. J.: Role of the epithelial cell in conjunctival and corneal infections, Am. J. Ophth., 7:909-936 (Dec.) 1924.
6. Johnson, L. V. and Eckardt, R. E.: Rosacea keratitis and conditions with vascularization of cornea treated with riboflavin, Arch. Ophth., 23:899-907 (May) 1940.
   Conners, C. E., Eckardt, R. E. and Johnson, L. V.: Riboflavin for rosacea keratitis, marginal corneal ulcers and catarrhal corneal infiltrates, Arch. Ophth., 29:956-967 (June) 1943.
7. O'Brien, C. S. and Allen, J. H.: Allergic keratoconjunctivitis, Arch. Ophth., 29:600-604 (April) 1943.
8. Oliphant, J. W., Powell, W. F. and Perrin, T. L.: Sulfadiazine in lymphogranuloma venereum ophthalmitis, J.A.M.A., 118:973-974 (March 21) 1942.
9. Parlato, S. J.: Corneal ulcers due to a common allergen, Arch. Ophth., 14:587-590 (Oct.) 1935.
10. Thygeson, P.: Etiology and treatment of blepharitis, Mil. Surgeon, 98:191-203 (March) and 98:279-288 (April) 1946.
11. Wise, G.: Ocular rosacea, Am. J. Ophth., 26:591-609 (June) 1943.


## DISCUSSION

C. L. SCHEPENS, M.D., Brussels, Belgium; I congratulate Dr. Thygeson for his excellent paper. Personally, I learned a lot from it.

The geographical distribution of corneal infections due to Koch-Weeks bacillus is of interest. It was frequent in Western Europe thirty years ago. Nowadays, it has practically disappeared. I believe that at Moorfields Hospital (London), there has not been one case in fifteen years. Personally, I have never seen a case in Brussels. The same remark applies to vernal conjunctivitis, which is frequent in Spain but quite seldom met with in Belgium, Holland or France.

It has been stated that penicillin is not active against the diplobacillus. I believe that some authors found it active (F. A. Juler, London and others). Perhaps more research would have to be done on that particular point.

As regards the vitamin therapy, I have never had any striking results using the vitamin A, $B_1$, $B_2$ or C. I have used the vitamin A locally and in intravenous injections. One has to be cautious with the injections, which sometimes cause diarrhea. I have tried intravenous injections of vitamin $B_2$, following the advice of Williamson-Noble (London), but with no favorable results. Intravenous injections of 500 mg. vitamin C, every other day for about two weeks, appeared more satisfactory.

One point of importance is the advisability of using atropine in every case of corneal involvement. It has been said that it is not necessary to use atropine in a certain number of cases of corneal ulcer. This is in contradiction with the teaching in many European universities. I believe the classical teaching is that atropine should be used when there is corneal involvement. If, in many instances, no atropine is necessary, it means a substantial shortening of the incapacitation. The advisibility of using atropine depends, in many cases, on whether one believes or not that it has a favorable action on the nutrition of the cornea.

I agree with Dr. Thygeson that cauterization is sometimes done much too freely. In stubborn cases of corneal ulcer, it often does more harm than good.

When dealing with a difficult case of marginal ulcer, the following therapeutic means are worth trying, although they are not always successful. Firstly, some marginal ulcerations heal more quickly with eserine than with atropine. Secondly, local applications of ultra violet light may be useful. The effect is enhanced if one per cent fluorescein has been instilled immediately before the light treatment. Thirdly, the local application of beta rays by means of 10 mg. of unscreened radium sometimes gives good results. Fourthly, small doses of x-ray may also be used. One applies locally between one-third and one-fiftieth of an erythema dose, every other day for six to ten days. This method

sometimes gives good results in ulcers following acne rosacea and in Mooren's ulcer.

The general health of the patient has to be cared for and much attention should be paid to his diet and to whether he takes enough fresh air. If the case does not do well, I like to take him in the hospital at an early stage, because general rest is very important. I give the patient milk injections and, if he does not improve, I do a tarsorrhaphy. This, I believe a good operation for every bad case of corneal ulcer. I prefer it to a conjunctival flap, because one does not see what happens under the flap and, in some cases, it remains permanently adherent to the ulcerated cornea and has to be removed surgically, after the ulcer is healed.

I am not very much in favor of paracentesis, unless the tension is high. As a general rule, when there is a bad ulcer, I avoid surgical interference with the cornea proper.

JAMES H. ALLEN, M.D., Iowa City, Iowa: My experience with marginal corneal lesions has been quite similar to that expressed to you by Dr. Thygeson this afternoon. However, there are some points on which we differ, and I should like to talk about a few of those points as well as on some of the points on which we agree.

I do not think that staphylococcic marginal corneal ulcers should be taken lightly for several reasons. One is because I saw an eye destroyed by a lesion which began as a simple staphylococcic marginal ulcer which spread, formed a ring ulcer, produced central necrosis of the cornea with rupture of the cornea and loss of the globe. That occurred in the days before we were using antitoxin and in the days before sulfathiazole and penicillin were available.

However, another reason for not taking these lesions lightly is that they undergo exacerbations even under treatment at times and one must not administer toxoid without caution. If one is dealing with a staphylococcic marginal corneal ulcer it is better to treat with antitoxin until the ulcer is healed before beginning active immunization with toxoid.

I should like to show slides of 2 cases of staphylococcic marginal corneal ulcers. (Slide) Infiltrations are seen at the upper nasal margin. The surface is ulcerated over these infiltrations. This patient was treated with 10,000 units of staphylococcic antitoxin intramuscularly daily for four days, with the result that the ulcerations healed rapidly. She was later immunized with staphylococcus toxoid and no recurrences developed.

(Slide) This is a drawing of a more severe staphylococcic marginal corneal ulceration. If it had extended further it might have produced a ring ulcer. This patient was given 10,000 units of staphylococcic antitoxin daily for four days, with apparent healing, and three days later, toxoid was injected for active immunization, but apparently we began the toxoid too soon, for corneal ulcers recurred, and only after this patient had received a total of 180,000 units of antitoxin did the corneal ulcer remain healed.

There is importance in emphasizing antitoxin treatment, even though penicillin is available. Dr. Thygeson has reported from his experience that 20 per cent of ocular strains of staphylococci do not respond to penicillin therapy. We can support that. However, it is our impression that more than 20 per cent of ocular strains of staphylococci are not affected by penicillin; therefore, antitoxin therapy should be considered if penicillin is not giving results in the treatment of staphylococcic marginal corneal ulceration.

(Slide) This is a marginal corneal ulceration produced by a streptococcus. This was a chronic streptococcic conjunctivitis with marginal corneal ulceration.

(Slide) This is another marginal corneal ulceration, produced by streptococcus viridans. These lesions respond well to penicillin.

(Slide) This is another marginal corneal ulceration associated with streptococcus viridans.

(Slide) This is a hypopyon ulcer, and it is shown at the present time because we recently saw a marginal corneal ulceration with hypopyon from which we isolated pneumococci. It responded well to penicillin.

(Slide) Our experience with contact keratitis has been that the lesion was marginal corneal infiltration rather than marginal corneal ulceration. This patient had a low grade uveitis which was treated with atropine ointment for a long time. She develped an atropine sensitivity, and marginal infiltration in the lower part of the cornea. The uveitis and the marginal corneal infiltration was unilateral, so we had an excellent opportunity to determine certainly whether atropine was the cause of the contact sensitivity. (Slide) This is the opposite eye after several drops of atropine had been instilled and it shows three marginal corneal infiltrations below.

(Slide) This is a marginal corneal infiltration resulting from sensitivity to contact with the oil from an orange peel.

(Slide) This is a drawing of that patient made with a slit-lamp beam to show the location of the marginal corneal infiltration.

We have seen numerous cases of superficial punctate epithelial keratitis as a result of vernal conjunctivitis, particularly of the limbic variety, but we have not seen marginal corneal ulcers or central corneal ulcers with vernal conjunctivitis except in secondarily infected cases. In one case a youngster developed a severe marginal keratitis with ulceration superimposed upon limbic vernal conjunctivitis after he had been vaccinated.

Our experience with acne rosacea has been similar to that of Dr. Thygeson. The ulcerated lesions have always yielded pathogenic organisms.

PHILLIPS THYGESON, M.D., San Jose, Calif.: I wish to thank the discussers for their very valuable comments, and I should like to discuss one or two of them.

Dr. Schepens mentioned atropinization. I have felt that in simple marginal ulcers with little iris irritation the incapacity caused by the atropine more or less outweighed the advantages gained. I have frequently substituted homatropine for atropine in such cases.

Dr. Schepens' experience with the Koch-Weeks' organism in Europe has paralleled ours here. Koch-Weeks in conjunctivitis in epidemic form in my experience has only been encountered in Florida and California. I actually saw no case of this infection in New York in a period of about six years, although Dr. Weeks made his original studies in the organism there.

I shall certainly try the use of x-rays and radium in Mooren's ulcer in view of my only partial success with other methods.

Time prevented me from discussing shock therapy in ulcers. It proved of definite value in my series of ring ulcers.

Dr. Allen's work on staphylococcic antitoxin in staphylococcic ulcers is certainly outstanding. It is true that penicillin-fast organisms are going to become more frequent, and whereas new antibiotics may be developed, it is certain that there will always be cases in which a staphylococcic antitoxin will be of great value.

# A METHOD OF CLOSING THE CATARACT INCISION BY COMBINING A CORNEOSCLERAL SUTURE AND A LARGE SLIDING CONJUNCTIVAL FLAP

P. H. DECKER, M.D.
WILLIAMSPORT, PA.

SUCCESSFUL removal of a cataract within its capsule requires a full half limbus incision. This relatively wide incision in turn requires surgical closure with sutures.

Two factors are of equal importance in this closure:

1. To secure adequate STRENGTH in closure of incision.

2. To secure adequate SEALING of incision.

The first, or STRENGTH factor, is best secured by some type of corneoscleral suture. The second, or SEALING factor, is best secured by a large sliding conjunctival flap. This flap, drawn down from above, temporarily buries the corneoscleral suture and also covers the entire incision line. Thus, only by COMBINING a corneoscleral suture with a large sliding conjunctival flap is completely adequate surgical closure secured.

Adequate STRENGTH of incision closure, obtained only by some type of well-placed corneoscleral suture, is the best insurance against vitreous loss. This applies to vitreous loss at the time of operation or later during convalescence, and is of such obvious importance that no more need be said concerning this complication.

Adequate SEALING of the incision, obtained only by a large sliding conjunctival flap, while adding but little to the STRENGTH of the closure, is the best insurance against DELAYED restoration of the anterior chamber. This delay in the reforming of the anterior chamber is not infrequent, and can result in many unpleasant and serious consequences.

Postoperative complications attributable to delay in reformation of the anterior chamber include such situations as—prolapse of the iris, hemorrhage from iris, infection of the open incision, intraocular infection, iridocyclitis, and degenerative changes of the cornea or vitreous with subsequent opacity of these structures. Secondary glaucoma may follow occlusion of the drainage angle by anterior synechia forming at the iris base, or by epithelial ingrowth and proliferation through the delayed closure of the incision.

Some modification of the following technique may suit you better. It does not matter just how this procedure is done provided both required features of closure, STRENGTH and SEALING, are accomplished. Any type of well-placed corneoscleral suture will do, provided it is followed with a sufficiently large sliding conjunctival flap to cover the entire incision line. However, the principle of burying both a well-placed corneoscleral suture and the entire incision line with the sliding conjunctival flap must be carried out. Neither procedure alone is sufficient: COMBINING them gives adequate surgical closure. During the past two years I have performed operations on over 125 cases by this method and now use the following technique:

Starting as in a simple enucleation the limbal conjunctiva is circumcised as

Presented as a *Motion Picture* at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

close to the cornea as possible, leaving no epithelial tags attached to the edge of the cornea to get caught later and implanted in the incision. This circumcision is done throughout the upper three-fifths of limbus. The conjunctiva is now undermined far back so no tension will be present when the flap is later slid down over the corneoscleral suture and the entire incision line. After the flap is thus prepared it spontaneously retracts out of the way and allows an easy opportunity to place the simple corneoscleral suture.

Using a Davis-Geck double armed black silk suture No. Seven-0 with an atraumatic needle, a very simple horizontal corneoscleral suture is placed, one bite in the cornea and one in the sclera. Firm corneal and scleral fixation is obtained by using a Burch double-pointed corneal pic. This firm fixation makes the placing of the corneoscleral suture relatively easy. It is important that these parallel bites be spaced EXACTLY opposite each other laterally, that is in the same vertical meridian, to avoid torsion of the lips of the incision in closure. When the needle has entered for the scleral bite and before the needle is entirely through, if it is found that the bite is not EXACTLY opposite the corneal bite, the needle must be withdrawn and reinstated so that it will be EXACTLY opposite the corneal bite. Needless to say both the placing of the corneoscleral suture and the tying of the corneoscleral suture should be done under the best of visual conditions, preferably under loupe inspection.

FIG. 1.—The box-like corneoscleral suture lying across the incision line shows dimly beneath the large conjunctival flap. This flap, temporarily burying the corneoscleral suture, covers and seals the entire incision line.

With the corneoscleral suture accurately placed, and the suture loops laid well back out of the way, a full half limbus incision is made with a Graefe knife—the knife emerging between the corneal bite and the scleral bite of the corneoscleral suture; these two bites having been placed about 2 mm. apart. For those who wish to use a keratome incision with lateral scissors enlargement this substitution could be made without any change in technique, and with probably slightly less danger of cutting the suture.

After an iridectomy, either peripheral or complete, and after the lens has been extracted, the iris pillars may be replaced either before or after the corneoscleral suture is tied, depending somewhat upon the apparent need for haste in tying the corneoscleral suture. This suture should be tied with a simple square knot and not too much tension used to avoid wrinkling or inverting of the edges of the incision. A tension knot should be avoided as it is apt to invert the edges. The suture, when properly placed and tied, now, appears as a box-like or mattress-like square suture entirely without, and lying across the surface of the incision, rather than incorporated within any portion of the lips of the incision. Fairly long ends are

left on the suture, approximately 2 to 3 mm. This will assist in its removal at a later date.

The conjunctival flap is now ready to be slid down and, for this purpose, it is both convenient and economical to use the discarded ends of the double armed suture which has already served for the corneal suture, each end being now armed with a single needle. These lateral conjunctival sutures are inserted at approximately 1:30 and 4:00 o'clock on the limbus and 10:30 and 8:00 o'clock on the limbus. Slight variation in the position of these sutures may be used, or they may be removed and re-inserted, or additional sutures may be used in order to get adequate coverage of the incision line. If the flap seems a little tense in any area it may be snipped slightly in the center with scissors to relieve any undue tension on the cornea. Usually two simple lateral sutures will suffice to give adequate incision coverage. In tying the conjunctival sutures the first knot of each suture should be a tension knot. Otherwise there is a tendency, due to the retracting of the flap, for the first knot to become partly untied before the second knot can be placed. These conjunctival sutures cut out spontaneously about the fourth or fifth day allowing the conjunctival flap to automatically retract upward and expose, or at least partly expose, the corneoscleral suture.

The corneoscleral suture is not removed until about the twelfth day thus insuring against any accidental opening of the incision during its removal. If the suture is not completely exposed by the spontaneous upward retraction of the conjunctival flap it may easily be exposed fully by teasing upward the still slightly loosened edge of the conjunctival flap. This upward teasing of the edge of the conjunctiva may best be done by a cotton applicator saturated with cocaine and adrenalin. This gives a well-anaesthetized and bloodless field for the removal of the suture. If the suture is still slightly buried and does not readily present itself for a scissors removal, a very easy way to remove it is to slide the point of a cataract knife, cutting edge out, under one of its loops. This procedure should be done under loupe inspection thus guaranteeing against any trauma in its removal.

If by accident the corneoscleral suture should be cut at the time of making the incision, or if the corneoscleral suture should break during the process of tying it, then no attempt should be made to slide down the conjunctival flap without first reinserting and tying down a new corneoscleral suture; otherwise the conjunctival flap, when sliding downward, will likely catch in the edge of the only partly closed incision and may result in unexpected complications. Under such circumstances the wound is probably already partly gaping, and, any attempt to slide down the conjunctival flap without the corneoscleral suture in place will simply make matters worse. Rather than attempt to do this it would be safer to close the eye at once without further manipulation.

The use of a sliding conjunctival flap alone to close the cataract incision, as suggested originally by Kuhnt, has largely been discarded. I believe that the reason this method is not more generally used today is that it is difficult to accomplish and is actually an unsafe procedure unless the lips of the incision are FIRST securely closed with a corneoscleral suture. A sliding conjunctival flap, if used without a primarily placed corneoscleral suture, must be pulled down simultaneously on both sides. This is a bothersome procedure and requires trained assistance. Such is not the case when a corneoscleral suture is first used to give a firm smooth non-buckling base over which the conjunctival flap slides easily and smoothly without any catch-

ing in the wound or tendency of the corneal lip to buckle. This tendency of the cornea to buckle is a definite danger when the corneoscleral suture is not used first.

I wish—at the danger of repetition—to emphasize the fact that if this method of closing the cataract incision has any merit, and I believe it has, it is because of the COMBININING of two older procedures, namely, a sliding conjunctival flap and a corneoscleral suture.

Routine therapeutic procedure consists of: three grains of phenobarbital several hours prior to operation, one drop of one per cent silver nitrate in eye several hours prior to operation, blocking of the seventh nerve, retrobulbar ciliary novocain block with one or two minims of adrenalin added if the patient's blood pressure is within normal limits, cocaine and adrenalin surface anaesthesia, homatropine at beginning of preparation. If capsule has been delivered without rupture, eserine is used; if capsule has ruptured and any cortex remains, atropine is substituted for eserine. White's ointment and routine dressing complete the procedure.

Patients upon whom this method of cataract closure has been used may with safety be allowed a great deal more freedom than with former methods of closure. I have no hesitancy in allowing them to turn upon either side within six hours after operation. If necessary, and conditions indicate, they may get out of bed safely on the second or third day, although this is not a routine procedure. Such increase of postoperative freedom and mobility is of course in direct conformity to the new era of ambulation being allowed all surgical patients, particularly elderly surgical patients.

In a series of 100 consecutive cases upon whom this type of closure was done all but 4 are able to read ordinary newspaper print as a final visual result. There were no eyes lost. Postoperative astigmatism has been reduced by an average of one or more diopters over previous results. There is more postoperative redness present in these cases for a few weeks than in cases where no large conjunctival flap has been used, however, this redness is not true pathologic redness and has no apparent clinical significance. Of the above 100 cases all but 3 had good anterior chambers present at the first dressing on the third day. The delaying factor in one of these three was a very slight incarceration of one iris pillar. One patient became confused, got out of bed, and removed all his dressings three hours after operation. Upon examination he was found to have a well formed anterior chamber.

In conclusion this procedure adds no more than five minutes to the time required for the usual cataract operation. Most of the manipulation is done prior to the section when the saving of a few minutes of time is of no importance. It adds no risk to the operation, and, at least in my hands, has materially lessened complications and given better end results.

# In Memoriam

George Viven Allen ..... Topeka, Kansas

Frederick James Bentley ..... Seattle, Washington
January 1, 1947

Howard S. Clark ..... Minneapolis, Minnesota
May 9, 1946

Louis Samuels Deitchman ..... Youngstown, Ohio
September 4, 1946

John Marvin Ingersoll ..... Miami, Florida
January 16, 1947

George Baigerie Jobson ..... Franklin, Pennsylvania
October 21, 1946

William Edward Patterson ..... Minneapolis, Minnesota
October 30, 1946

Charles Tiffany St. Clair ..... Bluefield, West Virginia
November 17, 1946

O. W. Sedgwick ..... Ionia, Michigan

Wayne Pulley Sirles ..... Herrin, Illinois
December 11, 1946

Carl M. Sneed ..... Columbia, Missouri
November 6, 1946

# In Memoriam

JOHN MARVIN INGERSOLL, M.D.
1869 - 1947

## JOHN MARVIN INGERSOLL, M.D.

### 1869 - 1947

*We record with deep regret the death of Dr. John Marvin Ingersoll on January 16, 1947, at his home on South Bayshore Lane, Miami, Fla. Dr. Ingersoll passed away after a long illness at the age of 77.*

*Dr. Ingersoll was born in 1869 in Youngstown, Ohio, and was graduated from Western Reserve University in 1893. He did his postgraduate work in Europe and studied under most of the great masters of otolaryngology of his day. He then returned to Cleveland, Ohio and became Professor of Otolaryngology at Western Reserve University, a post he filled until 1926 when he resigned and retired to live in South Florida. He was appointed Emeritus Professor of Otolaryngology in 1926.*

*Dr. Ingersoll leaves his wife, Mrs. Nora Ingersoll, a son and a danghter.*

*Always one of the leaders of his specialty, he took great interest in developing younger men and many of the well known nose and throat men of the United States had their training under Dr. Ingersoll. Among the many honors that were bestowed upon him were the presidency of two great societies in his specialty. He was president of the American Academy of Ophthalmology and Otolaryngology in 1919, president of the American Laryngological, Rhinological and Otological Society and secretary of the American Laryngological Association. He was a fellow of the American College of Surgeons and a member of the American Otological Society.*

*During his active clinical life he was interested in all phases of the specialty but he gained national renown for his research studies in a comparative anatomy of the ear, nose and throat. His dissections of the heads of vast numbers of animals marked him as a pioneer in comparative anatomy.*

*During World War I, Dr. Ingersoll left his professorship at Western Reserve and entered the Medical Reserve Corps of the United States Army. He served throughout the war in charge of some of the largest ear, nose and throat services in the Army. It was his request that he be buried in Arlington Cemetery.*

*Dr. Ingersoll left a host of friends and admirers, both among the medical profession and the laity. Those of us who were fortunate enough to have been favored by his friendship will be forever thankful for his guidance. As a medical educator he was meticulous, exacting and demanding, but at the same time he was generous and ever encouraging to the young medical men. The medical profession had lost a very great and brilliant member.*

TRANSACTIONS
American Academy of Ophthalmology and Otolaryngology
*Official Publication of the American Academy of Ophthalmology and Otolaryngology*
Published under the direction of the Editorial Board
WILLIAM L. BENEDICT, Rochester, Minnesota, *Editor*
W. HOWARD MORRISON, Omaha, Nebraska, *Associate Editor*
HENRY L. WILLIAMS, Rochester, Minnesota, *Associate Editor*
Printed by Douglas Printing Company, 108 North 18th Street, Omaha 2, Nebraska

All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, the American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

# Editorial

## Home Study Courses

Home Study courses were authorized by action of the Council in October, 1938, when the Activities Committee reported as follows: "Recommendation to the Council: Based upon motions from the Teachers' Section . . . 2. Academy to appoint a committee of two to work out a method of Home Study in Fundamentals for ophthalmic and otolaryngic residents and such prospective candidates as may be recommended by the American Board of Ophthalmology and the American Board of Otolaryngology. Executive Secretary to select one or two to work out plans and report to Program Committee. Passed."

The Council Minutes for 1938 include the statement that the Home Study Course has been "accepted wholeheartedly by American Board of Ophthalmology—syllabus basis of Home Study Course considered by Board of Otolaryngology."

Action on development of the Home Study Course was deferred because Dr. Wherry desired approval of the Council on Medical Education and Committee on Graduate Education. Dr. Harry S. Gradle urged that the courses be begun immediately and proposed the offering of a supervised list of reading to fill in gap between "now and when institutions themselves will fulfill their obligations."

In 1939 the Activities Committee of the Council recommended that a course of home study for residents and others be sponsored by the Academy. The proposal was approved by the Council.

Development of the Home Study Courses was assigned to Dr. Harry S. Gradle for ophthalmology and Dr. Frank J. Novak, Jr., for otolaryngology. Assistant to Dr. Gradle in ophthalmology was Dr. Daniel Snydacker and assisting Dr. Novak in otolaryngology was first, Dr. Jack Allen Weiss; since 1943, Dr. Benno L. Bernheimer.

The Council, realizing the importance of the Home Study Courses, determined that the courses should be under the management of a single director. To

carry out the purposes of the Council, Dr. Gradle was made Secretary for Home Study Courses and thereby became a member of the Council in 1943. The activities of the Home Study Courses have since been carried on under his direction with the assistance of a faculty selected from the membership of the Academy.

While the Home Study Courses of directed reading are designed for residents and interns, many men in private practice find the courses helpful and are to be counted among the subscribers. Acceptable residencies in hospitals supply specified books and journals for use by the Home Study Course students. Certain textbooks are out of date or out of print and are expensive. To supplement the list of books required for the Home Study Courses the Academy has sponsored publication of a number of manuals prepared especially for students of the Home Study Courses. Two such manuals are complete. Four other manuals are now in process of compilation and others have been proposed.

The Home Study faculty at present is comprised of 16 teachers. The courses for 1946-47 began in September and will continue through May. There are 522 subscribers. With return of medical personnel from the services grading of all papers has been resumed. During the war years, due to lack of personnel, only 50 papers in each specialty were graded by the faculty. The remainder corrected their own papers by comparison with the set of model answers prepared by the faculty.

The development of Home Study Courses is the largest single project facing the Academy today. This adventure in extramural education has been in operation long enough to prove its value. It has the support of the specialty boards where the value of the courses has been thoroughly tested. Good as they have been, there is room for improvement. Consideration must soon be given to the development of clinical courses to supplement the fundamental courses.

Early in 1946, due to Dr. Gradle's illness, Dr. Lawrence R. Boies of Minneapolis was named acting secretary for Home Study Courses and at the October meeting he was elected Secretary. The office was moved from Chicago to Rochester and consolidated with the office of the Executive Secretary. Dr. Boies has associated with him, Dr. Hendrie W. Grant who will supervise the ophthalmologic course and Dr. Robert E. Priest who will assist him in the course in otolaryngology.

## Notice

Pending revision of the Atlases of Ophthalmic and Otolaryngic Pathology a new printing of both has been completed. The new Atlases have library bindings instead of the old plastic ones. Under the new arrangements of the Registry, the National Academy of Sciences handles all the funds, and the American Academy of Ophthalmology and Otolaryngology has approved the recommendations of the Registry Committee that rental fees for sets of slides be charged to civilian personnel and that the sale of the Atlases be handled directly by the Registry Office at the Army Institute of Pathology, Washington 25, D. C. Applications, therefore, need no longer be sent to the Secretary of the Academy. The price for Atlases remains $5.00 each, and checks for purchases and deposits on slides should be made payable to the National Academy of Sciences.

## Letter to the Editor

Dear Dr. Benedict:

The medical student of today carries the heaviest load of study in the history of the profession. Before he can qualify to practice a specialty he has to devote

three or four years to premedical studies, then four years in medical school, one or two years as an intern and from three to five years as a resident, a total of from eleven to fifteen years. During this time he has ranged through science, philosophy, art, and economics, and has been saturated with information on many subjects having only the remotest connection with the practice of medicine, all with the idea of giving him a cultural foundation that will stamp him as an intellectual as well as a practitioner. So he steps out into the world loaded to the guards with information on comparative anatomy, biochemistry, physics, natural philosophy, social economics, embryology, to say nothing of the practical things like symptomatology and therapy. Since he has been told it is important that he keep abreast of medical progress by close attention to the literature, he spends his spare time (of which he has plenty in the beginning) reading magazines, literature from pharmaceutical houses and perhaps other material available in the libraries. He comes upon a reference to an article in a journal emanating from South America or Germany or France and finds all of a sudden that he is blocked by his inability to read the foreign language in which it is printed. Eleven to fifteen years of preparation for his glorious career and yet he is stumped at the very gateway of knowledge! He is a cripple who has to stumble along with such help as he may obtain from an abstract of the original or is forced to forego reading the article altogether unless he is lucky enough to have a friend or acquaintance who can read it to him or translate it. Since this involves too much time and effort, it is usually passed up. As a result his research insofar as it involves foreign literature is reduced to a minimum and he loses such knowledge and inspiration as this avenue might have afforded him.

Is it unreasonable therefore, to inquire why no emphasis is placed on the study of foreign languages in the curriculum of the medical student? Is it asking too much of the Universities who plan these elaborate curriculums covering every conceivable subject that might possibly contribute to the student's efficiency to include at least one foreign language? Compare our attitude in this respect with that of the foreign schools. My personal experience with physicians in Germany, France, Austria and many of the Latin American countries has convinced me of their superiority in this respect since practically every one of them not only reads English and German, but is able to speak it to a greater or less degree. Since we have been doing many things to foster closer relations with our Latin American neighbors, we might at least attempt to begin at the beginning by trying to learn their language and by making it compulsory for our students to learn how to read it. We realize how difficult it is for the older men to undertake the study of a foreign language for obvious reasons but there is no reason why young men who are devoting all of their time to study and are at the age when learning comes easier, should not be compelled to acquire something that will not only be profitable but will contribute a great deal of enjoyment to their studies.

(Signed) SAMUEL SALINGER, M.D.

# THE OPHTHALMOLOGIST AND THE INDUSTRIAL MEDICAL DIRECTOR

JEAN SPENCER FELTON, M.D.
OAK RIDGE, TENN.

LIVING as we are in the wake of an unprecedented world conflict, we find our industrial civilization attempting to embody into its methodology of manufacture the good features of wartime production. Comparably, in this transitional period, medicine in industry is building into its peacetime program those proven ways of bettering the worker's health. The war years saw the growth of the importance of optimum vision in the pattern of production; and the medical director in industry, desirous of having the richness of a full preventive medicine program, is carrying vision conservation over in its entirety.

Unfortunately, this utilization of visual knowledge is not always possible in the present day plant. The services of a specialist skilled in the application of this definitive information are not available because of current professional misunderstandings. It is the hope of those engaged in exercising industrial medical care, to demonstrate the advantages accruing to the ophthalmologist from participation in this program, and to underline the needs for his entry into the field — an entry which is deemed mandatory by the far-seeing medical director. There has been a distinct failure of these two individuals to enter into a joint effort whereby the worker could benefit from this fusion. The situation is ripe for this *combined-operation* for during this period of conversion, sufficient time may be taken for accurate planning without management, industrial medicine or ophthalmology having to plunge into a mêlée of confusion, thwarted effort, or misunderstanding of purpose.

The ophthalmologist has refrained, in the main, from entering the industrial field for several reasons. He knows little of the workings of industry, he has no appreciation of the mission of the medical director, he does not know of the many facets of industrial activity that are crying for his help, and what is of the greatest importance, he has grave misapprehensions as to the ethical status of such a step into industry. This discussion is predicated on the belief that there is an unequalled opportunity for the ophthalmologist in the industrial scene if we, who execute the in-plant medical care program, can demonstrate the many needs for his services. Management has shown its desire for such services; in fact, for any measure that will, as noted by Tiffin,[1] effect "hourly production, proportion of work rejected for defective workmanship, supervisors' ratings of employees, employee absences, rate of labor turnover, or some other measurable aspect of employee value to the company."

As has been described in a previous communication[2] the worker is seen in three phases by the industrial health service, as shown in figure 1. This same approach can be applied to the sphere of interest of the consulting ophthalmologist.

---

The following papers by Dr. Felton, Mrs. Leedke and Dr. Marshall were presented at the meeting of the Committee of Industrial Ophthalmology, October 13, 1946, Chicago, Ill.

Supt., Health Department, Monsanto Chemical Co., Clinton Laboratories, Oak Ridge, Tenn.

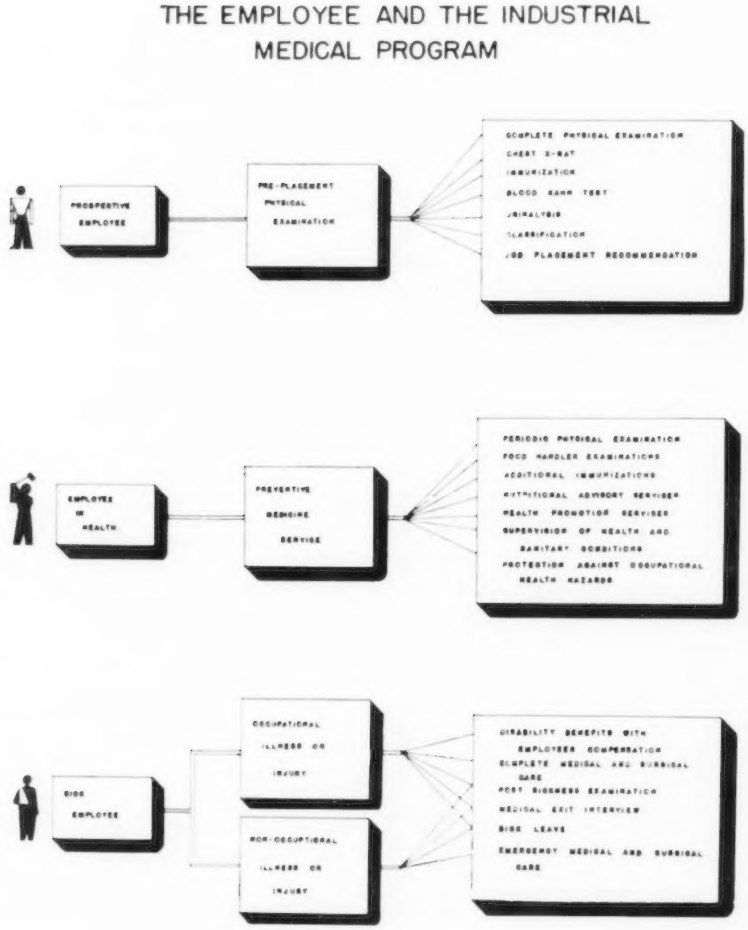



FIG. 1

THE PROSPECTIVE WORKER

Each individual applying for a position in the large plants undergoes a complete physical appraisal consisting of: a. physical examination; b. diagnostic laboratory procedures — chest film, hematologic examination, serodiagnostic test, and urinalysis; c. immunizations; d. classification according to local rating scale; and e. matching against the prospective job. As part of this pre-placement survey is the vision screening procedure, which in the more progressive plants, consists of a battery of tests supplied by a single screening or rating instrument. This is a far cry from the reading of a Snellen chart, badly illuminated, badly worn, badly distanced and badly interpreted. The basic tests (figure 2) offered to the new employee consist of a series well known to most of you, and of vital importance in industry as they help determine the worker's ability to meet the job's physical demands.

ESSENTIAL DATA IN DETERMINATION OF THE EMPLOYEE'S VISUAL SKILLS

1. Visual acuity
   a. Monocular and binocular
   b. Distant and near
2. Stereopsis
3. Color perception—appreciation
4. Muscle balance
   a. Distant and near

FIG. 2

The results from these procedures are then, in a well integrated program, matched against the visual requirements of the job. If the applicant fails to meet these, his visual skills are utilized in that occupation where they will serve him and management in their greatest capacity. It is in this role that the ophthalmologist plays so keen a part, by interpreting from these tests the visual skills of the applicant. In large plants he interprets the findings of testing done by nontechnical personnel, or in small organizations will do the examining himself. He must take these data, and in co-operation with the medical and personnel directors place that man in a job — actually fitting the eyes to the job.

In order to apply the preplacement examination findings, a detailed knowledge of the job is a must. Such information is derived from a visual analysis of the occupation, and this includes the factors seen in figure 3.

ELEMENTS OF A VISUAL JOB ANALYSIS[3]

1. *Description* of job.
2. *Working distances* — from eye to site of operation; fixed or changing.
3. *Size of central working area*: factors of depth perception.
4. *Type of visual attention required*: fixed or changing; casual or concentrated; detailed or gross.
5. *Work motion*: *slow or rapid; rotating,* vertical, or horizontal; fixed or intermittent.
6. *Footcandles of illumination at work point.* Footcandles in surrounding area. Direction of light. Glares.
7. *Type of working surface.* Glossy, nonglossy, uneven. Angle of working surface. Work position. Worker look up, down or ahead.
8. *Colors* to be perceived and discriminated.
9. *Eye hazards*: flying objects, particles or dust, fumes; splashing chemicals or molten metals; intense heat or light.
10. *Eye protection* required.

FIG. 3

These data, or visual skills demanded of the worker, are written into the job requirements and meet with eventual tabulation.

This visual survey must be accomplished by the ophthalmologist so that he can get into the shops, know the jobs intimately, learn the shop language and be completely familiar with the workers' daily environment. From this point on, he is of immeasurable aid to the medical director and the personnel director who are trying to place a certain man in a certain job, where full utilization of his capacities will be attained.

With our continuing industrialization, additional factors come onto the scene. The jobs, as they are now encountered, demand more and more exceptional visual skills, and more and more close work, for man now controls machinery for work he previously did by hand. Dials are watched, intricate mechanisms are assembled, and with 2 per cent of our male population color blind, 400,000 employees amblyopic in one eye, with acuity less than 20/40 in the poorer eye, and 20 per cent of employees demonstrating defects requiring examination and correction, a careful visual screening is mandatory. This is of unusual importance in such an establishment as that with which the writer is connected —the research center of the atomic energy program. There chemists, physicists, and chemical engineers come together where their brainpower is needed so vitally, and where limited visual capacities must come along with them. They must be protected against their jobs and the jobs against them, and it

is in this situation where a true evaluation of their vision is mandatory. One does not disqualify a top-drawer scientist because of limited vision—one utilizes him, and assists him in adapting to the work through a careful visual appraisal and follow-up reinventory.

From material gained at the time of the visual analysis the eye protection required by the job is provided the worker. This is in the form of safety glasses, that carry correcting lenses if needed, and offer protection against impact through use of case hardened glass. Through a single implementing device to his occupation, a two-fold result is obtained, specifically, good working vision and eye protection. To prescribe the proper lenses, a knowledge of the job is demanded, for occupational glasses carry with them a visual potential based on the *working distance* and a safety defense determined by the hazards characteristic of the job.

### THE EMPLOYEE IN HEALTH

Once the applicant has been added to the regular employee roster, follow-up and survey studies are initiated shortly thereafter. The astute ophthalmologist will seek out instantly those employees who have been in the plant years before a progressive eye program had been established. These workers, representing many man-years of skilled craft performance, must be studied to detect the concomitants of the aging process—glaucoma, degenerative diseases of the retina, lenticular changes, and early fundal evidence of hypertension. To these workers, most of whom do not deteriorate in their work skills with age, an eye study or visual appraisal is "brand" new. The multi-procedured examination of the ophthalmologist is many light years removed from the examining process of the "company doctor," and is a valuable adjunct of health education.

Morbid conditions of the eye that are detected can be followed for further change, can be corrected if remedial, or recommendations can be made to management for a change in the employee's job where he can fit his altered capacity to greater advantage to the company and to himself. Rehabilitation of the limited worker is a phase of industrial health to which the oculist can contribute so much. The institution of orthoptic exercises, the advice relative needed changes in lenses, the recommendation for corrective surgery—all such measures stem from the eye specialist intent upon his function in the industrial medical community of reinstating the worker in society as a fit member, whose effort contributes to the advance of our civilization. In addition, the individual is made to feel his usefulness, and a happy, well adjusted worker results.

The broad fields of industry carry with them known and unknown hazards to the worker and the ophthalmologist must know the toxic substances to which the employee is exposed, and the effects they produce. In this we think of contact dermatitis of the lids, the cataracts of glass blowers, the effects of ultraviolet radiation, and others seen in figure 4.

EXAMPLES OF INDUSTRIAL EYE IRRITANTS

| SUBSTANCES | EFFECTS |
|---|---|
| Amyl nitrate | Corneal necrosis |
| Analine dyes | Keratoconjunctivitis and chemosis |
| Beryllium | Keratitis |
| Carbon disulphide | Cataract |
| Lead | Optic neuritis |
| Methyl acetate | Loss of vision (central) |
| Tar | Corneal ulcers |
| Trichlorethylene | Keratitis—amaurosis—optic atrophy |

FIG. 4

Research is needed to determine the potential damage existing in new, and as yet untried, chemicals. The oculist should know the materials used in his plant,

their toxicity, and the methods of exposure control used to combat their harmful effects. Is eye coverage adequate? Should local exhaust ventilation be installed? Is remote control indicated? Can nontoxic materials be substituted? Should air supplied hoods be used? These are questions to be answered by the oculist in co-operation with the industrial hygiene engineer, the safety director, and the medical director.

Visual health is maintained further by the oculist in his studies of illumination, machinery and desk placement, color as it applies to the equipment used, and the surrounding environs. Through the elimination of glare, mal-placed and maldesigned dials, color clashes, poor contrasts for night work, and other sources of fatigue, the worker is kept in optimal visual health. These factors affect the individual, but collectively, the other danger source is the ever potential epidemic keratoconjunctivitis, for which the observer must be on the alert.

Health promotion, keynoting every industrial medical service, is another function of the consulting ophthalmologist, for no greater opportunity exists for the extrapolation of ideal visual health behavior into the worker's home. The use of "dime store" glasses, the necessity for frequent examinations, the use of the common goggles—all these items demand patient talking, as can be done only by a specialist equipped with this information. By further extension of thought, the worker and his family will learn of the wisdom of squint correction, the contagion of conjunctivitis, and the dangers of sling-shots, rocks, etc. These many opportunities are afforded the oculist in industry—opportunities not encountered elsewhere, or of so much advantage to the patient, the family, the industry and the ophthalmologist.

### THE SICK EMPLOYEE

Each case of permanent total disability resulting from an occupational eye injury cost $328.00 in compensation and every temporarily disabled worker from a similar injury received $28.00, as noted in the most current compensation studies.[4] With these figures one can see that the national program of eye care has not been working successfully. These injuries are continuing to the score of over 16,000 yearly. This means that the position of the oculist in industry remains unchallenged from the point of view of his having a burdening case load of injured employees.

The causes of eye injuries in order of frequency may be seen in figure 5.

CAUSES OF EYE INJURIES

1. Flying objects—especially those set in motion by hand tools
2. Abrasive wheels—small flying particles
3. Corrosive substances
4. Injurious light or heat rays
5. Splashing metal
6. Poisonous dusts or fumes

FIG. 5

These cases, apart from minor eye injuries are usually referred to the ophthalmologist by the medical director, and it is this situation that points up or epitomizes this discussion.

In the average industrial community there is one of you who serves as consulting ophthalmologist to the local industries. Heretofore, his main function has been to care for the eye injuries delineated above, and practically never has he been at the plantsite from which industry he draws his patients. Therein lies the opportunity for the ophthalmologist to acquaint himself with the workings of an industrial organization. This is mandatory, for in the near future management will demand more of the eye program than it is getting now in a care-for-injury program solely. It is up to the ophthalmologist to make the approach to the plant. How is it to be

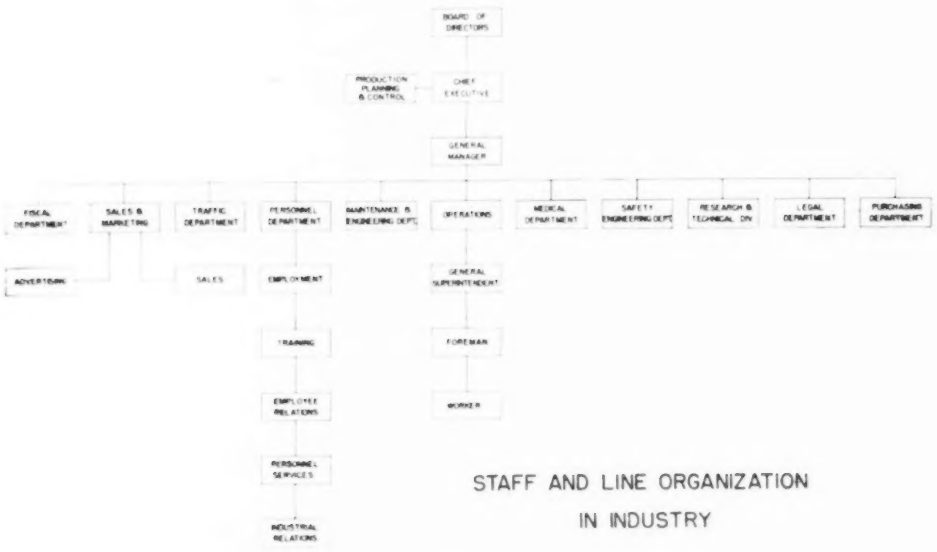



FIG. 6

done? In figure 6 we see the line and staff organization as customarily seen in the larger industries.

Ordinarily, the medical director reports directly to top management, except for certain plants where organization positions differ, and reporting is accomplished through the personnel, industrial relations or safety departments. In any event in these installations where a full-time medical director does exist, the initial liaison is effected through his office. The establishing of a full eye conservation program can be authorized by management, and management should be appraised of the values of such an effort through the combined recommendations of the consulting ophthalmologist and the medical director. The medical director will aid the initiation of such a program if prepared by the oculist, and ordinarily, management will act favorably on the advice of their medical chief.

In those plants smaller in size and scope, where perhaps the services of an industrial nurse only are available, contact should be made directly with the plant manager, or owner or the individual who heads the small organization. The utility of the ophthalmologist has not been realized to the fullest by the industrialist or small plant owner, and the step must be taken on the initiative of the ophthalmologist. What is the picture now in industry, and what do you, as practicing ophthalmologists, plan to do in the future?

These services that comprise the full eye program are being executed by professional *eye people*, if you will, specifically the optometrists, and in many instances by safety engineering personnel. They have been perhaps more far seeing in this field, and have either convinced management of the wisdom of such an activity, or have been secured by those of us in industry to get the job done for us. Frankly, this has resulted from the aloofness, disinterest, and disinclination of some oculists to leave their office buildings or hospitals. The present trend is further use of these individuals, for the work performed by them has been critical and completely satisfactory to the medical director. As Dr. Hedwig Kuhn has stated, "they [the ophthal-

MEDICAL SCHOOLS OFFERING TRAINING IN INDUSTRIAL OPHTHALMOLOGY

| *Number of Schools Surveyed* | *Replies* | | *Undergraduate Training in Industrial Ophthalmology* | | *Graduate Training in Industrial Ophthalmology* | | *No Training in Industrial Ophthalmology* | | *No Answer Received to Date* |
|---|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | No. | % | No. |
| 67 | 53 | 79.1 | 14 | 26.4 | 8 | 15.0 | 31 | 57.9 | 14 |

FIG. 7

mologists] must walk boldly out of their ivory towers and go directly to industry and offer their services." This is mandatory, for we want the very best medical prevention and care extended to the worker. This is your function, and unless a concerted effort is made by your group, this vast field will be barren of physicians at the skilled oculist level.

If there is concern as to any altruism involved, let us look briefly at the future. Management wants your expert opinion in medicolegal problems. They want your presence when insurance rates are being re-established. They want you to guide their employment and placement policies. They want you to conserve the worker's visual health, and they want you to protect the worker against his environmental hazards. Why? Because this is all good insurance—an insurance that pays dividends in actual dollars and cents, in health, and in availability and utilization of manpower. A protected, well-seeing, uninjured, nonabsent efficient worker is the goal of every personnel and medical policy. Wherein do you benefit?

Through the introduction of a vision conservation program, health promotion is being carried out, and the worker gets occupational glasses, he sees their value, becomes vision conscious, and endeavors to determine the value of his present all-purpose glasses. Information relative to the company's effort on his behalf is carried home, and his wife and Johnny and Mary wonder if they might need an eye examination and possibly new spectacles. Time after time, the medical director is asked about the need of a change of lenses, and most logically, this should be answered by the visiting oculist. Such a professional person conducting a health education program among the employees, can stress so easily the values of optimum vision, and how they may be attained. The opportunities are there to demonstrate to the workers the evils of the *five and dime* spectacles mentioned above — the fads of *throw away your glasses*—and the dangers to the eyes of certain cosmetics, and other examples of the harmful effects of self-medication in the ocular field. Health promotion without agencies available to effectuate the advice is a pointless program. By having an ophthalmologist at the plant, the employees will get to know him, trust him, and eventually go to him for their nonoccupational eye difficulties. That this rapport in industry is valuable, is a fact well supported by these oculists who have entered the portals of an industrial organization.

The ophthalmologist who will take the time in industry to educate his worker clientele as to good vision and maximum eye health, can help elevate the level of industrial worker morale by explaining

the actual pathologic or physiologic condition present that has given the patient the tag *weak eyes* or *bad eyes*. These stigmata do effect the psyche of the patient so labelled, and a discussion of the condition based on the philosophy of his (the patient's) own visual skills being of value, will elevate his morale, and eliminate his feeling of being a disabled person. In comparable fashion, may we always use the term refraction rather than eye examination. These are new designations to the worker, but his respect will increase when a value is placed on his visual efficiency, and what it means to the company.

The ophthalmologists who are now in industry can aid immeasurably by increasing the teaching in our medical schools of this specialized field. A survey of all the schools of medicine in this country was made, and from figure 7, the results may be seen.

## SUMMARY

In summary, it has been attempted to show the potential field available to the consulting ophthalmologist in industry. Through contact with the medical director in the larger industries, and with management directly in the plants of lesser size, the full program can be instituted, covering:

1. Efficient use of vision in industry
2. Prevention and treatment of eye accidents
3. Conservation of visual health
   a. Visual screening
   b. Occupational visual analyses
   c. Repeat visual surveys
4. Medicolegal ophthalmology
   a. Relation to constitutional disease
   b. Relation to occupational disease
   c. Compensation
   d. Protective insurance
   e. Liability

A plea is made for the ophthalmologist to associate himself with industry, for such an association, along with the efforts of the medical director, will result in: a. a happier worker; b. a healthier worker; c. a more productive worker; d. a lower absentee rate; e. lower accident frequency and severity rates; f. a lower employee replacement rate; g. a higher worker morale; h. better community relations; and i. better health behavior of the generations to follow.

## REFERENCES

1. Tiffin, J.: Industrial Psychology, New York, Prentice-Hall, Inc., 1943, p. 136.
2. Felton, J. S.: Three Cornered Co-operation. Nat'l. Safety News, 53:26, (Feb.) 1946.
3. Job Analysis for Occupational Vision, Supplementary Bulletin to Manual and Appraisal Form on Conservation and Utilization of Eyesight in Industry, Publ. No. 402 (Revised), New York, Nat'l. Soc. for the Prevention of Blindness (April) 1944.
4. Accident Facts, 1946 Edition, Chicago, Nat'l. Safety Council, Inc., 1946, p. 38.

ADDITIONAL REFERENCES

Kuhn, H. S.: Industrial Eye Health Problms, Am. J. Pub. Health, 33:1103 (Sept.) 1943.

The Field of Industrial Ophthalmology, a Report by the Committee on Industrial Ophthalmology of the Section on Ophthalmology of the American Medical Association, J. A. M. A., 118:610 (Feb. 21) 1942.

# THE OPHTHALMOLOGIST AND THE INDUSTRIAL NURSE

HAZEL H. LEEDKE, R.N.
KAUKAUNA, WIS.

MY SUBJECT can no doubt be approached from several points of view. In order to cover questions Dr. Kuhn has asked me to discuss, I have divided my remarks into two parts: 1. Building an industrial vision program for which material I have drawn upon my own experience; 2. What the industrial nurse wants from the ophthalmologist in the way of support. To try to bring a representative opinion on the latter, I have sought the advice and opinions of industrial nursing consultants from health departments and insurance companies and other leading industrial nurses.

That I have had the privilege of observing and participating in the building of an industrial vision program is a tribute to the guidance, encouragement and inspiration given me by Dr. Hedwig S. Kuhn. To have watched the evolutionary process of the development of an industrial vision program as it now operates at the Thilmany Pulp & Paper Company has been a rich nursing experience, and I hope my remarks will be considered from a nursing and nurse's viewpoint. They are not meant to be profound nor have any medical implication.

## BUILDING AN INDUSTRIAL VISION PROGRAM

As I look back over more than 17 years in the industrial nursing field, I realize my interest in the worker's vision as it relates to safe performance of the job has been due to various incidents. For example: one day, a good many years ago, an employee reported to me that he had lost some vision in one eye and he attributed his diminished vision to an injury he had sustained. Medical records revealed he had selected the wrong eye to blame his complaint on the injury to which he alluded. However, this set me to wondering how many compensation cases had been settled on the basis of impaired vision due to an injury when perhaps so-called normal vision had not been present at the time of injury. Using this case as an example, I gained desultory permission from my employer to try some vision testing, using a Snellen chart. Permission was granted provided I could persuade anyone to submit. Only a few cared to try, and as I look back I realize this amounted to nothing more than whetting my own interest. It was simply an inch in the long mile toward a goal.

About this time it was my good fortune to know Dr. Kuhn. Exposure to her philosophy only aroused greater interest on my part. Also, about this time I changed positions, going to the Thilmany Pulp and Paper Company where an expanding personnel and production program coupled with an enlightened management made possible a wider nursing experience.

Because of this fact, it was my opportunity to develop a broad health program. As a part of such a program, emphasis was placed on vision and teeth as two types of defects offering opportunity for improvement. From physical examination records, defect files were made. Our first efforts were directed toward development of a dental program, the success of which we are very proud.

Thilmany Pulp and Paper Company, Kaukauna, Wis.

Again, another incident gave impetus to a vision program though at that time there was no organized plan made. One day an employee broke his eye glasses on the job and came to me with the request that the Company replace them. Seizing upon this opportunity to talk safety glasses with prescription lens, I obtained consent of the Company to pay half on such glasses as an incentive to a broader safety goggle program. But this brought a new problem—why send good money after bad—in other words, were we wasting money unless we knew the worker's prescription was adequate?

An oculist was approached—the problem discussed. Under the panel system it was evident that any one person would be reluctant to take on the task of refracting every employee. Moreover, if a professional man could have been obtained, the cost of equipping a room for his use at the mill was prohibitive. To send around 1000 employees to his office would have been too costly in time and would have cluttered up his private practice. It is quite obvious why such a plan was impractical and probably very unnecessary. And, so, the question arose —what could the nurse do.

Under the direction of an oculist a Snellen chart was mounted in a black frame, lighted by two fluorescent tubes, and hung in the first aid room. Proper distance was measured and marked. A little later a reading test was added for 40 year olds. Subjects were selected from physical examination records as needing eye care, but these records were then more than 2 years old. I well recall trying to watch feet and eyes at the same time proved very difficult and that measuring 14 inches with a ruler was next to impossible as the subject pushed the card farther and farther away, saying, "Of course I can read it if you let me hold it so I can see it."

The sum and substance of the whole thing was a state of frustration on my part for I felt nothing was really accurate. I had no special performance record cards but recorded results on an employee health record card which was large and difficult to handle. The whole thing seemed a hit and miss proposition.

Knowing Dr. Kuhn's interest in employee-vision, we turned to her for help. Dr. Kuhn trained me in the proper use of the Keystone telebinocular (this not being as simple as it seems) and outlined for us a program for a screening survey. While very top management was interested or we would never have accomplished this much, there was a selling job to do to supervisors, foremen and the personnel. Cooperation was secured from the Union. No doubt the success we had in our dental program which had been built on employee and professional cooperation helped us on this job. Professional people were told about the plan. While we had our trials and tribulations, we succeeded in testing the entire personnel of 1143. Dr. Kuhn analyzed the performance records and indicated those in serious need of correction. The following is her analysis:

| | |
|---|---|
| Total No. of individuals who had acuity defect | 257 |
| Per cent of whole plant | 22.4 |
| Total No. of individuals with uncorrected acuity defect | 142 |
| Per cent of whole plant | 12.8 |
| Total No. of individuals with a muscle balance defect | 101 |
| Per cent of whole plant | 8.8 |
| Total No. of individuals with a stereoscopic defect | 106 |
| Per cent of whole plant | 8.8 |

Reports of passing or failure, based primarily on an arbitrary standard of 20/40 for distance acuity were mailed to every employee. The reaction was terrific from some sources but management remained solidly behind the program. On the basis of the above mentioned standard an attempt was made to get corrections. On a few occasions I was accused of getting a big cut-in on

glasses prescribed by the doctors, however, on the whole the program was accepted with deep appreciation in most cases. After a few corrections, these employees became our best salesmen. Management was kept informed on employee-reaction and a letter of policy was sent me with authority to use it, but I am happy to say that it has never been taken from the files. To have resorted to it, would, in my opinion, have been an acknowledgment of failure on my part.

As a selling point to our foremen's safety committee, I analyzed the 1943 injuries requiring medical care from a vision standpoint based on the 20/40 standard and on Dr. Kuhn's indication of failure on other items of the test. The record of the 230 cases showed:

| | | |
|---|---|---|
| Depth perception failures | 39, | or 17% |
| Color failures | 37, | or 16% |
| Muscle balance failures | 16, | or 7% |
| Visual acuity failures corrected to best less than 20/45 | 8, | or 3% |
| Muscle balance failures and depth perception failures | 5, | or 2% |
| Muscle balance, depth perception and color failures | 1, | or ½% |
| Visual acuity failures, uncorrected | 1, | or ½% |

Of the 30 lost time cases of this group:

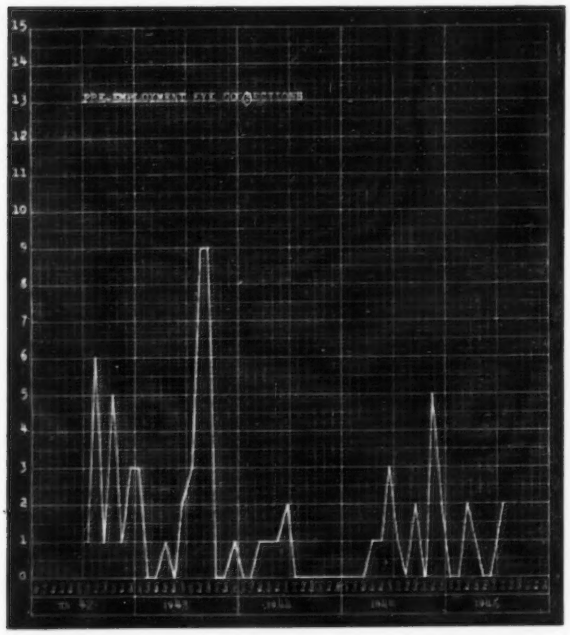
| | | |
|---|---|---|
| Color failures | 8, | or 27% |
| Depth perception failures | 6, | or 20% |
| Visual acuity failures, uncorrected | 1, | or 3% |
| Visual acuity failures corrected with best less than 20/45 | 1, | or 3% |

It is obvious that the portion of the test we were laying the most stress upon, namely, distance acuity, was a small factor in this analysis.

As an example, we had a very serious accident in which a hand was "drawn into the pinion and connecting gear which operated the knife of the cutter." While the employee had violated a safety rule by removing the guard to do some wiping up, the accident investigation and the employee himself failed to be able to give a satisfactory explanation how he could have been drawn into the machine at the particular spot. This man's eye test was brought out. He had failed on the depth perception test according to Dr. Kuhn's rating (less than 8 out of 10). For the want of a better explanation it was thought that his poor judgment of distance might have been a contributing factor. As this was a very severe injury, it was brought to the safety meeting and the department head estimated the cost of the case in terms of production. It was his opinion that this machine would have to run 17½ weeks, or 1388 hours, at 1200 pounds per hour to earn sufficient profit to pay the tangible costs of the case.

Labor was considerably more plentiful in 1943 and early 1944 and applicants were required to meet the 20/40 standard. Old employees were urged to get professional care but were never compelled to do so. The Credit Union waived all restrictions on borrowing in cases of optical or dental care as an incentive to the corrective program and evidence of cooperation with us in this program. In April, 1944, the Thilmany Employees Association became sufficiently interested to include dental and optical benefits to their members. Benefits amounted to one-half the bill up to $15.00 annually.

Then as 1944 came along, the pressure for labor came. It was not uncommon to hear personnel say, "Why a blind man can do that job." It became more evident that guesswork as to what the job required wasn't enough. Somebody had to know and no one did. Having heard about the Ortho-Rater and the fact finding system which provides for just the portion of the vision program we were struggling with — namely, job requirements, we applied for one and finally secured one to begin testing in October, 1944. No doubt you all know what the Ortho-Rater program is and its practical application to the industrial situation.




GRAPH 1. Pre-employment Eye Corrections.

In retrospect, it appears to me that we have learned the hard way. Compensatory factors in this long difficult method, however, seem to be that we had sufficiently conditioned management, personnel, union and professional people that no doubt some of the problems which might have been encountered were already water over the dam and we could proceed with more dispatch. Testing was not completed until around March or April, 1945. Statistical analysis and job standards were completed so that by July, 1945, reports were out to employees and corrective work could be started.

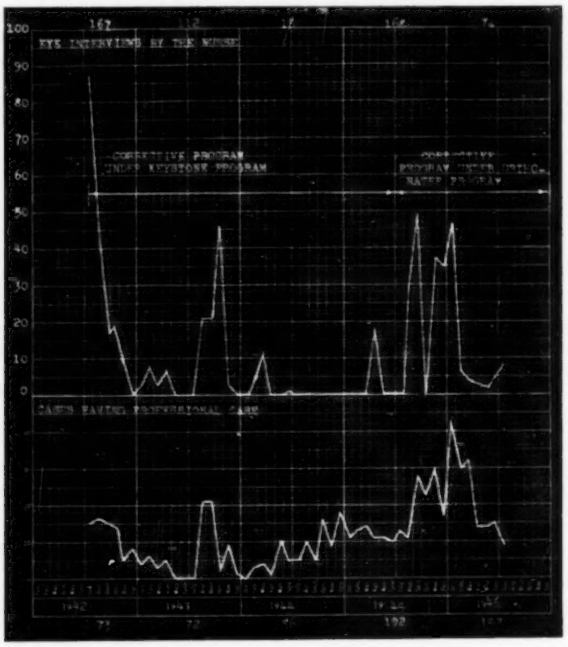
What did the fact finding system reveal at Thilmany's? It revealed just what has been found in other plants—that the good producers were those who were meeting their job vision standards. It is my understanding Thilmany's records were the first to be used to show correlation between visual performance and accident experience. The Ortho-Rater program has demonstrated to us that there is a relationship between efficiency and vision, between safe performance of the job and vision, and through job vision standards it is expected that better placement visually will prove effective.

By this time Thilmany's had worked up to the point where the Ortho-Rater program could be coordinated with other phases of employment. Job analysis had been done with regard to physical requirements of the job with an eye to placing disabled veterans. It was simple to add to it a visual analysis for the Ortho-Rater program. Physical capacities appraisal forms were added to the medical pre-placement examination. Job vision standards were added to the form. Placement is now guided by both physical and visual factors and control maintained through a system of transfer cards. Foremen are informed who needs to wear his prescription to meet vision

| | *Had Serious Accidents* | | *Had No Serious Accidents* | | *Total* | |
|---|---|---|---|---|---|---|
| | *No.* | *%* | *No.* | *%* | *No.* | *%* |
| Those meeting all major standards | 22 | 37 | 37 | 63 | 59 | 100 |
| Those not meeting all major standards | 30 | 67 | 15 | 33 | 45 | 100 |
| Total | 52 | 50 | 52 | 50 | 104 | 100 |

TABLE 1

Relation between visual performance and incidence of serious accidents among paper machine operators.



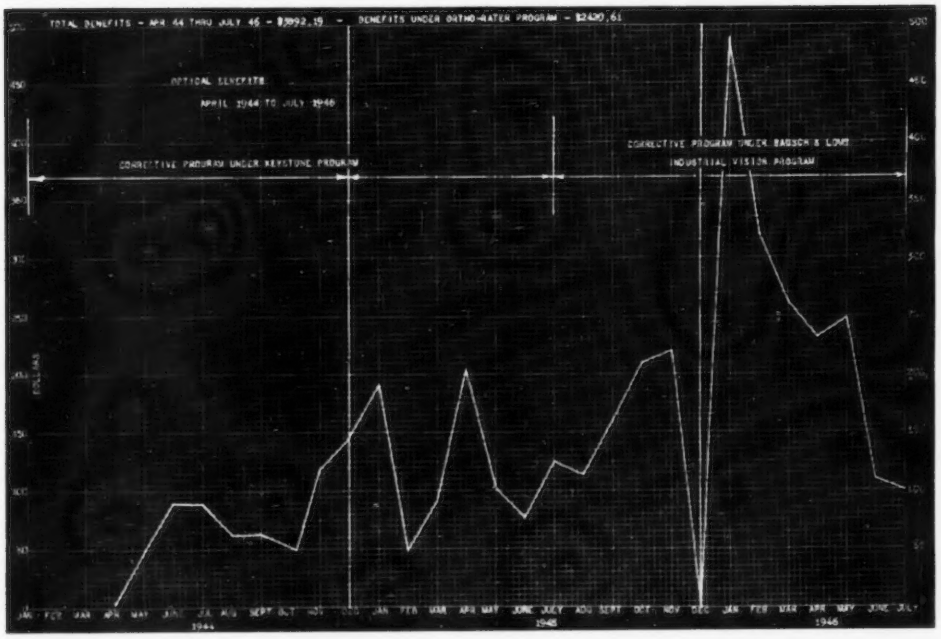

GRAPH 2. Corrective Program.

standards. Vision standards have been lumped into five groups, Plant, or the maximum, A, B, C, and D. It is our desire that as many as possible meet the plant standard in order that transfers may be facilitated.

### WHAT HAS THE VISION PROGRAM MEANT TO THE OPHTHALMOLOGISTS?

Since July, 1942, the beginning of our first concerted efforts toward correction, 580 employees have received eye examinations by professional people. The nurse has interviewed 539. As of September 1, 1946, our personnel of 1,243 showed 405 wearing eye glasses, 204 of which were meeting job standards with their prescriptions; 201 were still failing to meet job standards with their prescriptions. Two hundred and seventy-nine received professional care in 13 months (July, 1945, through July, 1946) under the Ortho-Rater program, or nearly 50 per cent (.481) of the total corrections over the past four years.

Insurance benefits became available in April, 1944. Eight months of 1944 showed benefits paid out amounted to


TOTAL BENEFITS - APR 44 THRU JULY 46 - $3892.19 - BENEFITS UNDER ORTHO-RATER PROGRAM - $2420.61
OPTICAL BENEFITS
APRIL 1944 TO JULY 1946
CORRECTIVE PROGRAM UNDER KEYSTONE PROGRAM
CORRECTIVE PROGRAM UNDER BAUSCH & LOMB INDUSTRIAL VISION PROGRAM
DOLLARS
1944
1945
1946

GRAPH 3. Optical Benefits Paid.

$671.26; 1945—$1503.75 and 7 months of 1946—$1717.18, making a total of $3892.19. Two thousand, four hundred twenty dollars and sixty-one cents ($2,420.61) of the total was paid out from August, 1945, through July, 1946. August, 1945, was selected as the first month in which bills from the Ortho-Rater corrective program would be coming in. To obtain a more complete picture of the monetary value of the program simply multiply this total by two as the optical benefits of the T.E.A. covered only half of the bill up to $15.00.

### WHAT DOES THE INDUSTRIAL NURSE WANT FROM THE OPHTHALMOLOGIST?

The industrial nurse wants above all else your understanding and support. From all sources contacted came one plea—namely, a plea for broader medical guidance and signed standing orders. This, of course, applies to nurses who have neither full time nor part time medical directors, but such nurses cover the majority in the industrial nursing field. Industrial nursing consultants have asked me to carry to you the message that most industrial nurses are ethical; that in general they are *afraid* of eye cases and are referring more eye cases to the doctor than any other type of injury. Further, they tell me it is a rare case to find a nurse removing embedded foreign bodies with a sharp instrument or eye spud but that foremen, well-meaning fellow workers and first aid attendants are the chief offenders. They would like your cooperation in interpreting the vision program and its relation to the industrial health program.

You can only understand the industrial nurse's problems by coming to the plant, knowing her, the management, the plant hazards and the safety program. Support her and the safety program by helping her interpret to management the vision program, need for eye protection and the effectiveness of such protection through safety glasses

with prescription lenses and all other types of protective eye ware. Help sell eye protection as you see injured employees in your office. Industrial nurses realize how little they know about vision programs and care of eye injuries and from all sources contacted came a request for institutes.

The industrial nurse has brought her problem to your doorstep. If she cannot get the assistance she needs from the medical profession, to whom can she turn?

Since I started to prepare this paper, I have been notified by Dr. Paul A. Brehm of the Industrial Hygiene Division of the Wisconsin State Board of Health that Wisconsin nurses are going to get their pleas answered. In a letter of Septmeber 20, 1946, Dr. Brehm reports that at a meeting of the Industrial Health Committee of the State Medical Society, September 18, in Milwaukee, "there was considerable discussion regarding the responsibilities of doctors in the community, whose names appear on the panel, in regard to their assistance to industrial nurses. It was agreed that where an industry employs a full time or part time physician as director of the medical department, he is responsible for signing standing orders for the industrial nurse. In plants not having direct medical supervision, it was felt that physicians listed on the panel should assume the obligation of signing standing orders for the industrial nurse."

Dr. Brehm's letter has another significant paragraph which gives great encouragement to Wisconsin industrial nurses. "Panel physicians on an *on-call* basis should be willing to render supervision and medical guidance to the industrial nurse. The Industrial Health Committee of the Wisconsin State Medical Society has very definitely gone on record urging those physicians to do that very thing." As a Wisconsin nurse, I am very grateful.

# NEW THOUGHTS IN THE TREATMENT OF OCULAR INJURIES

DON MARSHALL, M.D.
KALAMAZOO, MICH.

THE title of this paper was assigned with the thought that out of the experiences of the recent war must come ideas and methods that will prove of benefit in the handling of civil and industrial injuries. It is with much humility that I enter such a discussion, interesting as it is, and touch on some high points of ophthalmic traumatic surgery, based on experiences over a three year period in the ETO. Every month is bringing publication of excellent reports by other ophthalmologists covering sections of this field; reports that in a thoroughly scientific manner present analyses and statistics of ocular war trauma, the results of therapy, and even of research carried on during the pressure of warfare. Valuable progress is being reported by workers in this zone of the interior, who have advanced far the medical and prosthetic repair of the scars of war.

Because of the constant and usually rapid flow of patients past us, few ophthalmologists in the European theater are in a position to speak authoritatively on the complete care of large numbers of eye cases. I certainly am not one of them. There were thousands of eye patients and a shortage of eye specialists, so that before it was over we all had plenty to do, but few patients were with us from the beginning to the end of their treatment. A hospital in the rear received patients with injuries days or weeks old, with initial, and often final, surgery completed. The surgeon at the front could do only the initial surgery, then lost his patient to the rear areas.

Since several different surgeons worked on a given patient, with no contact with each other except the patient and his medical record, it will be difficult to assess differing methods of therapy. Those surgeons who were the busiest, had little opportunity to keep duplicate records for later personal study and analysis. The man at the end of the line in this country, with the patient and his record before him, is probably in the best position to make critical appraisal of therapeutic methods, but that man very likely was never at the front and therefore lacked full understanding of the trials of war surgery, of operating around the clock during a push, under bombing, of being short of time, anesthetists, sleep, and sometimes equipment, light and water.

Out of the hundreds of hospitals handling thousands of wounded must come conclusions, developments, and technics that should be applied to civilian surgery. What are they? The answer is something of an anticlimax. For the most part, ophthalmic war injuries have their counterpart in civilian life; war surgery of the eye presented few problems not already encountered in civilian accidents, although in infinitely greater numbers. The care of war injuries was best accomplished by following the standard methods developed in civilian ophthalmology. Experience taught modifications, and necessity hatched a few new ideas. It is these that will be considered here, without attempting to encompass the entire field of traumatic ophthalmology. It would be easy to give many illustrative cases, but to detail them would take too long.

Long before we entered the war, the British learned during the bombings

which they endured, that not all perforating eye injuries need be considered acute emergencies. If the patient is in shock from other injuries, his eyes with a little first aid care can usually wait temporarily without harm. With the dawn, both patient and surgeon may be in a better state for operation, the surgeon exhibiting "that deliberation and delicacy" so essential to good work, as described by a confrere in the Italian campaign. If the patient is to be anesthetized for his other wounds the eye surgery can be done simultaneously. Haste makes waste and poor work. It is essential, however, that the patient be relaxed and quiet on a bed or litter, with both eyes closed. A clean corneal or scleral laceration is easily converted into a collapsed, lost eye by a little squeezing or straining. Since pressure on the lids can do as much damage, examination under anesthetic, and the use of lid sutures instead of a speculum, is often safer than the usual methods. A patient suspected of a perforating eye wound, especially a large one, should sit or lie down awaiting a stretcher, close both eyes, and try to avoid squeezing. Too often in war and industry such men walk, stoop, strain and squeeze, to say nothing of pushing on the closed eye from pain.

With the first full flow of war wounded, it was soon realized that severe body injuries often distracted attention so much that a satisfactory eye examination was not made. Severe ocular injuries were passed off as minor, or the necessity of saving life before sight forced postponement of ocular study and care. In civilian industry this situation should seldom occur. In any question of ocular injury, the eyes deserve careful examination as early as possible. To omit it may hide ocular and even intracranial damage, and often the fundus can be visualized much better early than late, after lens opacities and hemorrhage obscure the view.

Corneal foreign bodies produced a frequent clinical picture in this war seldom seen in civilian life. The powerful explosion of shells, land mines and booby traps peppered the face with bits of sand, gravel, powder, metal and other debris that penetrated to all depths of the eye. The best treatment was deliberately conservative. To save vision, corneal scars had to be kept to a minimum. This meant first avoiding infection, and second establishing a nice balance between removing and leaving the foreign bodies. Too much digging produced keratitis and scar. Leaving the foreign bodies sometimes produced reaction and scar. Routine use of sulfa and penicillin both systemically and locally, nearly always prevented infection. Judicious removal of the superficial material and the larger deep ones, while carefully observing the rest for local reaction, seemed to work best. Most of the foreign bodies were inert, and many extruded themselves spontaneously in time.

Much has been done and much has been said in all the services about the localization of intraocular foreign bodies. It was a real problem. Many hospitals lacked good Sweets localization equipment. Many learned the failings of that method in war. It is not infallible, and in the presence of multiple small foreign bodies throughout the orbit and eyeball it may be useless, for the corresponding shadows in the two views cannot be paired off. To improve accuracy, or to improvise localization equipment, contact lenses and metal rings were used extensively, with satisfaction. A particle of lead can even be sutured where the localization indicates the foreign body to be, or near to it, and the eye re-rayed. Oxygen injected into Tenon's capsule helps in localization of a foreign body as being in or outside the globe. None of

these methods is new, but the war gave wide experience in their use to many men, and taught that we should not depend on a single localization technic, but should be ready to try others where indicated in a given case. The Berman locator was baptized in war at Pearl Harbor, and as adapted for the eye, seems highly valuable to those who have had opportunity to use it.

Ideally, every intraocular foreign body should be removed, and promptly. Neither requirement can always be met, especially in war. Ocular foreign bodies in war tend to be larger, and to strike with greater force than in peacetime. The force may be more important than the size. Glass, plastics, some stones, and certain metals like aluminum and lead may remain in the eye totally inert. Most other metals cause a chemical endophthalmitis, by electrolysis, of varying degree. Unlike civilian practice in war one can seldom judge from the history with any certainty the composition of an intraocular foreign body. Foreign material removed from other body wounds may give a lead, but it may be false. It is of course urgent that the presence of a foreign body be recognized, that it be located accurately, and, if intraocular, it is always desirable that it be removed. That does not mean that *any* procedure is justifiable to remove a foreign body.

When a foreign body is removed from the vitreous chamber it is usually done with the aid of a magnet. Entering the posterior segment for a nonmagnetic foreign body is not only difficult but hazardous. If a foreign body is magnetic, the chances of its removal are good unless it is very small or firmly embedded. Estimation of the certain, critical, and failure zones in the field of the magnet pull will help in the prognosis. The practice of our British allies of using a magnet pull as a test for foreign body, before x-ray studies and localization, finds little favor in this country. But one seldom-mentioned, simple procedure can be of considerable value as a preliminary test. In those many instances where a small foreign body free in the vitreous can be visualized with the ophthalmoscope, it is easy and safe to test its magnetic qualities by cautiously approaching a hand magnet to the eye while keeping the foreign body under direct observation. I did this many times, causing the foreign body to twist or swing slightly from its resting position, thereby establishing without doubt its attraction by a magnet.

Uveitis and siderosis may result from a retained foreign body in the eye, but these complications certainly are not inevitable, nor do foreign bodies always cause ultimate loss of vision. Two exceptions to prove the rule can be noted. A fighter pilot who had a piece of plastic, from his shattered hood, pierce an eye in combat over Africa in December, 1942, in two months had a quiet eye, with normal fields and vision, and returned to flying status six months after the injury. Our chairman may recall seeing in England a young man who applied for Officer Candidate School, and on routine physical examination was found to have a sausage-shaped mottled tumor attached to the posterior retina. It was suspected of being an odd melanoma, but x-ray proved it to be an encapsulated 6 mm. sliver of iron, magnetic to test under direct observation, incurred at the age of five, in a shop. The eye was free of symptoms, irritation, siderosis, and had perfect vision. It was left alone.

In the repair of lacerations of the globe, there is some controversy over the relative merits of a direct suture of the wound edges, and the use of a conjunctival flap. Well placed fine silk or cotton sutures uniting the sides of a laceration of the cornea or sclera are undoubtedly preferable and stronger. Some

corneal wounds do not gape and need nothing. If sutured adequately, as in a cataract operation, a conjunctival flap may be superfluous. But placing such sutures in an injured eye is often very difficult. If placing them necessitates pressure on the globe with further prolapse, they should be omitted, and a conjunctival flap used instead, even a double flap over a scleral wound. The British found in Africa that a total conjunctival flap with pursestring suture was the only type that could be depended upon to stay any length of time. This total flap certainly has its place in civil practice. Its objection is that one has no view of any cornea or globe to watch healing, infection and other complications. In the evacuation of war wounded this was especially difficult. Only the initial surgeon knew what he had covered up.

Nearly as much vision was lost permanently in this war by concussion and contusion as by actual lacerations. Blast injuries occurred, but were outnumbered by direct contusions, many of them noncombat, and by indirect contusions caused by a missile passing through the orbit or head, at great speed, near the globe. Extensive and extremely severe traumatic choroiditis was very common from that mechanism. In Cherbourg, at one time over half my in-patients were men with noncombat contusion injuries, blows by elbows, baseballs, bats, apples, etc., causing everything from commotio retinae to recurrent hemorrhage and glaucoma. This is a surprisingly frequent, and surprisingly serious, type of injury, in war or peace, and without effective treatment other than rest and care of complications. Many have anterior chamber hemorrhage. In that case, paracentesis should be done if there is glaucoma and no visible iris. Atropine benefits the iritis, but some recommend no atropine, in order to increase the surface of iris stroma for absorption of blood.

It would be helpful if someone could give us a rule on how to handle a fluffing traumatic cataract. Most likely the injury has involved both anterior and posterior lens capsule, and vitreous is present in the lens wound, or will appear if an early attempt is made to remove the lens. Waiting will produce much lens absorption, especially in young adults, and lessen the chance of vitreous prolapse, but brings the likelihood of iritis and glaucoma, and a prolonged irritability, as complications.

Without question, the sulfa drugs and penicillin reduced tremendously the incidence of ocular infection. Every wounded man received both routinely, and free use was made of both, locally, in the eye. They undoubtedly saved thousands of eyes from infection and loss. Local allergic reaction to their use was soon encountered, and must always be kept in mind. Now we can add streptomycin to this invaluable group of antibiotics. It has been shown that of all common local antiseptics for use in the eye, only penicillin and aqueous sodium sulfathiazole can be used repeatedly and continuously without some damage to corneal epithelium and interference with wound healing.

The war gave great impetus to solve the cosmetic and prosthetic problem of enucleation. Many war injuries completely shattered the globe, tearing it literally to shreds. The usual simple operation of enucleation became then a long and tedious procedure. Stock army glass sphere implants were usually used when possible, and when they were unavailable some made plastic spheres, even hollow plastic spheres, as improved substitutes. Implants may migrate in the orbit unless held by an eviscerated sclera. There is some question on whether they really aid in the final appearance

and movement of the prosthesis. There is much to be learned in this field, and praise and support should be given to the many who are working ingeniously to improve the socket, the prosthesis, and the sunken upper lid, after enucleations. The story of how the services pushed to rapid completion and extensive use an excellent method of making plastic prostheses has been told. It is one of the most satisfying steps forward in the ophthalmic history of this war.

In the months before Normandy, warning instructions had to be spread to all surgeons to be ultraconservative in deciding to enucleate. The average physician has a fear of sympathetic ophthalmia, and if put on his own is apt to be too radical on the point. Fortunately it is a rare condition. Most of us did not see a case throughout the entire war. Modern methods often save eyes that would have been lost twenty years ago. The post-traumatic safe period of about fourteen days should be utilized to treat and observe the eye, especially when both eyes are involved. After enucleation, insertion of a plastic conformer or temporary prosthesis is of great help in preventing postoperative conjunctival chemosis, and in maintaining the size and shape of the cul-de-sac.

Fortunately the training of all troops against war gas was not put to a test in our theater, except against phosphorus, and in a few accidents. The number of thermal and chemical ocular burns seemed as numerous from noncombat accidents as from combat incidents. British anti-lewisite ointment proved invaluable against arsenical burns. For all others, copious irrigation with water, and careful removal of all particles of chemical, was the tried and true therapy that stands the test of time well. It has been shown that simple plastic eye shields will protect the eyes against ordinary chemical droplets, as well as from most small flying particles. Their use as a precaution in any occupation hazardous to the eyes, from grinding to plastering to filling a battery, should be more widespread.

In point of numbers, the field of plastic ophthalmic surgery is bigger in war than in peace. A trained ophthalmic surgeon is better at repairing defects around the orbit, and at suturing lacerations of the face, than the average general surgeon. But plastic surgery has advanced much during the past decade. A capable plastic surgeon excels all but specially trained ophthalmologists in the repair of large defects, the use of various types of grafts, and the application of new methods. Ideally the two should work together. Some plastic surgery would be avoided if more time and pains were taken in doing the original primary suturing of the wounds. This is more feasible in peace than war. Ophthalmologists should be acquainted with two advances developed during the war in the field of wound repair, although in civil practice they will seldom have to use either method around the eyes. One was the delayed or secondary closure of soft tissue wounds; the other the early skin grafting of large defects. Another development has been the use of plastics to gradually enlarge contracted sockets by stretching, and making them fit for prostheses.

Although new developments have come out of the war for our specialty, by and large the training of our prewar schooling holds good, and the best civilian ophthalmology has little to learn from the recent war, except greater experience in technics and methods already worked out before Pearl Harbor.

# THE SPECIFICATION OF ILLUMINATION AND COLOR IN INDUSTRY

FABER BIRREN
NEW YORK, N. Y.

THIS is meant to be a practical story, based more on a summary of actual experience in industry than on theoretical premise. In recent years illumination and color have been extensively and intensively studied by ophthalmologists, lighting authorities and color specialists. Unfortunately much of the literature thus developed has been bewildering and even contradictory. In many instances, an awkward terminology has defied a clear understanding by others. Some authorities, with instruments and meters in their hands, have attempted to draw conclusions that disregard physiological and psychological factors. Illumination has been confused with brightness — and both with the anomalies of vision. Because of all this, the practical specification of illumination and color in industry has been difficult, controversial, and beset by statements of principle that seem to run in cross directions.

It should be understood at the outset that light and illumination are meaningless except as they are reflected or transmitted into the human eye. In the stratosphere the sky is black. Too much of illuminating engineering overlooks the fact that one or a thousand footcandles mean little or nothing until such energy is converted into brightness and thus perceived.

Again, brightness holds advantage over darkness in the action of seeing. Not only does it stimulate the retina and regulate the pupil opening, but it is psychologically more compelling. Yet while high brightnesses may add efficiency to illumination, they are not always desirable. Under some conditions they may interfere with the seeing task and handicap vision. Low brightnesses, incidentally, are the general rule in nature.

Today, one basic fact seems to be universally agreed upon—"that a human being sees best and visual fatigue is reduced to a minimum when the entire field of view is of approximately the same luminosity as that to which the fovea is adapted." (Parry Moon.) Thus an orderly discussion of recommended practice in the use of illumination and color may be set off to a good start. Where luminosities are not uniform and where severe contrasts exist within the field of view, vision may be taxed and glare conditions may be present.

If I understand rightly, most of the discomfort associated with so-called eyestrain is due more to efforts at accommodation, muscular imbalance and the like rather than to overstimulation of the retina. The human eye can look alternately at moderately high brightness and low brightness without undue discomfort and strain, if in each instance the whole field of vision is involved. However, if high brightness and low brightness exist in the *same* field of view, and if they must be concurrently rather than alternately accepted by the eye, trouble will set in and vision will be inimically affected.

In the consideration of any lighting system, exposed light bulbs or rows of fluorescent tubes or opalescent globes should obviously be avoided. Some il-

Presented as part of the instruction course on Industrial Ophthalmology at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

luminating engineers can quote actual figures for the upper limits of surface brightness in lighting fixtures. Such figures are not particularly needed, and they may be misleading. The requirements of a lighting system are very obvious and very simple—uniformity as against "spotty" contrast.

Illuminating engineers have argued at length for and against direct and indirect lighting systems. In my own experience in hundreds of industrial plants throughout the country, I have found both systems ideal—not one better than the other, but each highly satisfactory under certain conditions. Lighting authorities seem to favor the direct system because it will deliver more footcandles upon average working areas. Ophthalmologists often favor the indirect system because it effects a softer and more even distribution of light (and not so many footcandles).

To go back to the basic principle that the eye sees best where luminosity is uniform, there is unquestionably a vast difference between intensity of light and the ability to see clearly. If a lighting system is such that it exposes the eye to great extremes in light and dark (possibly bright overhead fixtures and dark floors and equipment) constant pupillary adjustments will necessitate a lot of light on the task itself for the eye to "fight" its way through the extremes. However, if uniformity of brightness is apparent —and eye adjustments unnecessary— the eye can actually see better and more clearly under relatively dim light (footcandles to the contrary).

The indirect lighting system is desirable and appropriate in offices, for example, and is almost always psychologically appealing. The best type of fixture is one with an opalescent bowl or reflector which will transmit an intensity of light that approximately matches the brightness reflected by the ceiling—thus blending fixture and ceiling into one relatively uniform area. Such fixtures are made both for incandescent and fluorescent sources. Indirect light (semi-indirect in the above instance) seems to be "easy" on the eyes. It effectively reduces shadows and specular reflections. The room appears "cheerful," lacking in harshness, and vision may operate efficiently under footcandle levels which many a lighting authority would disparage.

However, where floors or equipment may be unusually dark, the ceiling overhead in the indirect lighting system may become a glare source. This is no fault of the system. By using lighter colors on floors, desks, furnishings, more uniformity will be realized (and more footcandles because of multiple reflections). The system will be at its best.

Indirect lighting systems (at least in my experience) are the safest of all to assure visual comfort. They are not efficient from the lighting standpoint, for the mere fact that the bulbs or tubes are concealed and light distribution must depend chiefly upon reflectance. Turn the reflectors around, expose the bulbs or tubes, and footcandle readings will jump up—but visual efficiency may simultaneously collapse.

Hence the direct lighting system requires expert care and engineering. In industrial plants having monitors, high bays or exceedingly high ceilings, this method of illumination is generally the only practical one. In certain machine operations it holds the added advantage of giving form and shape to materials or products. (Indirect light tends to "wash out" highlights and shadows.)

The fault with many direct lighting systems is that the eye is too frequently exposed to "naked" bulbs or tubes. They may stand out in marked contrast with a dark ceiling or overhead, thus raising havoc with the principle of uniformity. The conventional type of RLM reflector is often inadequate. By introducing

deeper shields or reflectors, baffles, diffusing lenses and the like, glare may be reduced and the eye less bothered. Where the reflectors are near the ceiling they should also be painted white on top to blend with the overhead. Obviously, machines and floors should be as light as possible both to reflect more light and to reduce brightness differences.

Further, where the task may demand critical use of the eyes to distinguish fine details, supplementary light sources (over and above the direct or indirect lighting system) may be necessary. Some tasks require a lot of light. Yet, as ever, the surroundings should be kept as uniform as possible in brightness.

As to spectral quality (color) in light, a yellowish tint—natural to incandescent bulbs—is excellent. In fluorescent tubes, the so-called 3500° tube is superior to the "daylight" tube for general room lighting. "Daylight" illumination, in fact, has been found definitely objectionable, except where accurate color discrimination is required. Its bluish quality tends to "blur" the visibility of objects; it is "cold"; and it has a very unflattering effect upon human complexion (which may lead to a neurotic form of "eyestrain" among women).

"A visual task is inseparable from its environment." Thus a proper control of illumination must be supplemented by a careful regard for the use of color in the painting of ceilings, walls, equipment and machines. Luckiesh has written: "The general absence of controlled backgrounds for seeing and lack of consideration of them are simple illustrations as well as eloquent testimonials of the general absence of a consciousness and knowledge of the factors which influence seeing."

Industry sorely needs good eyesight. The correction of visual defects becomes a mere locking of the barn door until ophthalmologists comprehend that bad conditions often cause discomfort—and these conditions must be righted at their source within the industrial plant or office.

Because the human eye needs light to see, and because the process of vision depends almost wholly upon reflectance, the matter of color and brightness is all-important. The statement has been made that it is just as easy to see a strand of *white* thread on a black background under one footcandle of light as it is to see a strand of *black* thread on a black background under two thousand footcandles. "Seeing is not a matter of looking at light waves as such, but of looking at external things mediated by these waves; the eye has to instruct us, not about the intensity or quality of the light coming from external objects at any one time, but about these objects themselves." (Ewald Hering.) Thus while vision is dependent upon light, perception itself seems to be curiously independent. As a matter of fact, illumination is "seen" almost entirely in terms of the appearance of objects in the field of view. If light sources were concealed, a room painted a dull black would appear dark regardless of the volume of footcandles showered upon it.

In my opinion, illumination is too often given an unreasonable amount of attention in factory design—at the sacrifice of a sensible attitude in the application of color. Lighting systems designed to double or triple the volume of illumination within an interior very often lead to serious complaints and to a drop in human efficiency and production. At the same time, lighting is easily overdone. Any eye specialist has reason to be skeptical about contentions that certain tasks require a certain intensity of footcandles. They do and they don't, depending on the relative distribution of brightnesses within the field of view, degree of contrast, visibility and a dozen other factors.

However, to draw upon my own ex-

perience and to suggest how color and brightness specifications may be intelligently written, let me set forth a series of elementary principles and simple observations.

For the most part, uniform luminosity in the field of view may be said to be tolerable where brightness ratios do not exceed 1 to 10. The task itself, however, may be black on white if it is relatively small in area. What must be avoided are severe contrasts in the major field of view (generally about 60 degrees surrounding the task—or 360 degrees if the worker constantly moves or looks about).

In the average industrial situation the employee works on dark materials such as metal, fabricating them on dark machines placed on dark, oily floors. If the plant is old, he may have 10 footcandles or less of illumination—hardly enough to see clearly.

A new lighting system may be installed, possibly to deliver about 25 or 30 footcandles. And to effect the most impressive light-meter readings, the walls may be painted white. The higher level of illumination will no doubt be welcome, but the white walls may promptly cause trouble.

It is not difficult to analyze the errors that might be made. If the white walls are showered with light, brightness ratios (in relation to materials, machines and floors) may jump way beyond 1 to 100. Various things will happen. The pupil of the eye may be constricted to reduce visibility of dark materials and machines. Vision may be blurred and real distress noted. Pupillary adjustments to the brightness of the wall (which is meaningless) will be accomplished in a matter of seconds. The dark materials and machines (which are important) will not be so easily fixated—the reverse action of seeing (from light to dark) being slow and time-consuming. It is easy to appreciate that the bodily reflexes of the worker may be retarded and his productive capacity cut down. And all despite more light and brighter surroundings.

It would be wrong, of course, to argue against higher levels of illumination. However, if they introduce glare and excessive brightness ratios, then it would be better to sacrifice a few extra footcandles for a more uniform and comfortable seeing condition. However, other devices may be brought into play to effect good illumination as well as good seeing.

In the situation described above, the glare of white walls could be offset if other areas and surfaces in the interior were also lifted in brightness. If the floor were concrete, for example, it could be scrubbed and bleached out with a caustic solution. Machinery and equipment could be painted lighter. This would be "lifting" the matter of brightness from the bottom up—an excellent and recommended practice. Yet if the floor were cedar block, oily and dark, the machinery color and the wall colors would have to be kept on the soft side.

Those who think in terms of illuminating efficiency are always campaigning for white and off-white colors on walls. Those who understand human vision and the psychological aspects of seeing will be inclined to favor fairly soft hues. And in my experience, greater justification would follow this latter viewpoint. Practically all experimental evidence has shown that a surrounding field *darker* than the brightness of the task is far superior to a lighter surround! The lighter surround will, in truth, tend to constrict the pupil and very seriously compete for attention. Thus it is safer to be on the deep side than on the light side in almost any application of color. Yet the principle involved will generally irk the lighting specialist; he would rather look to his light meters than to the eyes of human beings.

It is quite possible to set forth ideal brightness specifications for factory and office conditions. Ceilings—almost without exception—should be white. This will be essential to the efficiency of indirect lighting systems. In direct systems, the white overhead will reduce contrasts between fixtures and their surroundings. Being "neutral," white will also attract less psychological notice and hence prove non-distracting.

Upper walls (generally to a line level with the bottom of roof beams or trusses) should have a reflectance between 50 and 60 per cent (if floors and equipment are on the dark side) or between 60 and 70 per cent if most areas and surfaces in the interior are or can be made fairly light. The contention is personal with me, but I oppose wall brightnesses higher than 70 per cent. I have tried them and failed with them. They seem to be allowable only where the most perfect and modern lighting system is installed and accompanied by pale floors and equipment—or for unimportant spaces such as storage where critical seeing tasks are not performed.

If a dado is required to conceal stains, the color tone should reflect not less than 25 per cent and perhaps not more than 40 per cent. Floors should reflect at least 25 per cent if such is practical. Machines, equipment, desks should have a reflectance factor between 25 and 40 per cent, lighter when the floor is light and deeper where the floor is dark.

These ratios and percentages have been successfully applied in numerous plants and thus have the benefit of widespread trial and research.

Certain refinements are also to be introduced. Window sash ought to be white or a light tint to lessen contrast without outside brightnesses. Machinery may be highlighted in accordance with the principles of Du Pont Three-Dimensional Seeing to reflect more light at important parts and concentrate the attention of the worker. In numerous fine seeing tasks, background shields may be constructed (a) to reflect light and provide immediate contrast with materials, (b) to confine the vision of the worker and hold eye adjustments relatively stable, (c) to blank off shadows or movement in the distance, and (d) to give the worker a better sense of isolation. Normally such shields should cover from 45 degrees to 60 degrees of the visual field.

End-wall treatments in medium tones also have widespread application. Where most workers may be engaged at difficult eye tasks and may be so oriented as to face in the same direction, the wall ahead may be colored in a pleasing tint having a reflectance of from 25 to 40 per cent. The end wall will help to overcome an unfavorable constriction of the pupil. Upon glancing up, it will afford relaxation rather than the stimulation of glare. It will likewise relax the strain of prolonged convergence and be psychologically pleasing and restful. Here again is a principle widely and successfully employed in industry.

In the discussion so far presented it will be noted that chief emphasis has been placed on brightness rather than chromatic quality, or color. Good illumination and well balanced brightness ratios will be easy on the eyes whether the particular hues involved are pink or blue, yellow or green. Psychologically, however, color holds a fair amount of "magic," although any extravagant claims as to its therapy would be difficult to defend.

For industrial purposes, soft, delicately grayish hues are best. They are lacking in aggression, less distracting, and they most effectively conceal dust and soiling. Ordinarily, primitive colors such as blue and yellow are tiresome. Where subtlety exists (bluish-green, peach, etc.) a more comfortable environment

FIG. 1 — A dark machine forces trying eye adjustments. Contrasts are bad and the worker has difficulty in seeing clearly.

will be found and one that will "wear well" over prolonged periods.

It is logical to use "cool" colors such as green or blue where the working condition exposes the employee to relatively high temperatures. Conversely, "warm" tones of ivory, cream or peach are suitable to soften up a vaulty or chilly space and compensate for lack of natural light.

One very practical color will be found in a soft tone of blue-green. Given a reflectance factor between 50 and 60 per cent, it has universal possibilities for walls in factories, offices, schools, hospitals. It has an ideal brightness for average lighting conditions and will relieve glare as well as provide a neat sequence between light ceilings and average medium or deep tones on equipment and floors. Blue-green is a "cool" hue. It lacks monotony, having a bluish cast under daylight and a yellowish cast under artificial light. Best of all, it is the direct visual complement of human complexion. When the retina of the eye is saturated with it, a warm pinkish afterimage is produced which is flattering indeed to employees.

In working out color plans for industry, good colors (such as the above) and some amount of variety is recommended. Illumination and brightness should be supplemented by an intelligent consideration of color. Beauty becomes functional when it improves mental attitude and contributes to emotional and psychic gratification. It is futile to overlook psychological factors. Some authorities have been known to argue that nothing is wrong except that people are prej-

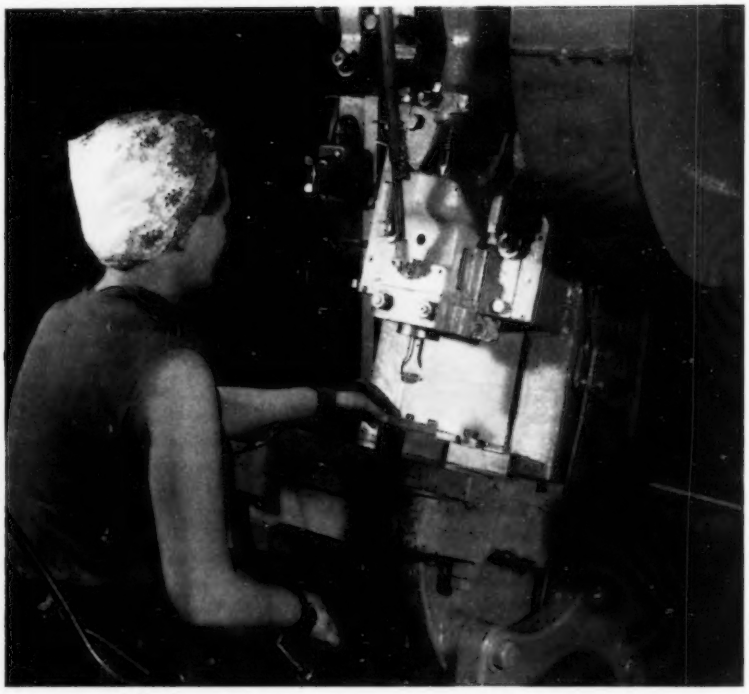


FIG. 2 — With the machine in a lighter color and with a shield placed directly back of the task, higher visibility and greater seeing comfort are achieved.

udiced. L. D. Morgan has stated, "As engineers we cannot shrug off complaints of bad lighting with the statement that the complaints are largely psychological. A patient or client with a psychosis suffers just as acutely as one with a physical injury." To the great credit of color is the fact that it appeals to all humans. It holds esthetic advantage and, added to good scientific practice in illumination and brightness engineering, it becomes the "crowning glory" of an intelligently and technically executed job.

In purely casual spaces, such as wash rooms, rest rooms, cafeterias, lighter and cleaner hues may be used. In view of average color preferences, blue becomes ideal for facilities devoted to men, and rose for facilities devoted to women. In stairwells and corridors, usually deprived of natural light, bright tones of yellow are effective. In storage areas, white is best and will make the most of existing lighting installations.

Where critical seeing tasks are performed, however, and where distractions are to be avoided, the best colors to use are soft variations of green, gray and blue. Large, vaulty spaces may be enlivened with ivory, cream or peach over all walls, or yellow over end walls. Gray machinery highlighted with buff on important parts and working areas will prove ceffctive. Medium gray is also ideal for unimportant elements such as bins, racks, shelving. One must remember that color is more compelling than neutrality. Hence, if it is strategically applied, it can make order out of chaos, distinguish important from unimportant things, and help the worker in his mental effort to concentrate on his job. In the-

FIG. 3 — View of industrial plant before rehabilitation with color. Dingy surroundings handicap vision, introduce hazards and lower the efficiency of the worker.

FIG. 4 — View after rehabilitation with color. With no change in lighting a safer and more cheerful environment is created. The worker is able to better his production.

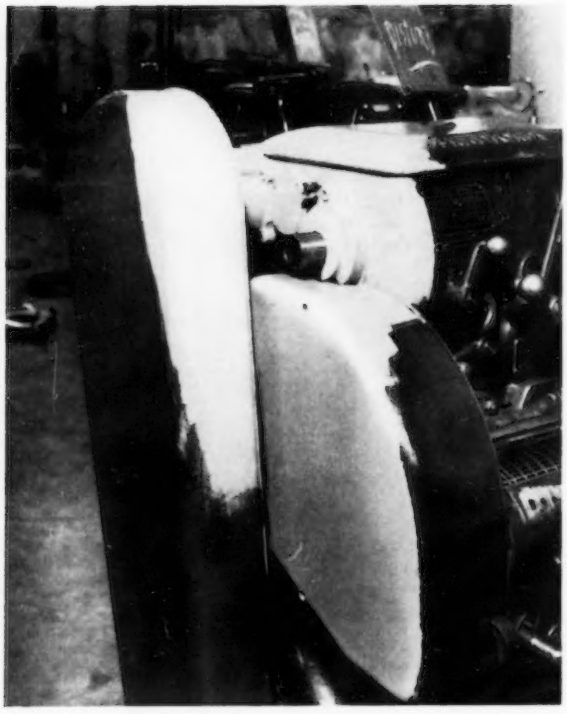


FIG. 5 — The simple application of a coat of paint creates higher levels of illumination, improves visibility, reduces severe contrasts, promotes safety and good housekeeping, and appeals to the worker.

ory as well as practice, the job of color is not so much to "pep" up the worker; too much of this attitude may lead to distractions and irrelevancies. To the contrary, color becomes integral with the task, not foreign to it. Improved efficiency and relief from fatigue become automatic because the human eye can see more easily, with less strain. Color is made to fit in rather than stand out. It contributes to better visibility and to an agreeable and cheerful frame of mind.

Finally, color serves a useful and functional purpose as a means of identification in safety practice. A well integrated code, developed by the author in cooperation with Du Pont and later accepted in part by the American Standards Association, has the following features.

Yellow (or yellow and black bands) becomes standard to mark strike-against, stumbling or falling hazards. It is painted on obstructions, low beams, dead ends, the edges of platforms and pits. Being the color of highest visibility in the spectrum it is conspicuous under all lighting conditions and well adapted to the above purposes.

Orange is standard for acute hazards likely to cut, crush, burn or shock the worker. It is painted around the edges of cutting machines and rollers. On the inside areas of machine guards and electric switch boxes, it "shouts loudly" when such devices are removed or left open.

Green is standard to identify first aid equipment, cabinets for stretchers, gas masks, medicines, and the like.

Red is reserved entirely and exclusively for the marking of fire protection

devices. It is painted on walls back of extinguishers, on floors to prevent obstruction, on valves and fittings for hose connections.

Blue is standard as a caution signal. The railroad industry employs it to mark cars which should not be moved. In factories it is placed as a symbol on equipment, elevators, machines, tanks, ovens, etc., cut down for repair. It may be used on switch control boxes as a silent and unobtrusive reminder for the worker to see that his machine is clear before he operates it.

White, gray or black are standard for traffic control and good housekeeping. They are used for aisle marks, painted on waste receptacles. White corners and baseboards may be used to discourage littering and to get the sweeper to dig into corners.

Color in industry has achieved remarkable results. Its use has been forced by improvements in lighting, by machine operations involving fine tolerances, by labor shortages and the need to conserve human energy. It has economic aspects as well as social ones, for it protects human welfare, improves human efficiency, and adds dignity and order to the working environment.

Good vision demands ample light and proper brightness contrast. Illumination and color thus become one and the same thing—meaningless without each other, but potent in the conservation and direction of human energies when perfectly coordinated.

# TRANSACTIONS

## AMERICAN ACADEMY
*of*
## OPHTHALMOLOGY
*and*
## OTOLARYNGOLOGY

Vol 51 No 4

MARCH - APRIL
*1947*

Published bimonthly, February, April, June, August, October, and December, by the Douglas Printing Company, 109 North 18th Street, Omaha 2, Nebraska, for the American Academy of Ophthalmology and Otolaryngology. Editorial and business office, 100 First Avenue Building, Rochester, Minnesota. Subscription price $3.00 yearly to members, $6.00 yearly to non-members. Entered as Second-Class Matter November 28, 1938, at the Post Office at Omaha, Neb., under Act of March 8, 1879.

ANNUAL MEETING OCTOBER 12-17, 1947, CHICAGO

# VITREOUS TRANSPLANTATION

NORMAN L. CUTLER, M.D.

WILMINGTON, DEL.

THE possibility of obtaining better vision in patients with vitreous opacities or hemorrhage has long interested ophthalmologists. In 1890 Ford[3] first withdrew vitreous from several human eyes. He reported improvement in vision in one patient from light perception to 20/60. Since that time many variations of this procedure have been used. The transplantation of animal vitreous to human eyes was first reported by Deutschman[2] in 1906. However, there is no record of the transplantation of human vitreous.

This report discusses the results obtained in the first 13 cases of vitreous transplantation. Three of these were reported in an earlier paper.[1]

Chemical analyses of the vitreous show that it contains approximately 99 per cent water yet it has a viscosity 20 to 200 times higher than water. Physically it may be described as a solgel, in an unstable state, subject to synaeresis under various conditions. Not as much is known about the relative importance of the 1 per cent of solids as about the importance of the physical nature of the vitreous. In certain operative procedures on the eye there is a disturbance of both; i.e., in loss of vitreous in a cataract extraction, and in release of fluid in a detachment operation. Certainly the eye may function in a satisfactory manner for a long period of time with both of those conditions in an abnormal state. How much these two conditions can be disturbed with satisfactory vision remaining is yet to be determined.

After synaeresis, an abnormal, or fluid vitreous, may be present in many instances. This situation is, of course, an exceedingly common occurrence in myopia and old age. It also occurs after injuries, hemorrhages, disease, detachment of the retina, etc.

The possibility of maintaining part of the vitreous in a gel state and part in a sol state is possible in many instances. The procedure of transplanting vitreous in the cases reported here was used to provide a clearer vitreous and to maintain the chemical composition of the vitreous rather than to restore entirely the normal physical properties to an already abnormal vitreous. In fact, the only standard used for donor vitreous (because of the scarcity of vitreous for transplantation) was that it be clear. It was felt that if the normal chemical composition could be maintained, then the vitreous fluid would not be irritating and there would be a normal physical balance. The fact that, in the eyes operated on, the tension returned to normal and remained there, suggests that this was accomplished.

The following is a summary of the previously published operative procedure with slight changes dictated by further experience.

## OPERATIVE PROCEDURE

With the patient under sodium pentothal anesthesia, the sclera is exposed in the selected quadrant. A 2 mm. incision is made through the sclera in the equatorial region with a cataract knife. Two 0000 silk mattress sutures are passed in

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

opposite directions across the incision and loose ties placed. An 18 gauge needle on a 5 cc. syringe is introduced into the vitreous chamber under direct observation by the assistant. If possible, 1.5 cc. of vitreous is withdrawn and the syringe detached. A syringe containing 2 cc. of clear vitreous from a recently enucleated eye and kept at room temperature is then attached to the needle. While two assistants exert tension on the knots of the two mattress sutures, the vitreous is injected until the eye has reformed. The needle is quickly withdrawn as the sutures are pulled up and the ties completed.

Atropine sulphate, 1 per cent solution, or ointment is instilled and a binocular dressing applied.

Postoperatively, mild sedatives are used as indicated. The patient is kept on his back for two days and then may be turned or elevated. He is allowed up the fourth or fifth day.

### RESULTS

Eight patients, including two already reported, have had vitreous transplants for vitreous hemorrhage. Of these four are considered successful, one partially successful and three failures. The failures were possibly due to the presence of detachments of the retina in each case, with intraocular foreign bodies in one.

### COMMENT

All patients were chosen for operation on the basis of an apparently normal light projection and a normal tension. Other factors which might unfavorably influence the result, such as, dense hemorrhage, the presence of bands in the vitreous or of foreign bodies were not considered. In some of these patients the vitreous was examined with a flat contact lens and a slit lamp. The normal vitreous appearance was not evident, nor was it necessarily expected to be.

In some of the patients, particularly No. 1 and No. 2, if the transplant had been done in the proper quadrant, possibly near-normal vision would have been obtained, because the clear vitreous was not over the macular area where it might have been.

### DETACHMENT OF THE RETINA

Five patients with detachment of the retina were operated on. All had had one or more unsuccessful standard diathermy operative procedures before it was decided to do a vitreous transplant along with a diathermy operation. All had detachments involving one-half or more of the retina.

In two of these patients the operations were entirely unsuccessful. In two there was a decrease in the area of the detachment, and in one, in which there was an almost complete detachment pre-operatively, there was a complete re-attachment.

### PROCEDURE

Anaesthesia: Local. The usual preparation and exposure of operating field of the eye for a retinal detachment operation was done. The incision and sutures for vitreous transplant were made and a needle, size 22, on a 5 cc. syringe, containing clear vitreous was introduced into the vitreous chamber under observation of the assistant. Six to twelve penetrating diathermy punctures over the chosen area for drainage were made with a La Carrere electrode. The vitreous was injected under pressure, the sub-retinal fluid being forced out like water through a sieve. The needle was quickly withdrawn and the sutures tied. With this size needle almost any desired tension could be obtained at the end of the operation. In some cases 35 Schiotz was recorded at the completion of the operation.

### COMMENT

In patient No. 1 there was a considerable period of time between the occur-

TABLE I.

| Patient | Pre-Operative Condition | Operation Date | Pre-Operative Vision | Postoperative Vision | Comment | Period of Post-Oper. Observation |
|---|---|---|---|---|---|---|
| 1. J. M. | Oct. 26, 1944 Vitreous hemorrhage traumatic | Nov. 3, 1945 | light perception | (16/60) 20/70 | Vitreous was continuing to clear, further improvement was expected. | 4 months |
| 2. R. C. G. | Feb. 1, 1945 Vitreous hemorrhage traumatic F. B. in eye | May 22, 1945 | light perception | 6/200 | Continuing slow improvement—the area of transplant was very clear. A second transplant should offer good prospects for improvement. | 15 months |
| 3. C. O. L. | Jan. 16, 1945 Spontaneous hemorrhage | July 18, 1945 | 20/200 | 20/25 | | 4 months |
| 4. W. S. | Aug. 29, 1944 Vitreous hemorrhage traumatic | April 12, 1945 | 20/300 | 20/25 | Many large floaters. A small amount of clear vitreous was injected. A second transplant would probably help. | 15 months |
| 5. I. V. H. | June, 1944 Spontaneous vitreous hemorrhage | Feb. 19, 1945 | light perception | 20/50—1 | Vitreous very clear—a macular lesion prevented better vision. | 8 months |
| 6. A. F. H. | Struck in L. E. by fist Dec. 25, 1944 Vitreous hemorrhage | Mar. 9, 1945 | light perception | light perception | Apparently had a detachment. | 3 months |
| 7. J. L. A. | Nov. 22, 1944 Vitreous hemorrhage. Retinal detachment. | Oct. 8, 1945 | light perception | light perception | No improvement. | 3 months |
| 8. L. E. S. | Densely organized hemorrhage | Aug. 7, 1945 | hand movement | C. F. | Fundus cleared very well but showed large detachment below. Retina was not re-attached by additional transplant and diathermy procedures. | 9 months |

TABLE II.

| Patient | Date of Detachment | Type of Detachment | No. of Previous Operation | Operation Date | Pre-Operative Vision | Post-Operative Vision | Period of Observation | Result |
|---|---|---|---|---|---|---|---|---|
| 1. G. L. Z. | Sept., 1944 | Spontaneous, 2 quadrants partly bullous. | 1 | Jan. 11, 1946 | 20/100 | H. M. | 4 months | Vitreous hemorrhage 2 weeks postoperative not absorbed—one quadrant still detached. |
| 2. L. E. S. | March, 1945 | Traumatic with vitreous hemorrhage, densely organized in 2 quadrants. | 1 | Feb. 11, 1946 | H. M. | H. M. | 2 months | Pt not cooperative. Bumped head while A. W. O. L. |
| 3. C. L. L. | Jan., 1944 | Traumatic, 2 quadrants. | 3 | Jan. 21, 1946 | 20/300 | 5/400 | 5 months | Detachment unchanged, macula detached. After another detachment operation and scleral resection retina came off completely. |
| 4. E. D. S. | Oct. 3, 1945 | Traumatic, 3 quadrants partly bullous. | 1 | Jan. 21, 1946 | 20/200 | 20/200 | 7 months | 2 quadrants remained off. |
| 5. S. M. A. | Dec. 18, 1944 | Spontaneous, partly bullous, all but about ¼ of one quadrant off. | 3 | Oct. 1, 1945 | C. F. | 20/80 | 4 months | Complete reattachment. |

rence of the detachment and the operative procedure. Patient No. 2, because of the strands in the vitreous, probably offered a poor prognosis. All attempts including scleral resection with patient No. 3, six in all, failed. Although patient No. 4 had a large detachment he seemed to be a suitable case. Patient No. 5 was the only successful operation.

The results of vitreous transplantation combined with a modified diathermy procedure are far from satisfactory. The cases operated on were not such as to properly evaluate this method.

## SUMMARY

Thirteen vitreous transplants are reported, eight for vitreous hemorrhage and five for detachment of the retina. Four of the former are considered successful, and one partially successful. One of the latter is considered successful. In all patients the tension returned to the pre-operative level. In the hemorrhagic cases no eye had less vision at the end of the procedure. In the detachments two eyes had less vision although one was expected to improve.

The improvement in vision in the successful hemorrhagic cases ranged from light perception to 20/50 — and from 20/300 to 20/25. Although but one of the five retinal detachments was successful all of these cases were considered difficult and had had retinal detachment operations previously.

## REFERENCES

1. Cutler, Norman L.: Transplantation of human vitreous, Arch. Ophth., vol. 35 (June) 1946.
2. Deutschmann, R.: Zur operativen Behandlung der Netzhautablosung, Klin. Monatsbl. f. Augenh., 4:364-370, 1906.
3. Ford, V.: Proposed surgical treatment of opaque vitreous, Lancet, 1:462-463 (March 1) 1890.

## DISCUSSION

FREDERICK C. CORDES, M.D., San Francisco, Calif.: As a civilian consultant in ophthalmology to the Surgeon General of the Army, it was my privilege to observe most of Dr. Cutler's cases, both pre- and postoperatively. In the first case it was of great importance to determine whether or not human vitreous could be transplanted successfully. From a surgical point of view the result was excellent, and but for a macular lesion, as Dr. Cutler has already pointed out, the vision would have been very materially improved.

Case 4 on the table was the third case to be operated on and it was probably the most spectacular. This patient was on the blind program and, having finished his orientation training, was ready to be sent to Avon, Connecticut, to Old Farms, for his advanced training. The restoration of vision from 20/300 to 20/25 in a period of six weeks was most dramatic, particularly after a period of almost complete blindness for eight months.

The result of vitreous transplant in retinal detachment cases has not been so good. However, in my opinion not enough work has been done on this to condemn the procedure. The procedure may offer very definite possibilities probably in carefully selected cases. It must be admitted, of course, that eight months, the longest time interval after vitreous hemorrhage, does not exclude the possibility of further spontaneous clearing. Most of us have seen cases in which as long as a year was necessary before complete absorption had occurred and in many of these cases vision was again restored to normal. On the other hand, we have also seen cases which at the end of that time had developed organization of the hemorrhage together with retinitis proliferans. Experience has shown that in these latter cases very little can be done once organization and retinitis proliferans has set in.

Of interest in these cases to me was the fact that for a period of several weeks the clouded vitreous immediately surrounding the implanted vitreous continued to clear rather rapidly, while the vitreous at the periphery away from this area showed no appreciable change.

Further experience will be necessary before we can determine whether or not the risk involved in transplanting vitreous is small enough to make it advisable to use it in case of vitreous hemorrhage, with the hope that it may prevent the organization that occurs in some of these cases. In addition it is going to be important to determine whether or not some method can be devised of preserving vitreous, so that it can be available on short notice in cases where its use may be indicated.

I feel that this paper must be considered as a preliminary report that will require further experience before the use of and indications for vitreous transplants can be evaluated. However, the work done by Dr. Cutler does prove that human vitreous can be transplanted and that it can be transplanted without regard to blood grouping.

Dr. Cutler is to be congratulated on this piece of very interesting work that required a good deal of courage to perform.

JOHN MCGAVIC, M.D., Phoenixville, Pa.: It requires courage to report the first results of a new operative procedure, especially one which appears at first glance as bold as this one. When a new operation is described one can be certain that the original technic will undergo modification, that the indications will later be better defined, and that there will be some resistance to the introduction of a new idea.

Ophthalmologists generally have such high regard for the vitreous that they avoid it whenever possible. We, therefore, owe Dr. Cutler our great thanks for doing the pioneer work in the transplantation of human vitreous.

Dr. Cutler did some experimental work in animals along this line some years ago. Those of us who have seen the eyes with which he has been dealing realize that they have been damaged globes which had more pathologic changes than the vitreous hemorrhage and detachment of the retina for which he has done the transplants. I have had the privilege of seeing several of these patients and have dealt with many similar patients at another Army General Hospital. I assure you that these eyes tax the ingenuity of any ophthalmologist. Therefore, let us not be too critical of the visual result in these cases, but examine the technic of the procedure, the potentialities of it, and the indications for using clear vitreous to supplant abnormal vitreous.

Dr. Cutler has pointed out that the blood groups of the donor are of no importance. We all know that the vitreous has no metabolism of its own and that it reflects chemical changes rather slowly. It is, therefore, rational to suppose that it could be transplanted.

In addition to the indications given by Dr. Cutler, we may find that aspiration of multiple small, nonmagnetic foreign bodies and aspiration of parasites from the vitreous will be carried out more often now that replacement of vitreous with vitreous has been proved feasible. I mention these because the incidence of foreign bodies and parasites has increased in the war

and they will be seen by many of you in the postwar years.

In describing the operative procedure, Dr. Cutler mentioned withdrawal and injection of the vitreous in the equatorial region of the eyeball. One might well consider using the pars plana, which is avascular, or relatively so, to lessen the danger of hemorrhage and detachment of the retina. While the lens will be close to the needle when the globe is collapsed there should be little danger of injuring the lens, since the needle will be directed posteriorly. When the retina is already detached it would seem advantageous to inject vitreous back toward it rather than through a new hole in the retina made by the injecting needle. The pressing out of aqueous through the diathermy holes would be more effective and perhaps more so if galvanic current rather than the diathermy were employed as these holes stay open better.

Dr. Cutler has pointed out that attention to placing the clear vitreous in the visual axis is important. In the cases of hemorrhage in the vitreous, it would be most helpful if we had some means of foretelling the fate of the blood in a given eye. We have all seen three types of reaction: complete absorption, partial absorption with retention of fluffy, cloud-like blood in the lower periphery of the vitreous over a very protracted period, and organization of blood into bands. The first two call for no operation. Perhaps if one could foretell that bands would form, and this may be due to concurrent inflammatory reaction, the transplantation could be done while the blood was still loose. After the bands have formed it is impossible to do anything more than clear the area between bands, and one may be afraid to do this lest the retina become detached by traction during temporary hypotony.

Spontaneous hemorrhage in the vitreous in young males does not seem to me to be an indication for this procedure. We saw a rather high percentage of patients with Eales' disease in the Army, and I cannot recall any who did not have spontaneous absorption with normal vision and normal fundus. I do not refer to patients who have sustained injury or to eyes complicated by perivasculitis, uveitis, or diabetes with hemorrhage into the vitreous. This procedure does not affect the underlying cause of the hemorrhage, which we do not know, and would not, therefore, present the recurrence of hemorrhage. It might result in less active absorption of blood when recurrence does take place.

Dr. Cutler reports discouraging results in patients with detachment of the retina. It should be reemphasized that these were severe detachments, complicated by other factors, including previous diathermy operation. I believe there is reason to be more optimistic about using vitreous instead of air or saline. The maintenance of normal intraocular pressure is very gratifying. One of Dr. Cutler's five patients had a spectacular result. The role of the vitreous in the production of so-called idiopathic detachment is very important, but its mechanism has not been fully clarified as yet. It will be interesting to note whether or not the foreign vitreous shrinks or remains detached from the native vitreous. We may learn as much about the mechanism of idiopathic detachments as we gain in therapy by this procedure.

For those who plan to employ this procedure the suggestion is made that it be tried on animals first. Vitreous is tricky to handle and only enough for one injection may be obtained from a donor globe. It would be embarrassing to be ready to inject donor vitreous and have something go wrong. Some of us at Valley Forge General Hospital used rabbits and utilized human eyes which had to be enucleated to develop our technic. We did not get beyond this, but we did find that we were glad to have practiced on animals and on eyes which had to be enucleated rather than to using it in clinical cases first.

We are indebted to Dr. Cutler for this fine presentation of another of his truly original pieces of clinical research. We hope that he will go ahead and show the chemical, physical and histologic pictures resulting from transplantation of vitreous.

JOHN M. MCLEAN, M.D., New York City: I should like to ask Dr. Cutler if he will enlarge a little further on the advantages of maintaining that 1 per cent of normal vitreous chemistry in the material which is put back into the eye or is put into the eye and its advantages as contrasted with saline or some other physiological solution.

The reason I ask is that some three years ago at New York Hospital we had an unusual opportunity to transfer vitreous, and I should mention that this was done at the suggestion of Dr. Bertrand, who was then president. The patient was a man with known optic atrophy, who had an injury to one eye with a large vitreous hemorrhage which would not absorb. His other eye was completely blind from his optic atrophy and had no light perception. In that patient we had an opportunity to transfer vitreous from one eye of a given patient to the other eye of the same patient, a rather unusual opportunity. It was done in this fashion: The vitreous was removed from the blind eye

first and replaced by a salt solution and then it was used to replace the bloody vitreous in the other eye, thereby eliminating the various potential difficulties of transfer from one individual to another. The result was not too satisfactory and in the follow-up the visual result was not at all satisfactory. In the follow-up of the state of the vitreous over a long period of time, the eye in which vitreous had been replaced by saline seemed to have clearer and more satisfactory media than the eye in which vitreous had been replaced by that same patient's clear vitreous. They were equally fluid. That is what makes me ask the question and wonder if we have adequate theoretical chemical reason in detachment cases to attempt to switch from saline in the vitreous, to restore the position of the retina, to vitreous in the vitreous to do so, because certainly when we pass normal human vitreous through a needle even once, much less twice, we have so thoroughly destroyed most of its physical properties.*

*Friedenwald, J. S. and Stiehler, R. D.: Arch. Ophth., 14:789-808, 1935.

D. K. PISCHEL, M.D., San Francisco, Calif.: I had the opportunity to examine two of the patients in whom Dr. Cutler had performed the operation of the vitreous transplant, both being retinal detachment patients. I examined the vitreous by means of a Koeppe flat contact glass and the slit-lamp. In one of these patients the vitreous cavity showed no signs of any vitreous structure. In other words, it was an optically empty space as the normal anterior chamber. I would suppose that the vitreous which had been injected as well as the original vitreous had become entirely degenerated and liquefied, so that the same condition prevailed throughout the whole vitreous cavity.

In the second case, a similar examination with the slit lamp and contact glass showed a small mass of fairly normal looking vitreous right behind the lens. The posterior two-thirds of the vitreous cavity, however, was optically empty, as had been true in the first case, suggesting again that liquefaction of the injected vitreous with a marked shrinkage of the original or native vitreous. Both eyes were absolutely quiet and showed no sign of irritation from this operation.

I would imagine, as Dr. McLean said, that any vitreous injected through a hypodermic needle would be considerably altered, and even if slightly gelatinous to start with, would be quite fluid after it had been injected.

The fact that the eyes stood the operation without any sign of inflammation is a very favorable sign, and would lead one to think that this rather radical sounding operation was not so radical after all and that we should all be justified in trying to use this new method whenever possible.

NORMAN L. CUTLER, M.D., Wilmington, Del.: I want to thank Dr. Cordes, Dr. McGavic, Dr. McLean and Dr. Pischel for their comments. Perhaps I should briefly say in answer to some of the questions that I do not know the answers. The withdrawal of vitreous and the injection of saline has, of course, been done by a great many surgeons. Zurnedden reported on approximately 300 cases, although his data was not detailed enough to give one a real lead as to the possibilities.

It was not my purpose in attempting these procedures to bring out the possibility of injecting vitreous as contrasted with injecting saline solution, but to see if vitreous could be injected and what the result would be. I am of the opinion that the 1 per cent of protein in the vitreous must be important. I do not know why and I do not think that we are in a position to say why. There are so many things that we do not know about the vitreous, except perhaps to be a little afraid of it, I feel that we should make the attempt to know more about it.

Dr. McLean spoke of the effect of passing vitreous through a needle as destroying its structure. I am sure we do not know really whether that is so. After all, the structure is ultra-microscopic. We do need to know what happens to cause vitreous to change from a sol gel state to a sol state.

With regard to the observations of Dr. Pischel, which we were very happy to have, the fact that the vitreous was injected and apparently became fluid was fairly evident to us and it was confirmed in the few cases that we were able to use a flat contact lens on. What the condition of the vitreous was before other vitreous was injected we do not know. It was certainly abnormal, and how fluid it was we could not determine. Whether we will be able to maintain the viscosity of the vitreous after subjecting it to some trauma, which is present in a transplantation procedure, remains to be determined. I was, of course, careful to point out that this was not our hope in this procedure, and, in fact, some of the vitreous which we injected was fluid at the time we injected it.

Thank you.

# THE EFFECT OF VISUAL TRAINING ON EXISTING MYOPIA

H. Rommel Hildreth, M.D., William H. Meinberg, M.D.,
Benjamin Milder, M.D., Lawrence T. Post, M.D.,
*and*
T. E. Sanders, M.D.
ST. LOUIS, MO.

This research was undertaken in an effort to determine the value of visual training as a means of improving the visual acuity in patients with myopia. During the past few years ophthalmologists have often been questioned by the parents of myopic children and by myopic individuals relative to the possibility of improving their sight, or of overcoming their near-sightedness, so that they might no longer have to wear glasses to see well at a distance. These queries have become so frequent that it seemed incumbent upon ophthalmologists to try to evaluate such methods of training as are employed by many optometrists and a few ophthalmologists.

The first efforts to improve vision by means other than glasses that received wide public attention were those advocated by the late W. H. Bates, M.D., who caught the public notice by means of a book entitled "Perfect Sight Without Glasses," published in 1920. This idea, so fascinating to the wearers of glasses and especially to those who were forced to use strong corrections, naturally had great appeal and his training program developed a considerable following. The fact that very few have been able to dispense with their glasses is sufficient evidence of the ineffectiveness of the method. The idea, however, was so attractive that it was seized upon and efforts made to find more satisfactory methods than the Bates training for accomplishing the same purpose.

Many different training technics have been studied and are employed. But most of them have a common background of thought that has been standardized to a considerable degree in some of the optometric schools of the United States. Variations in details are, of course, practiced by almost everyone who gives this type of training. For the purpose of this study a method that closely approaches the most frequently employed ideas was used and will be given in detail later in this article. Longer or shorter periods of training might have produced different results. Two of the most outstanding cases of improvement observed during this time were not treated in this group, but by a practitioner not engaged in this particular project.

Although visual training for myopia had been in use for a number of years by optometrists at the time of the inception of this program, there was no report found in the American ophthalmic literature of a series of cases of myopia in which such training had been done with careful ophthalmologic examinations. It is interesting that coincidental with our study a somewhat similar one was being carried out in Baltimore. This series of 103 patients has been reported by Alan Woods, M.D., in the American Journal of Ophthalmology for January,

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

From the Department of Ophthalmology, Washington University and the Oscar Johnson Institute.

Funds for this research were contributed by the American Optical Company, Aloe's Optical Company, and the Department of Ophthalmology, Washington University.

1946. Their work differed in that the entire training program was controlled by an independent group of optometrists whose detailed technic was not reported, although ophthalmologists of the Wilmer Institute examined the cases before and after training.

There are obviously innumerable phases to the problem of exercises in myopia, so it seemed best to attempt only a small part of the study in order that one phase at least could be comprehensively investigated; hence, the limitation to the study of the possibility of improving vision of myopic patients by training. Other pertinent questions are: How long does improvement, if any, continue? In what types of myopia is the training most effective? Does the treatment have any effect on the rate of increase in progressive myopia? What would be the effect on other refractive states, as hyperopia, astigmatism, and anisometropia? What is the mechanism by which improvement, if any, is brought about?

As the primary objective of the problem as outlined was the determination of the possibility of improvement of the visual acuity in the myopic patient, it was evident that the group of patients should be standardized as much as possible, eliminating variables and unfavorable conditions so as to obtain the maximum improvement in the myopic factor. Because of this, the following standards were decided on:

1. Age 10-30 years
2. Range of myopia in the greatest meridian -0.50 D. sphere to -3.00 D. sphere
3. Astigmatism of 1 diopter or less
4. No anisometropia of more than 1 diopter
5. Correctable visual acuity of at least 20/20 in each eye
6. No abnormal heterophoria, fusional defects, or substandard accommodation
7. No internal or external organic condition.

The actual investigation resolved itself into the following steps:

1. The preliminary ophthalmologic examination
2. The optometric survey
3. The actual visual training
4. The final ophthalmologic survey
5. The analysis of the results
6. The interpretation of the results with any possible conclusions.

In order to study the subject as intelligently as possible, it seemed wise to utilize the services of optometrists who were giving this type of training and of ophthalmologists who had not used these methods. Accordingly a team was formed for this study composed of ophthalmologists, optometrists, and opticians. The following arrangement of the team was decided upon: For the actual training, J. R. Bockhorst, O.D.; for the preliminary and final ophthalmologic surveys, H. Rommel Hildreth, M.D.; William H. Meinberg, M.D., Benjamin Milder, M.D., and T. E. Sanders, M.D.; and as a consulting group, E. C. Ebeling, O.D., Lawrence T. Post, M.D., and J. L. Stevenson. A full-time technician, Mrs. Harriet Thursby, did the majority of the actual training under the direct supervision of the optometrist.

### THE OPHTHALMOLOGIC EXAMINATION

The ophthalmologic examination was divided among several individuals, the conditions and the examiner remaining constant for all patients examined. On the first visit (T.E.S.) a short history was taken including familial tendencies toward myopia, the length of time that myopic correction had been worn, the date of the last change, and the frequency of change of correction as related to progression. A general external and ophthalmoscopic examination was then made. Finally, the uncorrected visual acuity at 20 feet was determined

with a Snellen letter chart. If the vision was less than 20/100, a shorter distance with the 20/100 letters was used. It was thought that this line would be more accurate than the 20/200 letter when used as a "walk-up," as it interpolates between 20/100 and 20/200.

Obviously the accurate determination of the visual acuity is the essential of the whole program. Theoretically, a chart that is variable with similar and unfamiliar characters, as the Landolt broken ring, is much more desirable as it eliminates memory and past experience. At the beginning of the series the Landolt ring was used along with the ordinary Snellen chart with constant illumination. Three different Snellen charts were used interchangeably to eliminate memory. It was found early that lack of correction between the Landolt and the Snellen charts was so great that it was difficult to interpret the results satisfactorily. The results with the Snellen chart alone were consistent and satisfactory. The findings were also consistent with those of the optometrist using a Projecto-Chart. On the first visit the optometric examination (J. R. B.) was also done, as it was considered necessary to do this before a cycloplegic was used. This procedure is given in detail later.

On the next visit (H.R.H.) homatropine 2% was instilled at 10-minute intervals for five times. Following this, a retinoscopy and a static refraction were done. The residual accommodation was then measured. At the third visit (B.M. and W.R.M.) a post-cycloplegic refraction was done and the ocular muscle balance examined, which included: (1) The near point of accommodation as measured with Prince's rule, (2) The near point of convergence, (3) The degree of fusion as found on the Synoptophore, (4) The lateral and vertical muscle balance at 20 feet and 16 inches as determined with prism dissociation, and (5) The lateral and vertical fusional amplitudes as determined with rotary prisms.

Following this preliminary examination, the patient was then referred for visual training (J.R.B.) as outlined hereafter.

After completion of the visual training the above ophthalmologic examinations were repeated exactly. At this time the individual patients were questioned as to their ideas of the value of the training and their opinion as to any subjective improvement in vision.

### OPTOMETRIC EXAMINATION ROUTINE

The optometrist giving the training submitted a very detailed report on the examination routine and training technic he used throughout the program. This is available in the files of the Oscar Johnson Institute for those desiring to refer to it. A brief outline of this routine is as follows:

Steps 1 and 2. Visual acuity at 20 feet without correction and with habitually worn glasses

Steps 3 and 4. Near vision with and without glasses

Step 5. Measurement of interpupillary distance

Step 6. Ophthalmoscopic examination

Step 7. Ophthalmometric measurements

Steps 8 and 9. Phoria test at 20 feet and 16 inches with habitually worn glasses. A 6 degree prism was placed base up and the horizontal phoria measured by the Risley rotary prism.

Step 10. Static retinoscopy at 20 feet

Step 11. Dynamic retinoscopy at 20 inches

Step 12. Dynamic retinoscopy at 40 inches

Step 13. Subjective refraction test at 20 feet

Step 14. Horizontal phoria at 20 feet with correction found in Step 13

Steps 15 and 16. Adduction and convergence at 20 feet
Step 17. Abduction at 20 feet
Step 18. Vertical phoria test at 20 feet
Step 19. Horizontal phoria at 16 inches with correction found in Step 13
Steps 20, 21, 22, and 23. Listed as monocular and binocular cross-cylinder tests at 16 inches. A cross-cylinder was placed before each eye and a grid-like target observed. Spherical lenses were changed until the vertical and horizontal lines on the grid appeared alike.
Steps 24 and 25. Positive and negative fusional reserve relative convergence at 16 inches
Step 26. Vertical phoria at 16 inches
Step 27. Amplitude of accommodation
Steps 28 and 29. Positive and negative relative accommodative reserve

The detailed report ends with the sentence, "The twenty-ninth step completes the tests used and provides the basis for diagnosis of the subject's functional visual problem."

### TECHNIC OF VISUAL TRAINING

The procedure outlined here required the use of an Arneson Squint Corrector, a Keystone Tel-Eye Trainer with certain Keystone cards whose numbers are given later, and an American Optical Company Wells Head. The technic was broken into eleven individual steps laid out for a training period of twenty-four days averaging about one hour per day.

*First Sequence, Six Days of Training*—Step 1. Subject was seated before the Arneson Squint Corrector approximately 20 inches from the instrument. The instrument was equipped with a large disc with a medium-sized fixation target that could be rotated clockwise and counter-clockwise. The right eye was occluded with a patch. The instrument was started rotating clockwise with instructions to the subject to fixate constantly the medium-sized target as it moved in a circular fashion. While fixating the target, his attention was also directed to the referential background of the disc which consisted of a black, red, and blue "E." The disc was rotated for three minutes. After the three-minute period the instrument was reversed to a counter-clockwise direction, the whole procedure being repeated for three minutes. The same procedure was repeated occluding the left eye. Total working time for each eye was six minutes.

Step 2. Subject was placed before the Tel-Eye Trainer. The instrument was set at infinity position on the graduated slide, using Rotor 4 giving a four-second flash. No lenses were used. Two sets of cards were used, different sets on alternate days. The sight of the right eye was blanked out by manual push-button control and the left eye only was permitted to view the card, BO1-EC1. There were numbers interspersed throughout the field of the picture and the subject was instructed to locate the numbers in sequence from 1 to 10. If the picture or numbers appeared blurred, no changes or alterations were made in the setting of the instrument. The subject was instructed to keep watching the card until the numbers began to clear. This training was continued for five minutes. For training of the right eye the instrument setting remained the same with the exception that the left eye was now blanked out and Card BO1-EC3 inserted. This card was the same as the first with the exception that the numbers were in different positions throughout the field. On alternate days Cards AN18 and AN 16 were used in the same manner. Instructions in procedure remained the same. Step 2 was all monocular training.

Step 3. Subject remained at the Tel-Eye Trainer. No lenses were used. Slide

remained at infinity and Rotor 4 was used. Both eyes were uncovered and for five minutes the Card BU5 was used with the subject seeing a picture of a room with the right eye in black and white, the left eye viewing a solid red field with numbers scattered through the field from 1 to 7. Instructions were to fuse the two unlike backgrounds into a single picture with the subject locating the black numbers and attempting to maintain clearness. When blur was reported, the setting of the instrument remained the same with emphasis placed on the subject's concentration to clear the numbers in spite of blur. Card BU5 was removed and Card BU6 inserted. The subject now viewed the black and white background picture in front of the left eye with a solid red field in front of the right eye, with numbers interspersed in the background from 1 to 8. The same instructions were given to the subject in viewing this card as in Card BU5. The procedure covered five minutes. Step 3 was monocular training in binocularity. Total training time for Step 3 was ten minutes.

Step 4. Subject remained at the Tel-Eye Trainer. The instrument was set to flash alternately using Rotor 3 (three-second flash). Cards BO1 - EC2 and BO1-EC4 were used. These cards were the same as the cards noted in the second step with the exception that the numbers were scattered in new positions. Instructions to the subject were to clear the numbers and the picture alternately with each eye. The second series of cards used in Step 4 were the AN1 and AN3. The cards of series one and two were used on alternate days. The AN1 card was a yellow background card with numbers arranged in a circle and a start figure in the center connecting the numbers. When the alternating flash was started, the subject located the number 1 with the right eye, the number 2 with the left eye, number 3 with the right eye, number 4 with the left eye, etc., until he had gone through twelve numbers. Procedure was repeated for five minutes. The AN3 card was a similar card with these exceptions; the background of the card was now white with considerably smaller numbers. In addition to locating the numbers alternately, the patient was instructed to see the numbers clearly. Step 4 was an alternating monocular training procedure.

The above four steps were repeated for six days. Beginning the seventh day Step 5 was started, all of the previous steps being discontinued.

*Second Sequence, Six Days of Training*—Step 5. Subject was placed before the Arneson Squint Corrector, disc approximately 20 inches from the subject. A vertical dissociating prism, 6 degrees base up, was placed before the right eye. A vertical displacing prism, 4 degrees base down, was placed before the left eye. The subject was instructed to see two targets constantly. The instrument was turned on, rotating clockwise, with instructions to concentrate attention on the top target for 10 to 15 turns, always being aware of the lower target. Attention was then directed to the lower target as the fixating target for 10 to 15 turns with instructions to the subject always to be aware of the upper target. The procedure was followed for five minutes. The change in the prism setting was as follows: a 4 degree prism base down was now placed before the right eye and a 6 degree prism base up was placed before the left eye with the same instructions followed for five minutes in the viewing of the two targets. Step 5 was a rotation exercise, monocularly, in binocularity.

Step 6. Subject was placed before the Tel-Eye Trainer. The instrument was set at infinity on the slide, using Rotor 2 (two-second flash). Both eyes were uncovered. Cards BU8 and BU7 were used

in the slide holder. When viewing Card BU8, the subject had a black and white picture in front of the left eye and a red and blue background in front of the right eye with numbers scattered throughout the red and blue field from 1 to 8. The color arrangement of the picture in front of the right eye was that the top half was red, the lower half, blue. When the light flashed on, the black and white picture and the red and blue backgrounds had to be fused into one. The numbers scattered in the red and blue fields had to be clear. Procedure was followed for five minutes. Then Card BU7 was inserted into the instrument, which card was exactly reversed as compared to Card BU8. The black and white picture appeared in front of the right eye, the red and blue background appeared in front of the left eye. Numbers ranged from 1 to 9 in the colored field. The same instructions to clear numbers were given for this card. Procedure was followed for five minutes. Step 6 was monocular training in binocularity.

Step 7. Subject remained before the Tel-Eye Trainer instrument. Slide remained set at infinity, using Rotor 8 which gave alternate flash followed by binocular flash. Cards placed in the instrument card holder were ST2, Series I. This series consisted of three cards that produced stereoscopic vision and had control marks located in this sequence: control marks on Card 1 were set towards the left side; control marks on Card 2 were located centrally; and control marks on Card 3 were located to the right. The subject was instructed to see the pictures clearly and to fuse the control marks into a perfect cross. Each card was left before the subject for a period of five minutes. On alternate days ST2, Series II, were used with the same instructions to the patient as covered in the Series I. Step 7 was in stereoscopic development and binocular control.

Step 8. Subject remained seated before the Tel-Eye Trainer. Slide holder was moved into the 2.50 position. Light flash, using Rotor 7, was set to operate simultaneously before both eyes. Cards SST2 were placed in the card holder. The series used were the cards numbered 0115, 0118, and 0113. Each card was viewed for five minutes. Subject's instructions were to fuse the cards into a single clear picture with emphasis being placed on his ability to produce a perfect cross of the control marks which appeared first to the left, next to the center, and lastly to the right. Step 8 was training in near point stereopsis and binocular control.

*Third Sequence, Twelve Days of Training*—Step 9. After Steps 5, 6, 7, and 8 had been repeated for six days, a re-examination was made after which the third phase of the visual training was instituted. Step 9 was the repetition of Step 7.

Step 10. Repetition of Step 8.

Step 11. Part I. Subject was placed before the Wells Head. Steady light was used. Cards were placed at 4 inches and an auxiliary pair of plus 10 lenses was put in the instrument. Rotary prisms were placed before the subject's eyes. This setting produced a stereoscopic effect. At the zero markings subject looked at Card E2 with instructions to see the object with clearness and a sense of depth. Subject received frequent rest periods by closing eyes for approximately five seconds at a time. The rotary prisms were turned base out 2 degrees at a time with the caution to the subject that the card must remain clear and single. The strength of the rotary prisms was constantly increased in these 2 degree base out jumps until the subject could maintain single and

clear vision through a total of 32 degrees base out, 16 degrees over each eye. Training on this card was performed for five minutes. For five minutes Card C4 was placed before the patient. Rotary prisms were started at 0, again increased 2 degrees base out at a time until a total of 32 degrees prism base out was reached, 16 degrees over each eye, with the same instructions that the object must appear clear and single. Card 6CM was then inserted. Rotaries were placed at 0 and increased in 2 degree steps until a total of 32 degrees prism base out, 16 degrees over each eye, had been reached with the same instructions to the patient that the target must be clear and single. Total training time was fifteen minutes.

Part II. Subject was placed before the Wells Head. A 6 degree prism base in was placed in the instrument. The target viewed first was a type approximating Jaeger 3. The target was set at the 16-inch mark on the reading rod. The instructions to the subject were that the print must remain clear and single at all times. Then plus lenses were introduced in quarter diopter steps with the instruction to the subject as the lens was changed that again the print must remain clear and single. At the first increase of plus in the quarter diopter steps that produced a very slight blur, the subject was told to attempt to sharpen the print. When it became impossible for the subject to improve vision with increase in plus, the 6 degree prism base in in the rotaries was reduced 2 degrees at a time until the prisms were back to 0. At this time alternate size types of print were used varying between the original Jaeger 3 down to Jaeger 1.

The above steps, 9, 10, and 11, were continued for twelve visits. Again the subject was examined. Only those patients that appeared to have the ability to accomplish any further results in the above handling were permitted to repeat for an additional twelve visits.

Steps 9, 10, and Part I of 11 were used in those cases that appeared to be a basic problem of faulty convergence ability.

Steps 9, 10, and Part II of 11 were used in those cases that appeared to be a basic problem of faulty accommodative faculty.

## RESULTS

This study represents a series of 87 patients examined, but a number of others were refused because of obvious defects. Thirty-three cases were either rejected or incomplete so that 54 case records were available for study. The majority of these patients were obtained by referral from the private practices of a number of St. Louis ophthalmologists, but some were referred by interested local optometrists. The series was not larger because of the strict limitations listed above.

As expected, no change was noted in the external or ophthalmoscopic examination of any patient. In no case was there any significant difference in the cycloplegic refractions done before and after the period of visual training. There was no more than 1 diopter of residual accommodation under cycloplegia remaining in any instance.

There was no appreciable effect of the visual training on the near point of convergence, near point of accommodation, or the fusional ability. About 50 per cent of the cases showed an increase in esophoria of at least 5 prism diopters, or a decrease in the exophoria of about the same amount. In almost one-half of the cases there was an increase of at least 10 prism diopters in adduction (positive fusional amplitude). There was no apparent relationship between the amount of the myopia and the effect of the visual training on the muscle balance. There seemed to be no

relationship between the induced changes in the muscle balance and the final visual result.

As our patients had no, or only small amounts of, astigmatism and of anisometropia, the refractive error of the fellow eyes of all of our patients were quite similar. Because of this, the average spherical equivalent of each patient based on his homatropine refraction could easily be determined. On this basis we were able to divide our patients into three groups: Group I, those patients with average myopic errors of 1 diopter or less; Group II, those patients with average myopia from -1.25 sphere to -2.00 sphere; and Group III, those with average errors from -2.25 sphere to -3.00 sphere. It was also noticed that rather than try to analyze changes in the vision of separate "eyes," it was much more satisfactory to tabulate the binocular vision, particularly as this is the actual useful vision of the patient. Table I shows the pre- and post-training acuities of all the 54 patients, each patient being represented by his spherical equivalent.

The problem of how to evaluate an actual change in visual acuity on an accurate quantitative basis is extremely difficult. The simplest method is merely the number of lines improvement as measured on the ordinary Snellen chart. This presupposes that the difference between each line is the same, which is obviously untrue. Another method, which Woods used in evaluating his results, determines any improvement in terms of percentage points. This is done by determining the difference in the percentages of visual efficiency of the pre- and post-training visions, using the tables developed in industrial ophthalmology for evaluation of losses in visual efficiency following injury. As Woods points out, this weighs the results in favor of the higher degrees of myopia in as much as a case with improvement from 20/200 to 20/100, or one line, shows an increase of 28 percentage points in contrast to a case with improvement from 20/50 to 20/20 (four lines) which totals 22 percentage points. Obviously, this is not valid either clinically or statistically.

After studying our cases, it was evident that the easiest method was a simple qualitative one. Thirty patients, or 55.5 per cent, showed no change in the visual acuity following the course of visual training. No patient had a reduction in vision. Twelve patients, or 22.2 per cent, showed a questionable improvement which consisted of a change of one line or less in the acuity. It was thought that this improvement was meaningless as it was within the limits of error. This is especially true since the patients during the period of training did not wear their ordinary corrections, thus giving them experience in the judgment of blurred images. Twelve, or 22.2 per cent, of the patients showed definite improvement of vision. These results in relation to the three separate groups are shown in Table II. Using the visual efficiency table given in Snell's Medico-Legal Ophthalmology, the improvement in percentage of visual acuity was found to average 27 per cent in the favorable cases.

Group III, those with an average myopia of -2.25 sphere to -3.00 sphere, consisted of twenty-two patients. Of these only three, or 13.6 per cent, showed any appreciable improvement. There were six with some change and thirteen with no change. The three cases representing improvement improved from 15/100 to 20/70, 20/100 to 20/50, and 12/100 to 20/50. This represents an average improvement of 35.7 percentage points.

Group II, consisting of patients with average myopia from -1.25 sphere to -2.00 sphere included twenty-three cases. No change was noted in fifteen

**TABLE I.**
**Pre- and Post-Training Visual Acuity,**
**The Patients Being Represented by Their Spherical Equivalent**

| | 5/100 | 10/100 | 12/100 | 15/100 | 20/100 | 20/70 | 20/50 | 20/40 | 20/30 | 20/25 | 20/20 | TOTAL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 5/100 | | —2.50 | | | | | | | | | | 1 |
| 10/100 | | —3.00<br>—2.75 (3) | | —2.50<br>—3.00 | | | | | | | | 6 |
| 12/100 | | | | | —2.25 | | —2.50 | | | | | 2 |
| 15/100 | | | | —2.50<br>—2.75 | —1.75 | —2.25 | | | | | | 4 |
| 20/100 | | | | | —3.00<br>—2.75<br>—2.50<br>—2.00<br>—1.75 (2) | —3.00 | —2.50 | | | | | 8 |
| 20/70 | | | | | | —3.00<br>—2.25 (2)<br>—2.00<br>—1.75 (2)<br>—1.50 (3) | —2.25 | —2.00<br>—1.50<br>—1.25<br>—1.00 | | | | 14 |
| 20/50 | | | | | | | —2.25<br>—2.00<br>—1.50<br>—1.25 | —0.75 | —1.25 | | | 6 |
| 20/40 | | | | | | | | —1.25 (3)<br>—1.00<br>—0.75 | —1.50<br>—1.25 (2)<br>—0.50 | —1.00<br>—0.75 | | 11 |
| 20/30 | | | | | | | | | | | —0.75<br>—0.50 | 2 |
| 20/20 | | | | | | | | | | | | 0 |
| TOTAL | 0 | 5 | 0 | 4 | 8 | 11 | 7 | 10 | 5 | 2 | 2 | 54 |

**TABLE II.**
**Distribution of All Cases.**

| | Group I. | Group II. | Group III. | TOTAL |
|---|---|---|---|---|
| | Myopia —1.00 Sp. and under | Myopia —1.25 Sp. to —2.00 Sp. | Myopia —2.25 Sp. to —3.00 Sp. | |
| No Change | 2 cases<br>22% of Group I. | 15 cases<br>65.2% of Group II. | 13 cases<br>54% of Group III. | 30 cases<br>55% of all cases |
| Questionable Change | 2 cases<br>22% of Group I. | 4 cases<br>17.4% of Group II. | 6 cases<br>27.2% of Group III. | 12 cases<br>22.2% of all cases |
| Improvement | 5 cases<br>55% of Group I.<br>Average Change—12.1% | 4 cases<br>17.4% of Group II.<br>Average Change—18.7% | 3 cases<br>13.6% of Group III.<br>Average Change—35.7% | 12 cases<br>22.2% of all cases<br>Average Change—27.1% |
| TOTAL | 9 cases<br>16.6% of all cases | 23 cases<br>42.6% of all cases | 22 cases<br>40.7% of all cases | 54 cases<br>100% |

**TABLE III.**
**Late Visual Acuities.**

| Group | Patient | Spherical Equivalent | Pre-Training Acuity | Post-Training Acuity | Interval Months | Final Acuity | Result |
|---|---|---|---|---|---|---|---|
| GROUP I. | 1 | —0.75 | 20/30+3 | 20/20—4 | 15 | 20/25—2 | Unchanged |
| | 2 | —0.50 | 20/30 | 20/20 | 16 | 20/20—3 | Retained Improvement |
| | 3 | —0.75 | 20/40—2 | 20/25—3 | 16 | 20/25—3 | Retained Improvement |
| | 4 | —1.00 | 20/40+1 | 20/25—1 | 19 | 20/20—3 | Retained Improvement |
| | 5 | —1.00 | 20/70 | 20/40—3 | 15 | 20/40+3 | Retained Improvement |
| GROUP II. | 6 | —1.25 | 20/50+2 | 20/30+3 | 16 | 20/40—2 | Unchanged |
| | 7 | —1.50 | 20/70+1 | 20/40+1 | 17 | 20/70+ | Unchanged |
| | 8 | —2.00 | 20/70 | 20/40+3 | 16 | 20/50+3 | Partially Improved |
| GROUP III. | 9 | —2.25 | 15/100 | 20/70—1 | 23 | 10/100 | Worse |
| | 10 | —2.50 | 12/100 | 20/50—1 | 22 | 20/50+2 | Retained Improvement |
| | 11 | —2.50 | 20/100 | 20/50—3 | 18 | 20/70+1 | Partially Improved |

and questionable changes in four. Four, or 17.4 per cent, showed real improvement averaging 18.7 percentage points. These included three with change from 20/70 to 20/40, and one with improvement from 20/50 to 20/30.

Group I consists of nine patients with a myopic error of 1 diopter or less. Two each showed no change and questionable improvement. Five, or 55 per cent, of these patients with small errors showed definite improvement with an average improvement of only 12.1 percentage points. Two of these patients showed improvement from 20/30 to 20/20, these being the only ones with improvement to 20/20. Two showed change from 20/40 to 20/25 and one 20/70 to 20/40.

A late recheck of the visual acuity was possible in 11 of the 12 cases showing improvement, the interval between the post-training acuity and the final acuity ranging from 15 months to 23 months. These results are found in Table III. It will be noted that five (Nos. 2, 3, 4, 5, and 10) retained all of their improvement, while three (Nos. 1, 6, and 7) reverted to their pre-training acuity. Two (Nos. 8 and 11) retained a slight improvement, while only one (No. 9) lost vision over the pre-training acuity.

## CONCLUSIONS

In the first place, we realized before undertaking this problem that, no matter what our results, they would probably be criticized by both proponents and opponents of this controversial subject. If our percentage of improvement was not high enough to please the proponents, it would be charged that our technic of training was inadequate, omitting a critical or essential step. On the other hand, those who doubt that visual acuity can be altered by such training will say that the methods of taking the vision were at fault and that removing a person's glasses will improve his unaided vision within a period of weeks without training.

Although different technics or a larger and more varied series of cases might alter our exact figures, we believe that study of these results shows that vision in certain myopic individuals can be improved by training. Even if we ignore the figures of the whole group showing improvement in 22.2 per cent of our patients averaging 27.1 percentage points, this contention can be proved by the results in individual patients.

It is surprising how closely these figures compare with those of Woods. His favorable group, consisting of those patients showing a consistent improvement of each eye of at least ten points, makes up 29 per cent of his entire series compared to our figure of 22.2 per cent showing definite improvement. It is interesting that the average improvement in the favorable group in each series is 27 per cent.

As might be expected, the best results occurred in the cases of low myopia of 1 diopter and under (55 per cent improvement). There is less defect to be overcome in these and the better initial vision would probably make training easier. Although the series is not large enough to give accurate figures, the results of Group II (17.4 per cent improvement) and of Group III (13.6 per cent improvement) are quite similar. As Woods was unable to divide his cases on a basis of amount of myopia, these results cannot be compared.

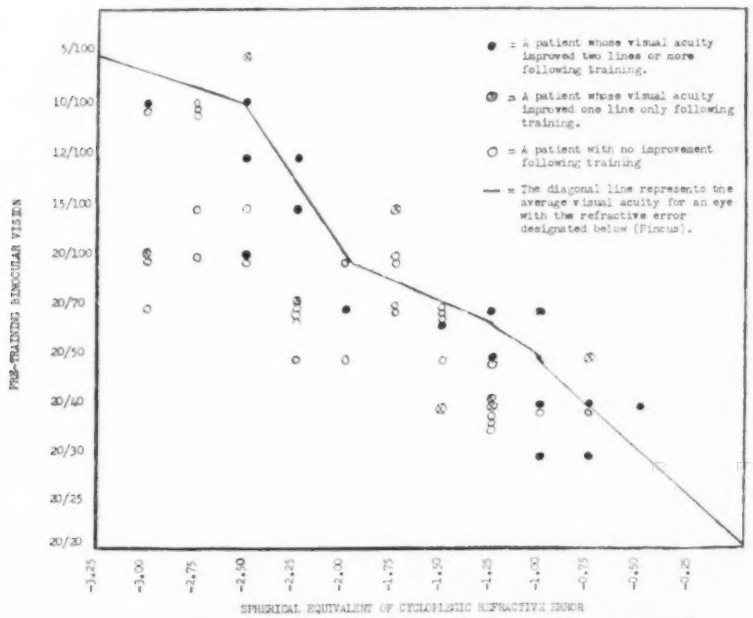
On first study there seemed to be little direct evidence from our results as to the exact mechanism of the visual improvement. It is apparent that there is no change in the amount of the refractive error. There is also no indication of significant change in the ocular neuromuscular mechanism, such as might give rise by relaxation of the lens to a "negative accommodation." The

impression gained from these patients was that much of the improvement occurred at the cerebral level rather than in the retina. Although there was no method for absolute evaluation, it seemed evident that the most visual improvement occurred in those patients who were most intent in their training, putting into it more actual effort and concentration, and vice versa. It also seems significant that many of the patients whose visual acuity did not change, thought that they could see better after the training. Also the subjective impression of improvement in the majority of the patients was out of proportion to the measurable change. Many stated that they thought they could "use" their eyes better.

One of us (B. M.) in analyzing the results made a significant observation that may well explain the benefit of training and may give the refractionist a criterion for selection of cases for training. It was noted that the patients with subnormal pre-training acuity seemed to get the best results.

Recently Pincus has reported observations of 50,000 examinations on eyes free of organic defects and correctable to 20/20. He correlated the unaided visual acuities with the refractive errors, including myopia, hyperopia, and astigmatism, in amounts up to 6 diopters. All of our cases were grouped according to their spherical equivalent and their pre-training acuity. Using the Pincus figures as a norm, it was found that in the 11 patients with an acuity of less than expected for their refractive errors, 8 of these cases (73 per cent) showed some improvement. In the

TABLE IV




The diagonal line represents the average visual acuity for an eye with the refractive error designated below (Pincus).

Above the solid line each patient has a visual acuity less than normal for his refractive error. Of these patients 8 of 11 (73%) showed some improvement.

Below the solid line each patient has a visual acuity greater than normal for his refractive error. Of these patients 16 of 43 (37%) showed some improvement.

43 patients having an acuity better than expected, only 16, or 37 per cent, showed improvement. This is expressed graphically in Table IV.

Realizing that our series was somewhat small, the same one of us subjected Woods' series of cases to a similar analysis, using only those cases that fulfilled our limitations. Since Woods did not report binocular vision, the analysis was done on individual eyes, 110 being available. In the group with pre-training vision worse than the norm, the percentage of improvement was 76.4 per cent, while in the group with better vision than the norm, improvement was 38.8 per cent. This again shows the remarkable similarity between our results and those of Woods. This also definitely confirms these observations.

Since the great preponderance of improved patients were those whose acuity was not as good as was to have been expected for the amount of their myopia, we may conclude that in these there had been a failure to utilize their possibilities to the best advantage and that training had improved the neurologic reception of the images. Better vision is not obtained by changing the anatomy, which remains stable, but by stimulating the visual effort and by improving the interpretation of the images seen.

It is probable that similar training would, in manner, benefit some hyperopic patients.

The results of the late re-examination, about one and one-half years after training, may have significance. This showed that approximately one-half of the patients retained their visual improvement, while only one patient became worse than pre-training acuity. This would indicate that, at least in a small number of patients, the effect of training is not transitory, although an investigation of the possible relationship of visual training to progression of myopia is indicated. This is without doubt the most important problem in myopia at present and is much more urgent than that of the improvement of visual acuity. Obviously, this question could be definitely answered only after a carefully controlled study over a period of years.

We must conclude that visual training has a definite but limited value in about one-fourth of selected myopic patients, and none in the remainder. Good effects are preponderantly in those whose vision is less than expected for their known myopia. Considering the time and effort expected by the patient, the results, both in percentage of patients improved and in the actual amount of improvement per patient, must be definitely increased if the procedure is to be generally applicable to the routine management of myopia. It would seem desirable that some efforts be made to increase the effectiveness of the present methods of visual training.

In conclusion we believe that visual training merits further study from the ophthalmologist, particularly in relation to progression in myopia. It should not be left solely to the optometrist and to the psychologist as has been almost the case in the past.

SUMMARY

1. In an attempt to improve the visual acuity, a series of 54 selected cases of myopia was given a course of visual training which was preceded and followed by a complete ophthalmologic examination.
2. The technic of training, which was based on standard accepted procedures in this field, was given by an optometrist while the pre- and post-training examinations were made by a group of ophthalmologists.
3. Thirty, or 55.5 per cent, of the cases showed no change in their acuity, while twelve, or 22.2 per cent,

showed a definite improvement; the best results being obtained in the cases with small amounts of myopia. Twelve patients, that is 22.2 per cent, showed a change so slight as to be excluded in the results. The change in the improved group averaged 27 per cent. Eleven of the twelve improved patients were rechecked at an interval of 15 to 23 months following completion of training. Five of these retained their improvement, while only one was worse than pre-training acuity.

4. There was no change in the refractive error, nor significant alteration in the ocular neuromuscular mechanism. It was thought that the improvement probably occurred because of an improved reception due to stimulation of the visual effort, as the best results occurred in those patients with a pre-training acuity less than would be expected from their refractive error since 73 per cent of this type improved.
5. Visual training has a definite, but limited, value in some myopic patients, preponderantly in those whose vision does not correspond with their known myopia. The effectiveness of visual training must be increased if it is to be generally applicable. Visual training merits further study from the ophthalmologist particularly in relation to progress in myopia.

The optometrists associated with this project concur fully with the results, interpretations, and conclusions.

## BIBLIOGRAGHY

Pincus, M.H.: Unaided visual acuities correlated with refractive errors, Am. J. Ophth., 29:853-858 (July) 1946.

Woods, A. C.: Report from the Wilmer Institute on the results obtained in the treatment of myopia by visual training, Am. J. Ophth., 29:28-57 (Jan.) 1946.

Snell, A. C.: A. Treatise on Mediocolegal Ophthalmology, The C. V. Mosby Company, St. Louis, 1940, pp. 312.

## DISCUSSION

ALAN C. WOODS, M.D., Baltimore, Md.: Drs. Hildreth, Post and their collaborators deserve the thanks of this society for their meticulous investigation of this plaguing and perplexing problem of the value of eye exercises in the treatment of myopia. None except the five who have labored on such an investigation can appreciate the endless and tiresome labor entailed. I congratulate Dr. Hildreth, Dr. Post and their associates, not only on the thoroughness and conscientiousness with which this study has been set up and executed, but also upon the clarity of their presentation.

My only experience with this subject has already been set forth in a report from the Wilmer Institute upon a closely similar investigation done by Dr. Frank B. Walsh and associates on the staff of the Wilmer Institute and which was reported by me.

The investigations reported today differ from the Baltimore report only in several minor respects: 1) high degrees of myopia or astigmatism were excluded, 2) a full 20/20 corrected vision was required in each eye, and 3) a normal muscle balance was required. In our series we took all grades of myopia up to 10 diopters. A full 20/20 corrected vision was not required, although this was the visual level of 81 of the subjects, while 18 had 20/30 + in each eye, 2 had from 20/50 to 20/30 vision, and 2 had unilateral amblyopia with 20/20+ in the second eye. We did not require a normal muscle balance, or investigate the question of binocular vision, our investigation being conducted on the basis of improvement in the individual eye, rather than in binocular vision. We tested both the pre-training and post-training visual acuity on four charts: Snellen letter, Snellen number, Snellen E, and a modified Landolt broken ring chart, taking as the final visual acuity the mean of these four tests. Further, in the St. Louis investigations the visual training was conducted under the direction of Drs. Hildreth and Post, while in the Baltimore investigation it was conducted independently by a group of optometrists and experimental psychologists under the direction of Drs. Skeffington and Rershaw. The two courses of visual training, however, appear to be closely similar if not identical. Despite these differences, which I believe are unimportant, the results of the two investigations are startlingly similar.

Drs. Hildreth and Post found that in 42 of their 54 cases, or 77.8 per cent the final vision was unaffected by the visual training, while in 12 patients, or 22.2 per cent there was a definite improvement. We found no

significant change in the visual acuity in 73 of our 103 cases, or 71 per cent, while in 30 or 29 per cent there was definite improvement. In both the experiments the average degree of improvement was exactly the same—27 points in the visual efficiency scale.

In only 9 of our patients showing this improvement were we able to obtain later examinations to see if the improvement was maintained. In 2 of these patients the improvement had been fully maintained, in 4, partially maintained, while in 3 nearly all had been lost. Drs. Hildreth and Post found the improvement maintained in 50 per cent of their patients showing improvement. There is no statistical difference in these figures. Both the Baltimore and the St. Louis groups found that the visual training had produced no change whatsoever in the underlying refractive error. We concluded that the improvement noted in 29 per cent of our patients was due to teaching the patients to interpret their blurred retinal images more carefully. Drs. Hildreth and Post concluded the improvement occurred at the cerebral rather than the retinal level—a happy phrase!

Thus the results of these two studies are practically identical, and the conclusions drawn from them by two entirely separate groups of investigators are the same with one exception. Drs. Hildreth and Post state "We must conclude that visual training has a definite, but limited, value in some myopic patients." We concluded that "the visual training used on these patients was of no value in the treatment of myopia." Thus we have the paradox of two groups of investigators pursuing the same investigation, reaching results which are identical, yet drawing radically different conclusions as to the value of the visual training. I should like to defend my view-point.

The conclusion of Drs. Hildreth, Post and their associates is clearly based on two facts, a) that 22.2 per cent of their patients showed an improvement of 27 points in the percentage visual efficiency, and b) that 50 per cent of these permanently retained the improvement. The question before us is: Are these facts significant?

In Major Pincus' paper on the unaided visual acuities correlated with refractive errors he specifically notes that in individuals with identical refractive errors, i.e., 1.50 sph= —1.00 gl × 180′, the vision may vary from 20/100 to 20/300, or from a percentage visual efficiencies of 48.9 to 8.2, a swing of 40.7 points. In our series we found that patients with exactly the same refractive error likewise varied up to 42 points in the percentage visual efficiency, the mean variation being 29 points. We further found that when the same individual was tested on the 4 different charts at the same examination he might show an even higher variation, on different charts, swings of over 50 points in the visual efficiency being observed. In evaluating any beneficial results we should therefore first determine if on the pre-training determination of visual acuity the patients who showed the definite improvement had higher, average, or lower visual acuities than would be expected with their refractive errors. Were the pre-training visual acuities higher than would be normally expected, any improvement obtained would be the more significant; if lower than would be normally expected, the improvement would lose its significance.

An analysis of the 30 cases showing improvement in the Baltimore series, and 11 of the patients showing improvement in the St. Louis series, shows that the pre-training visual acuity was the average to be expected with their refractive errors. Thus in the Baltimore group, 7 subjects had higher than expected uncorrected vision, 16 had about the expected vision, and 7 had lower than the expected vision. In the St. Louis group 4 had higher than expected vision, 4 had the average expected vision, and 3 lower than the expected vision. If we accept 40+ points as the normal variation in vision with the average refractive error, a swing of 20+ points in the average individual is without significance. The average improvement noted in the groups benefited by the visual training was 27 points in each series.

In our report I called attention to the fact that the experiment was uncontrolled. This is also true of the St. Louis investigation. If an experiment is fruitless, the absence of controls is unimportant, but when any positive results are obtained, these results must be interpreted against the behavior of a control group. We found it impossible to assemble a proper group of controls, and I have no doubt Drs. Hildreth and Post encountered this same difficulty. A proper control in this experiment would be an equal number of intelligent myopes with refractive errors and vision comparable to the training group. This control group should have their uncorrected vision determined, and they should be advised that myopes of the same degree may have different levels of vision on the Snellen chart, depending on the care with which they interpret their own blurred retinal images. They should then be dismissed to follow their usual lives with their vision uncorrected and with no visual training whatsoever save that which they gave themselves by being more meticulous in their visual habits. At the

end of training given the test group of myopes, the control group should be reassembled and their visual acuities again determined, to ascertain what change, if any, had been accomplished by the simple explanation of the situation and the advice to be careful in their visual interpretations. What the results would be in such a control group, one can only surmise. I should not be at all surprised if 25 per cent of them did not show an average of 27 points improvement in their visual efficiency.

In the St. Louis investigation Drs. Hildreth, Post and their associates found that 50 per cent of those showing improvement maintained their improvement, while 50 per cent reverted to their former sloppy visual habits and lost what they had gained. In other words, in 5 out of 54 patients, roughly 10 per cent, there was a final definite gain. Our figures on this point are not so accurate, since we were able to recapture only 9 of our 30 patients showing improvement, for the later final examination. However, accepting the higher and more convincing figures of the St. Louis group one may well ask the question, is this gain of 27 points in the visual efficiency, gained in only 10 per cent of the subjects, worth the time and the effort? This improvement corresponds to an improvement from 20/200 to 20/100, or in the lower brackets from 20/80 to 20/40. It is noteworthy that even in the lower degrees of myopia, only 2 of the trainees gained the 20/20 level, improving from 20/30.

To my mind these meager results do not justify the expenditure of the time, the effort, or the expense normally entailed by such visual training. My admiration for Dr. Post as a chairman, an administrator, an investigator, and a friend is unbounded. In this instance however, I am unconvinced. On the evidence so far advanced, I do not believe that visual training has a definite or even a limited, value in the practical treatment of myopia. It may have, in some cases, a psychological value in reconciling the myope to his handicap. All in all, however, I believe visual training should regretfully be consigned to the limbo of well-intended, but fruitless experiments.

KENNETH C. SWAN, M.D., Portland, Ore.: We are indebted to the speakers and their colleagues for providing us with statistical data to discuss a well-publicized controversial problem. We can best attempt to pay our debt to these men for their tedious and intensive work, by properly interpreting it to the public, to optometrists and to our colleagues.

Both authors stress that their studies were limited to visual acuity, but the training procedures described in the published report would be beneficial to patients with anomalies of convergence and accommodation. Dr. Post included no cases in his report, but such anomalies commonly accompany uncorrected myopia. This fact must be taken into consideration in discussing, with patients, visual training in myopia. For example, a young competent attorney stated that he was receiving great benefit from visual training administered by an optometrist. It was expensive but he was going to continue because until he had visual training he was unable to read without glasses.

Spurred by the improvement in his reading ability without glasses, this man falsely hoped to discard his glasses for distant vision. I say falsely because an analysis of his previous symptoms reveal that he was able to read with one eye at a time before training, but that when he attempted to use both eyes the print ran together. He had other symptoms typical of convergence insufficiency. So in discussing visual training with our patients we should not be satisfied to say that visual training does not alter the refractive error. We shou'd add that in a few cases increased visual performance may result from improved coordination of the eyes and better interpretation.

I should like to call attention to another class of patients who are not uncommon and who fall into the hands of quacks unless properly directed to psychiatrists. These patients characteristically profess great improvement in their visual acuity from training but an objective change cannot be demonstrated. Rationalization is of no avail because this is no simple matter of vanity concerning glasses. For example the intelligent daughter of a university professor professed great improvement from expensive training received from an optometrist, but objectively we were unable to demonstrate improvement in her vision. This patient was a schizoid type. This type of individual expresses subconscious thoughts in a distorted form. When this girl wore her myopic correction she saw sharply and was aware of the faces of other people. They appeared to be staring at her; consequently, with her glasses she was awkward and selfconscious. She was in a hard, sharp world of critics. Without glasses people's faces seemed blurred but the girl was less conscious of them as individuals and felt at ease. It was a soft, blurred and kindly world. Unconsciously this girl had accepted visual training as a means of staying in this friendly world. Rationalization and attempts to force this girl to accept the fact that she was getting no benefit from training might have done her harm. We must direct these patients to psychiatrists

rather than drive them into the hands of quacks.

Dr. Post and Dr. Woods brought up the point that the improvement in visual acuity was on a mental level. We use two simple, little slides to demonstrate to our myopic patients the mechanism by which improvement in visual acuity occurs after they discontinue wearing glasses for a period of time. Experience, rather than visual training, is the deciding factor.

(Slide No. 1) Any child can recognize that these simple block letters are: T - H - E.

(Slide No. 2) This slide shows only the shadows cast by the letters. The actual lines of the letters are missing but the average adult mentally fills in the defects from experience and interprets the letters as three dimensional. On the other hand, the average child who has had no experience with three dimensional letters has difficulty reading the word. In other words, we demonstrate to our patients that with or without training they will, by experience, learn to interpret and to fill in incomplete or blurred images when they discontinue their myopic correction.

Thank you, Dr. Post, and thank you, Dr. Woods.

S. JUDD BEACH, M.D., Portland, Maine: It is high time that the so-called myope cures by unorthodox methods should be evaluated. We are indebted to Dr. Post and previously to Dr. Woods and their associates for these thorough investigations. It seems generally believed that the gain is in the improved interpretation of blurred images, the position previously taken by Duke-Elder.

Though this is doubtless true of most cases studied, in last year's TRANSACTIONS, I described a different sort of case. An individual trained to squeeze his lids together was able to improve his vision during that act from about 4/200 to around 20/30. One keen observer thought he actually shortened the globe; another believed it simply the pinhole phenomenon.

I have since found in patients in our office, that this is a knack possessed by only part of the myopes. It is not purely the result of pinhole vision, as some, while looking through a pinhole, can still further improve their visual acuity by squeezing the lids together. An 18-year old boy whose relaxed vision is 2/60, with a 1 millimeter pinhole saw 6/60 in one eye and 6/30 in the other, but with the same pinhole plus squeezing he saw 6/20 in the 6/60 eye, and 6/20-1 in the 6/30 eye.

It will be recalled that Bates and his followers in this country and Simpson in England teach that by action of the extrinsic muscles they produce genuine shortening of the eyeball. Such a result can be conceived as effected by action of the orbicularis. It is certainly not a pinhole phenomenon in the cases I have described. Admitting that the improvement is mostly at the cerebral level we should not wholly close our minds to other possibilities.

WILLIAM H. CRISP, M.D., Denver, Colo.: The patient's judgment and experience and general cerebral capacity are very important in these cases. Sometimes, of course, peculiar ways of thinking about things may account in the general objection to wearing myopic lenses. I had a patient some years ago who came in complaining about the glasses that I had prescribed for her. She said, "Doctor, I cannot possibly wear these glasses because when I sit at a table I see every pore of the person in front of me." The anatomic basis that has been reported on by Dr. Woods and by Dr. Post and his colleagues seems to be the determining factor as to whether we give credence to the claims that have been made by the people who charge $175.00 for improving the vision of a myopic patient.

If the patient's anatomic condition remains the same, I cannot see the ultimate advantage of these so-called exercises. A patient comes to me and says that he has paid an optometrist $175 to get him into the Army because he was not able to get into the Army on the first test. He is just as badly off anatomically as he was before and generally speaking he has gained nothing as to getting into the Army.

I should think that almost any fairly intelligent patient would gain somewhat by experience in interpreting what was seen on the test card. It is a common experience with most of us when we have an illiterate patient who says he does not know the letters but knows the numbers to find that he has difficulty—usually, of course, he is a foreigner—in recognizing numbers; which obviously is due to a lack of familiarity with the special shapes of the number on the chart, whereas he could recognize the numbers more easily if they were shaped like those with which he is familiar.

I have had a number of patients who, at the first examination and the first fitting for a myopic correction, could only read down to a certain line on the chart, and who six months or a year later could read a line or two or three lines further down the chart. Surely some value must be given to experience of things seen which could not be seen before the correction was worn, and judgment comes into that result just as it has come into the work done on

such patients by the investigators who have spoken today.

BENJAMIN MILDER, M.D., St. Louis, Mo.: I should like to thank Drs. Woods, Swan, Beach and Crisp for their very interesting and thought-provoking comments upon this very highly controversial and scarcely yet touched upon field.

I believe that it is possible to see in Dr. Woods' comments a significant interrelationship between the various points that he has discussed. They would tend to make it seem that, having tasted of this new concoction and not having attained an extremely significant and highly remarkable positive result, we should, therefore, reject it and return, shall we say, to the ostrich attitude regarding this point which has been prevalent in ophthalmology prior to this time.

I believe that a careful consideration of the paper as read by Dr. Post would indicate that our approach has been a carefully limited and highly conservative one. That would explain why we limited so carefully the patients whom we studied in this case. We felt that people on the fringe, at the edges of the distribution curve, may throw our result off, so that we might get an abnormal improvement or abnormally poor results.

With regard to the question of binocular vision and the use of binocular evaluation, it should be pointed out, although that part of the paper was not read, that all of the training procedures which were utilized by the people performing these tests were procedures in binocularity, and therefore it is only logical that, since visual acuity as such in the average person is a binocular function and the tests were binocular tests, one would evaluate the vision of the two eyes together.

I should like further to read a sentence that Dr. Woods quoted from our paper. Dr. Woods has the feeling that the 25 per cent, plus or minus, definite improvements which were obtained both in his study and in ours are of no value in the treatment of myopia and have no significance. He quoted from our paper: "We must conclude that visual training has a definite, but limited, value in some myopic patients, preponderantly in those whose vision does not correspond with their known myopia." I should like to read the sentence which follows: "Considering the time and effort expended by the patient, the results, both in percentage of patients improved and in the actual amount of improvement per patient, must be definitely increased if the procedure is to be generally applicable to the routine management of myopia."

It is entirely possible that future studies will demonstrate that there is no applicability or clinical significance of this type of training in the treatment of myopia. However, when we see oil on the ground, we usually dig for oil there and not at some barren part of the ground where we have no suspicions.

Dr. Swan mentioned the interrelationship between muscle anomalies and malcorrecetd myopia. His point is very well taken and I believe that that would be the next logical point of departure in the investigation of the effect of visual training on myopia. I believe there is nothing unusual or even highly surprising about the success that we achieved in this investigation. You are all acquainted with the fact that in amblyopia ex anopsia, where we have an instance of a visual organ not being utilized to its fullest capacity, we see with even more simple methods some times a much more remarkable improvement in the visual acuity.

# NONPERFORATING OCULAR INJURIES IN SOLDIERS

BENJAMIN RONES, M.D.
*and*
HELENOR CAMPBELL WILDER
BY INVITATION
WASHINGTON, D. C.

APPROXIMATELY four thousand eyes enucleated in army hospitals between Pearl Harbor and V-J Day have been received at the Army Institute of Pathology. This material is being subjected to pathologic study in an effort to obtain additional information regarding the reaction of the ocular tissues to trauma. The great majority of injuries were perforating wounds with or without retention of foreign bodies. These will be considered in a subsequent paper. This report deals only with those injuries which did not include penetration of the eyeball. One hundred four soldiers had incurred ocular wounds of this description; 47 during combat or training; 57 before entering the service.

The interval between injury incurred during combat or training and enucleation varied from less than one day to two and a half years. These cases have been studied by subdividing them on the basis of time elapsed between injury and enucleation: less than one day, one day to two weeks, two weeks to three months, and three months to two and a half years.

Four eyes enucleated on the day of injury illustrate the changes which were immediate.

*Case* 1. *A.I.P. Accession* 80348

The patient was unconscious and was bleeding from the nose and right eye when brought to the hospital immediately following an airplane crash. He was found to have a penetrating wound through the right orbit, which had caused fracture of the orbit, laceration of the cerebrum, and cerebral hemorrhage.

Pathologic observations: Hemorrhagic and serous exudate with a high polymorphonuclear leukocyte content was present in the orbital tissues attached posteriorly to the globe. There was a little hemorrhage into the anterior chamber; red blood cells clung to the posterior surface of the iris and to the ciliary processes. On one side there was hemorrhagic detachment of the choroid. Degenerative changes were beginning to take place in the lens.

*Case* 2. *A.I.P. Accession* 88565

A 50-calibre cartridge exploded in the patient's hand, injuring the eye.

Pathologic observations: Massive intra-ocular hemorrhage had occurred in the shallow anterior chamber, in Schlemm's canal, beneath the detached ciliary body, choroid, and retina, and in the vitreous chamber. A metallic foreign body was found in the adherent orbital tissues.

*Case* 3. *A.I.P. Accession* 91571

The patient was struck by an automobile, receiving a compound comminuted fracture of the right side of the skull, and an orbital in-

FIG. 1—Rupture of the meshwork of the anterior chamber angle. Hemorrhage in the canal of Schlemm. ×240. A.I.P. Negative No. 90121.

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

Army Institute of Pathology.

jury. The eyeball was dislocated external to the lids, and the optic nerve appeared to be ruptured.

Pathologic observations: On one side hemorrhage engorged Schlemm's canal (Fig. 1). Rupture of the filtration angle may have resulted from the concussion, but the coloboma of the iris seemed to be an artefact. Hemorrhage had occurred in the ciliary body, and there was hemorrhagic detachment of the choroid. Hemorrhage, with a rather high polymorphonuclear leukocyte content, was present in the anterior and vitreous chambers. The ciliary processes were edematous. The nerve head was covered by hemorrhage. Beginning degenerative changes were apparent in the cortical lens fibres.

*Case* 4. *A.I.P. Accession* 123954

The patient was in a vehicle wreck, and the right eye was gouged out.

Pathologic observations: Recent hemorrhage was observed in the angle of the anterior chamber, in the posterior chamber, and around the ciliary processes. There were small subretinal hemorrhages near the disc. The optic nerve was severed traumatically.

The dominant finding in these four eyes enucleated the day on which the injury was incurred was fresh hemorrhage. Blood was found in all the ocular interspaces and throughout all intraocular structures. The mechanical effects of the blow and the amount of blood were the factors which determined the variations in the ocular architecture. It is noteworthy that in two of these cases incipient cataract was identified less than 24 hours following injury.

Eight eyes removed within two weeks of injury illustrate the changes which took place relatively soon after trauma.

*Case* 5. *A.I.P. Accession* 104621

The patient, who was in a truck accident, had severe proptosis with apparent evulsion of the optic nerve.

Pathologic observations: The optic nerve appeared to have been pulled out. There was dense thickening of the sclera. Massive hemorrhage was both intra-ocular and orbital, with hemorrhagic detachment of the ciliary body, choroid, and retina. The lens was cataractous. There was purulent panophthalmitis and orbital cellulitis (Fig. 2).

FIG. 2.—Evulsion of the optic nerve. Rupture of the filtration angle. Massive subchoroidal hemorrhage. A.I.P. Negative No. 90178.

*Case* 6. *A.I.P. Accession* 101534

The patient was struck in the eye with a baseball, which caused hemorrhage into the chamber. Two days later a massive intraocular hemorrhage produced secondary glaucoma. Paracentesis was performed five days after injury.

Pathologic observations: Polymorphonuclear leukocytes infiltrated the corneal stroma. The iris was hemorrhagic, necrotic, and infiltrated by purulent exudate, and on one side was torn away from the ciliary body. Hemorrhage from the filtration angle filled the anterior chamber, and was evident beneath the retina at the ora serrata, in the anterior vitreous, and in the cupped optic disc.

*Case* 7. *A.I.P. Accession* 112891

The patient was struck in the eye by a hard object; hemorrhage and secondary glaucoma resulted. Posterior sclerotomy and iridectomy were performed, but the iridocyclitis and secondary glaucoma persisted.

Pathologic observations: Organized hemorrhage was present in the anterior chamber, occluding the pupillary space, and in the vitreous. Chronic iridocyclitis was evident, and iris tissue was encarcerated in the corneal operative scar. The lens was cataractous.

*Case* 8. *A.I.P. Accession* 102835

Injury to the eye from an exploding cartridge caused hemorrhage into the anterior chamber with resulting secondary glaucoma.

Pathologic observations: The filtration angle was ruptured. Hemorrhage was present in the anterior and posterior chambers (Fig. 3).

FIG. 3—Rupture of the filtration angle. Hemorrhage in the anterior and posterior chambers. ×70. A.I.P. Negative No. 90132.

*Case* 9. *A.I.P. Accession* 116154

Shell fragments had entered the patient's left cheek and cut the right optic nerve.

Pathologic observations: There was a ring abscess of the cornea. Purulent exudate occupied the anterior and posterior chambers and the filtration angle, and also infiltrated the ciliary epithelium. There were broken posterior synechiae. The ciliary processes were partially hyalinized. On one side the retina was torn and extensive subchoroidal and subretinal hemorrhages were present. The optic nerve appeared edematous and degenerated, and purulent exudate infiltrated the trabeculae and nerve sheaths.

*Case* 10. *A.I.P. Accession* 131220

A mine explosion in battle caused a penetrating wound of the left cheek and contusion of the left eye.

Pathologic observations: There was massive intraocular hemorrhage. The cataractous lens was dislocated into the posterior vitreous; the retina was detached; and gliosis had occurred. The pigment epithelium of the ciliary processes was proliferated, with wide-spread scattering of pigment cells. Organization of the vitreous hemorrhage had begun.

*Case* 11. *A.I.P. Accession* 115207

The patient was struck by an automobile and incurred a head injury which resulted in death.

Pathologic observations: The retina was edematous and folded in the macular region. Small hemorrhages were present in the inner nuclear layer of the retina. Although there was slight edema of the optic nerve head, physiologic cupping was still present.

*Case* 12. *A.I.P. Accession* 136147

The patient was struck by a mortar shell which caused a severe contusion of the left eye with iridodialysis, traumatic cataract, and intra-ocular hemorrhage.

Pathologic observations: There was massive hemorrhage into the anterior chamber. On one side the iris was torn away from the ciliary body, and beneath this the lens capsule had been ruptured at the equator. The lens was cataractous. Extensive posterior synechiae were present. The epithelium of the iris and ciliary body was proliferated; the ciliary body hemorrhagic and infiltrated with chronic inflammatory cells. There was a massive vitreous hemorrhage. The lamina cribrosa was markedly depressed.

In the eight eyes described, enough time had elapsed after the injury for beginning organization of the intra-ocular hemorrhages to have become apparent. Inflammation, present in five eyes, was purulent in three. Secondary glaucoma had resulted from massive intra-ocular hemorrhage in four eyes.

Fifteen eyes enucleated between two weeks and three months following injury were also examined, and in them the organization of hemorrhage was more advanced, the incidence of chronic inflammation had increased, and early atrophic changes were seen.

*Case* 13. *A.I.P. Accession* 116524

The patient was hit in the eye by a fragment from an exploding booby trap. Ciliary injection and hypotony appeared. The eye was enucleated one month after injury.

Pathologic observations: The pupillary margin of the iris on the temporal side was ruptured, and posterior synechiae and a pupillary membrane had developed. There was phagocytized hemosiderin in the anterior chamber and in the residuum of hemorrhage in the vitreous. There was hemorrhagic and serous separation of the retina with edema of the macula.

*Case* 14. *A.I.P. Accession* 118213

Fragments from an exploding booby trap struck the eye, causing iridodialysis. Recurrent intra-ocular hemorrhages produced secondary glaucoma. Paracentesis was performed. The eye was enucleated six weeks after injury.

Pathologic observations: Traumatic iridodialysis was present, together with hemorrhages and atrophy of the iris. There were anterior and posterior synechiae, and a massive hemorrhage in the anterior chamber (Fig. 4). The lens showed an anterior cortical cataract. The macula was edematous, and secondary glaucoma

was manifested by the depressed lamina cribrosa (Fig. 5).

FIG. 4—Massive hemorrhage in the anterior chamber. ×12. A.I.P. Negative No. 90124.

FIG. 5—Early depression of the lamina cribrosa. ×40. A.I.P. Negative No. 90133.

*Case* 15. *A.I.P. Accession* 81675

Flying fragments of the skull of another soldier struck the patient's right eye, causing severe contusion with intra-ocular hemorrhage and traumatic cataract. Active panophthalmitis developed two weeks after injury and did not respond to treatment. The eye was enucleated 34 days after injury.

Pathologic observations: Blood pigment was present in the interlamellar spaces of the cornea. Hemorrhagic and serous exudate containing pigment-laden phagocytes filled the anterior chamber. On the nasal side, the iris was almost completely replaced by organizing hemorrhagic and inflammatory exudate which formed an adhesion between the cornea and the lens. On the other side were peripheral anterior synechiae, atrophy of the iris, disorganization of the iris pigment epithelium, and scar tissue on the anterior surface of the iris. The lens had undergone degenerative change (Fig. 6). The ciliary body was atrophic, and on the nasal side its processes were pulled forward by organizing hemorrhage in the anterior vitreous. Chronic inflammatory cells infiltrated the uveal tract. Lymphocytic periphlebitis was evident in the retina with edema and folding in the macular region. Secondary glaucoma was evidenced by the depressed nerve head and lamina cribrosa.

FIG. 6—Organizing hemorrhage in the anterior chamber, resulting from traumatic iridodialysis. Extensive anterior and posterior synechiae. Atrophy of the iris and ciliary body. Cataract. ×8. A.I.P. Negative No. 90149.

*Case* 16. *A.I.P. Accession* 112157

The eye was struck by a stream of water from a fire hose, with resulting intra-ocular hemorrhage, dislocated lens, and lacerated iris. Paracentesis was performed. The eye was enucleated 25 days after injury.

Pathologic observations: Organizing hemorrhage was present in the anterior chamber. The cornea was blood stained; the iris lacerated; the retina partially detached, and there was hemorrhagic detachment of the choroid. The lens was cataractous. Subacute keratitis and iridocyclitis were observed.

*Case* 17. *A.I.P. Accession* 122874

Contusion caused severe hemorrhage into the anterior chamber, and secondary glaucoma. The eye was blind and painful. An attempt was made to extract a traumatic cataract. The eye was enucleated six weeks after injury.

Pathologic observations: The cornea and scar of the operative wound at the limbus were infiltrated with chronic inflammatory cells. Organizing hemorrhage filled the anterior chamber. Remnants of the lens capsule and cortex were visible. A massive abscess and hemorrhage filled the vitreous. A large subchoroidal hemorrhage was present, and the retina was detached.

*Case* 18. *A.I.P. Accession* 86403

The eye was injured by a gun-powder explosion, and enucleated seven weeks later.

Pathologic observations: Schlemm's canal was filled with serous exudate and surrounded by dense lymphocytic infiltration. Chronic inflammatory cells and polymorphonuclear leukocytes clung to the posterior corneal surface, to

the anterior surface of the iris, and to the zonular fibers. The iris was edematous and infiltrated by plasma cells and lymphocytes, as were also the ciliary body, the inner layers of the retina, and the nerve head. The ciliary body and the peripheral choroid were stretched by the traction of plastic inflammatory exudate in the anterior vitreous.

*Case* 19. *A.I.P. Accession* 118751

A shell fragment penetrated the orbit. The eyeball was not perforated, but the globe became shrunken and painful. The eye was enucleated 18 days after injury.

Pathologic observations: The lens was cataractous and dislocated into the posterior vitreous. The iris was ruptured. There was organizing massive intra-ocular hemorrhage. The retina was detached and disorganized.

*Case* 20. *A.I.P. Accession* 105515

Injury to the eye resulted from a powder explosion. The eye was enucleated two months later.

Pathologic observations: Epidermalization of corneal epithelium had taken place over the scarred and vascularized substantia. Pigment granules were seen in the spaces of Fontana. The iris was edematous. The ciliary body was atrophic and sparsely infiltrated by chronic inflammatory cells. Its processes were partially hyalinized. There was microcystic degeneration of the outer plexiform layer of the retina, as well as serous exudates in the outer plexiform and nerve-fiber layers. Degenerative changes were present in the anterior cortical fibers of the lens.

*Case* 21. *A.I.P. Accession* 101532

A blow caused hemorrhage and secondary glaucoma in an eye which had been defective since birth. Calcareous deposits were seen in the cornea; the lens was opaque, and annular posterior synechiae were present. The eye was enucleated two months after injury.

Pathologic observations: The cornea showed pannus degenerativus with calcareous deposits and vascularization. Inflammatory tissue and organizing hemorrhage formed a membrane which covered the anterior surface of the iris, occluded the pupillary space, and encarcerated the degenerated remains of the lens. The ciliary processes adhered to the posterior surface of the iris. Posteriorly there was ossification of the choroid. Lymphocytes and plasma cells infiltrated the uveal tract. The retina was detached, and the subretinal space was filled with serous exudate containing hemorrhage, cholesterin slits, and phagocytic wandering cells. The retina which had undergone gliosis and microcystic degeneration, contained serous exudate and laminated drusen, and obliterative changes had taken place in some of the retinal vessels.

*Case* 22. *A.I.P. Accession* 105522

The patient was struck in the eye by a piece of wood, which caused laceration of the iris and retinal detachment. The eye was enucleated two months after the injury.

Pathologic observations: On one side, the iris was torn away from the ciliary body and was hemorrhagic and partially necrotic. Hemorrhage filled the coloboma and extended into the posterior and vitreous chambers. On the opposite side, strands of organizing hemorrhage and pigment-laden cells were present in the filtration angle. Degenerative changes had occurred in the cortical lens fibers. Hemorrhage had caused detachment of the retina, and there was massive subretinal hemorrhage in the macular region.

*Case* 23. *A.I.P. Accession* 146081

The patient was struck by a baseball on the left side of the forehead. At first no injury to the left eye was apparent, but two days later a large hemorrhage occurred in the anterior chamber. An intractable secondary glaucoma followed, and repeated paracentesis of the anterior chamber did not relieve the pain. The eye was enucleated three weeks after injury.

Pathologic observations: The massive hemorrhage in the anterior chamber contained considerable scattered pigment from the epithelial layer of the iris. On one side, the iris and filtration angle were ruptured, and the iris was necrotic. The pigment epithelial layer of the retina showed considerable hyperplasia.

*Case* 24. *A.I.P. Accession* 131247

A shell fragment penetrated the lid and the floor of the orbit, causing iridodialysis and massive vitreous hemorrhage. The eye was enucleated 15 days after injury.

Pathologic observations: A traumatic coloboma with organizing hemorrhage occupied the defect. Extensive rupture and detachment of the choroid and retina were present. There were large hemorrhages in the anterior and vitreous chambers, and in the subretinal and subchoroidal spaces.

*Case* 25. *A.I.P. Accession* 140195

Fragments from an exploding booby trap struck the eye and caused severe intra-ocular hemorrhage, rapid swelling of the lens and secondary glaucoma. One month after injury the

lens was removed. The eye was enucleated seven weeks after injury.

Pathologic observations: There was a large operative scar in the cornea to which there were extensive adhesions of the iris, and of lens fragments and capsule. Inflammatory scar tissue occluded the pupillary space. A large organizing hemorrhage was present in the anterior vitreous, and there was extensive serous detachment of the retina. Subacute iridocyclitis was complicated by eosinophilia.

*Case* 26. *A.I.P. Accession* 132983

Injury to the eye in battle caused severe vitreous hemorrhage and complete detachment of the retina. Mi'd iridocyclitis developed in the other eye, and enucleation was performed two months after injury because sympathetic ophthalmia was feared.

Pathologic observations: There was a large hemorrhage in the posterior vitreous chamber, with organization over the disc involving the retina. The retina was partially detached and there was hemorrhage in the subretinal space. Hemorrhage in the anterior chamber and peripheral anterior synechiae were observed, but there was no evidence of sympathetic ophthalmia.

*Case* 27. *A.I.P. Accession* 136110

A bullet entered through the right zygomatic region and made its exit through the left. There was marked displacement of the left infra-orbital plate, with downward displacement of the globe. The eye was enucleated ten weeks after injury.

Pathologic observations: The optic nerve was partially evulsed and the choroid had prolapsed into the arachnoid space. Organizing hemorrhage extended from the nerve head and adjacent choroid into the vitreous chamber.

In 20 eyes enucleated between three months and two and a half years following injury, the incidence of inflammation and secondary glaucoma was further increased, and there was one instance of equatorial staphyloma. Inflammatory pupillary and cyclitic membranes were seen and, for the first time in this series, calcium deposits in the lens and phthisis bulbi were observed.

*Case* 28. *A.I.P. Accession* 122877

The eye was hit by a fragment of exploding shell. Enucleation was performed three months after injury.

Pathologic observations: The remains of a hemorrhage were still present in the anterior chamber, and an organized hemorrhagic membrane covered the anterior surface of the iris. There were peripheral anterior synechiae. The ciliary processes were elongated and their epithelium proliferated. The retina was detached and had undergone gliosis.

*Case* 29. *A.I.P. Accession* 101964

The eye was struck by a stone. Cataract and secondary glaucoma resulted and enucleation was performed three months after injury.

Pathologic observations: There was chronic iridocyclitis with serous exudate in the anterior, posterior, and vitreous chambers. Iris bombé was present. The lens showed early degenerative changes. The lamina cribrosa was depressed from glaucoma.

*Case* 30. *A.I.P. Accession* 104773

After the eye was struck by a squash-ball, repeated hemorrhages in the anterior chamber resulted in secondary glaucoma. Pain persisted despite repeated paracentesis. The eye was enucleated three months after injury.

Pathologic observations: The anterior chamber was almost completely obliterated by organizing hemorrhage which occluded the pupil. There were anterior and posterior synechiae and anterior displacement of the cataractous lens (Fig. 7). Hemorrhage in the vitreous chamber and in the subretinal space, chronic iridocyclitis, and secondary glaucoma were present.

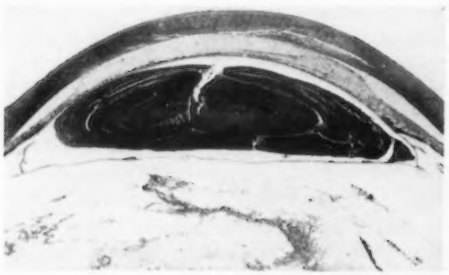


FIG. 7—Organizing hemorrhage in the anterior chamber occluding the pupil. Anterior displacement and degeneration of the lens. Hemorrhage in the vitreous chamber. ×12. A.I.P. Negative No. 90125.

*Case* 31. *A.I.P. Accession* 107046

A baseball had struck the eye causing dislocation of the lens and secondary glaucoma. The eye was enucleated three and a half months after injury.

Pathologic observations: There were chronic iridocyclitis and peripheral anterior synechiae. A small hemorrhage in the vitreous was beginning to organize. An equatorial staphyloma was present with serous detachment and cystic degeneration of the retina at this point. Secondary glaucoma was evidenced by marked excavation of the optic disc (Fig. 8).

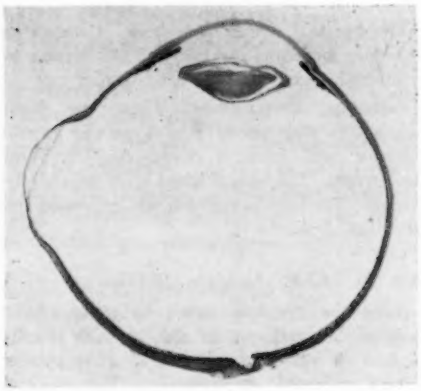


FIG. 8.—Chronic glaucoma with equatorial staphyloma. ×2. A.I.P. Negative No. 97213.

*Case* 32. *A.I.P. Accession* 105295

A blow from a fist caused severe intra-ocular hemorrhage and secondary glaucoma. Tension was not lowered by posterior sclerotomy. The eye was enucleated four months after injury.

Pathologic observations: There were peripheral anterior synechiae, atrophy of the iris and ectropion uveae. The pigment epithelium was somewhat scattered and a few of its cells clung to the anterior surface of the iris. Anterior to the sclerotomy scar the ciliary body was atrophic, and posterior to it the retina was detached. The lamina cribrosa was depressed and there were atrophic changes in the optic nerve. The lens was dislocated laterally.

*Case* 33. *A.I.P. Accession* 87772

After a blow from a fist severe intra-ocular hemorrhage occurred and endophthalmitis developed. The eye was enucleated four and a half months after injury.

Pathologic observations: On one side, the iris was torn away from the ciliary body and almost completely replaced by scar tissue which extended across the anterior surface of the lens, encarcerated the iris pigment epithelium and sphincter muscle, and covered the anterior surface of the iris on the other side. The ciliary processes showed some hyalinization. Organizing hemorrhage extended across the anterior vitreous behind the lens, which was cataractous. There was microcystic degeneration of the retina, and the choroid was infiltrated by lymphocytes and plasma cells. The lamina cribrosa was somewhat depressed.

*Case* 34. *A.I.P. Accession* 88843

The blow of a fist caused hemorrhage in the eye. A traumatic cataract was removed two months after injury. Three months later phthisis bulbi had developed and the eye was enucleated.

Pathologic observations: The perforating, operative wound at the limbus had healed. The iris was absent and ciliary processes and scar tissue occluded the filtration angle. There was a large hemorrhage in the anterior chamber and there were serous and hemorrhagic exudates in the subretinal space and vitreous. The retina was detached and showed marked lymphocytic periphlebitis. Organizing hemorrhagic and inflammatory exudate was beginning to form a cyclitic membrane. The nerve head was pulled forward and the lamina cribrosa was not depressed.

*Case* 35. *A.I.P. Accession* 116864

Injury to the eye from an exploding landmine caused iridodialysis, vitreous hemorrhage, endophthalmitis, and secondary glaucoma. The eye was enucleated five and a half months after injury.

Pathologic observations: There were traumatic iridodialysis and vascularization of the anterior surface of the iris. The ciliary processes were adherent to the meshwork in the angle of the anterior chamber at the site of the iridodialysis. There was slight chronic iridocyclitis. Organizing hemorrhage was present in the vitreous. The optic nerve was atrophic and the lamina cribrosa depressed.

*Case* 36. *A.I.P. Accession* 87429

In an automobile accident the patient sustained a skull fracture and an arteriovenous aneurysm of the right internal carotid with resultant proptosis of the right eye. The eye was enucleated six months after injury.

Pathologic observations: A purulent corneal ulcer was secondary to paralysis of the trigeminal nerve and exposure. Serous exudate was present in the angle of the anterior chamber. The vascularity of the iris was greatly increased. Lymphocytes and plasma cells infiltrated the iris and the anterior ciliary body; the ciliary processes were edematous and their vessels engorged. On one side, there was peripheral detachment of the retina with subretinal serous exudate. Multiple hemorrhages had occurred in the nerve

fiber and outer plexiform layers of the retina. The optic disc was cupped and filled with serous exudate, and the lamina cribrosa was depressed.

*Case* 37. *A.I.P. Accession* 115860

The eye was struck by a soccer ball, with resulting chronic iridocyclitis and phthisis bulbi. It was enucleated seven months after injury.

Pathologic observations: The iris had been ruptured, and its stroma replaced by organized hemorrhage which also filled the pupillary space. There was blood-staining of the cornea. The lens was cataractous. Organizing hemorrhage filled the vitreous, and a plastic hemorrhagic and inflammatory cyclitic membrane had formed. Subretinal hemorrhage had caused retinal detachment. There were chronic endophthalmitis and beginning phthisis bulbi.

*Case* 38. *A.I.P. Accession* 105658

Injury to the eye by water under pressure caused repeated hemorrhages in the anterior chamber and secondary glaucoma. Paracentesis was performed, but endophthalmitis developed. The eye was enucleated nine months after injury.

Pathologic observations: An organized hemorrhage was present in the anterior chamber. The lens was cataractous. There was mild chronic iridocyclitis, with some disorganization of the iris pigment, and several posterior synechiae. The lamina cribrosa was depressed.

*Case* 39. *A.I.P. Accession* 113277

The patient was hit in the eye by a shoe and secondary glaucoma developed. A trephine and lens extraction were performed several months after the injury but the glaucoma persisted. The eye was enucleated 18 months after injury.

Pathologic observations: Fragments of proliferated lens capsule and cortex were encarcerated in the operative scar. There was mild chronic iridocyclitis with peripheral anterior synechiae. The pigment layer of the iris showed some destruction, with pigment-laden wandering cells in the anterior and posterior chambers. The lamina cribrosa was depressed.

*Case* 40. *A.I.P. Accession* 113298

During training on an obstacle course the patient fell and struck his eye. Acute glaucoma necessitated iridectomy. The lens then became swollen and was removed 17 months after injury. Iridocyclitis developed. The eye was enucleated 20 months after injury.

Pathologic observations: The remaining iris tissue was adherent to the capsular and cortical fragments of the lens. Chronic iridocyclitis and a large intra-ocular hemorrhage were evident, and the lamina cribrosa was depressed.

*Case* 41. *A.I.P. Accession* 117649

The patient was struck in the eye while in commando training. The eye was enucleated two years after injury.

Pathologic observations: A traumatic, calcified cataract was present. There was chronic iridocyclitis, with wide anterior and posterior synechiae. The lens was displaced anteriorly and the anterior chamber was obliterated. There was an inflammatory cyclitic membrane and complete detachment of the retina, with massive subretinal serous exudate (Fig. 9). Colloid excrescenses were observed on Bruch's membrane.

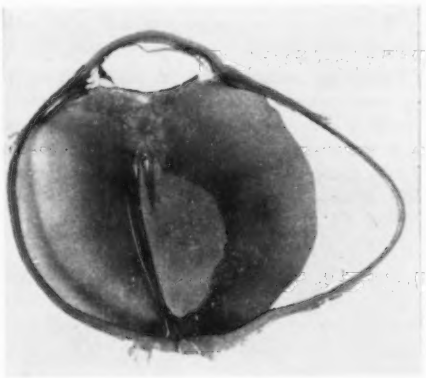


FIG. 9.—Inflammatory cyclitic membrane with detachment of the retina and subretinal serous exudate. ×2. A.I.P. Negative No. 97212.

*Case* 42. *A.I.P. Accession* 117900

The eye was struck by a baseball; cataract occurred, followed by recurrent inflammation and phthisis bulbi. The Kahn reaction was positive but became negative under treatment. The eye was enucleated two years after injury.

Pathologic observations: Anterior synechiae almost completely obliterated the anterior chamber. A dense mass of scar tissue occluded the pupil and formed an adhesion between the cornea, the anteriorly displaced lens, and the iris. There was an anterior capsular cataract, and the cortical lens fibers had degenerated. A granulomatous inflammatory process, in which foci of epithelioid cells with occasional giant cells were sparsely surrounded by lymphocytes and plasma cells, involved the vascular layer of the ciliary body and the ciliary epithelium, forming adhesions to the lens. The retina was partially detached, was cystically degenerated, and contained serous exudates.

*Case* 43. *A.I.P. Accession* 110660

The blow of a fist caused cataract, chronic iridocyclitis, and phthisis bulbi. The eye was enucleated two and a half years after injury.

Pathologic observations: A traumatic cataract contained calcium deposits. Chronic endophthalmitis had produced posterior synechiae and inflammatory pupillary and cyclitic membranes. The retina was detached and had undergone cystic degeneration; serous exudates were in and beneath it. Colloid excrescenses were seen on Bruch's membrane. Phthisis bulbi had developed.

*Case* 44. *A.I.P. Accession* 122205

A baseball struck the eye, causing rupture of the iris, repeated intra-ocular hemorrhages, disorganization of the globe, and secondary glaucoma. The eye was enucleated five and a half months after injury.

Pathologic observations: A coloboma of the iris was present, also anterior and posterior synechiae and a pupillary membrane. The ciliary body was atrophic and the lens cataractous. Secondary glaucoma was manifested by marked cupping of the optic disc.

*Case* 45. *A.I.P. Accession* 137517

A bomb explosion injured the right orbit, causing detachment of the retina and iridocyclitis. The eye was enucleated three and a half months after injury.

Pathologic observations: The iris was edematous, and there were wide posterior synechiae. The hemorrhage in the anterior vitreous had been partially phagocytosed. Chronic chorioretinitis and extensive cystic degeneration of the outer plexiform layer of the retina were present.

*Case* 46. *A.I.P. Accession* 130076

The eye was struck by an inner tube, with resulting iridocyclitis and traumatic cataract. It was enucleated nine months after injury.

Pathologic observations: A mass of scar tissue bound the atrophic iris to the cataractous lens, completely occluding the pupillary space. There was chronic iridocyclitis. The retina was edematous in the macular region.

*Case* 47. *A.I.P. Accession* 127493

The eye was struck by a sling shot. Cataract developed, followed by attacks of chronic iridocyclitis and then by secondary glaucoma. The tension was not relieved by an iris inclusion operation, and the eye was enucleated 18 months after injury.

Pathologic observations: The operative scar at the limbus was cystoid and there was a traumatic cataract. Extensive serous detachment of the retina was associated with chronic iridocyclitis and secondary glaucoma, with marked excavation of the disc.

TABLE I

PATHOLOGIC CHANGES DUE TO RECENT NONPERFORATING INJURIES

| | Same Day | 2 Wks. | 2 Wks. to 3 Mos. | 3 Mos. to 2½ yrs. |
|---|---|---|---|---|
| Hemorrhage | | | | |
| Intra-ocular | 4 | 6 | 2 | |
| Organizing | | 1 | 9 | 10 |
| Cataract | 2 | 2 | 6 | 13 |
| Detachment | | | | |
| Choroid | 2 | 1 | 2 | 1 |
| Ciliary Body and Choroid | 1 | 1 | | |
| Retina | 1 | 2 | 7 | 10 |
| Rupture of Filtration Angle | 1 | 1 | 1 | |
| Evulsion of Optic Nerve | | 2 | 1 | |
| Purulent Ophthalmitis | | 3 | | |
| Secondary Glaucoma | | 4 | 3 | 11 |
| Iridodialysis | | 2 | 5 | 3 |
| Nonpurulent Endophthalmitis | | 2 | 8 | 17 |
| Retinal Hemorrhages | | 1 | | 1 |
| Rupture of Pupillary Margin | | | 2 | |
| Edema of Macula | | | 2 | 2 |
| Blood Staining of Cornea | | | 1 | 1 |
| Dislocated Lens | | | | |
| Posterior | | | 1 | |
| Anterior | | | | 2 |
| Lateral | | | | 1 |
| Cystic Degeneration of Retina | | | | 2 |
| Phthisis Bulbi | | | | 3 |

Table I is a recapitulation of the types of lesions resulting from recent nonperforating injuries to the eye. Intraocular hemorrhage is most frequent, and is variously situated depending upon the vessels damaged. Rupture of an iris vessel can cause a massive hemorrhage in the anterior chamber. In the posterior segment of the globe the site and extent of hemorrhage depend upon

whether a ciliary, retinal, or choroidal vessel is ruptured.

Concussion cataract was observed in 23 cases in this series, two of them in eyes enucleated the day they were injured. Such cataracts may be due to rupture of the lens capsule resulting from compression of the eye, which allows access of the aqueous to the lens fibers. Dislocations of the lens also occur, anteriorly, posteriorly, or laterally, depending upon the direction of the blow and the manner in which the suspensory ligaments are torn.

A blow upon the anterior surface of the globe pushes the aqueous backward and to the side. Since the aqueous cannot be compressed, the force is reflected backward against the iris and the lens, causing them to recede or tear where they are weakest, namely, at the iris root and through the zonular ligaments (Figs. 10, 11, 12). Such an iridodialysis commonly involves only a portion of the circumference of the iris root, although the circumferential tear may be complete.

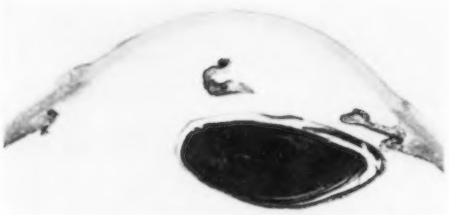


FIG. 10 — Traumatic iridodialysis, recent. ×10. A.I.P. Negative No. 90131.

The force of a blow from a blunt instrument on the anterior segment of the globe may also be dispersed so as to cause rupture of the pupillary margin of the iris, including the sphincter, or rupture of the filtration angle. The force of such a blow may be reflected posteriorly to produce detachments of the retina, the choroid, and the ciliary body. In addition, evulsion of the optic nerve has been encountered in three cases of this series.

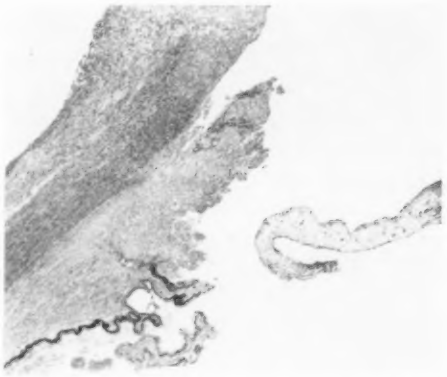


FIG. 11—Traumatic iridodialysis, recent, with hemorrhage into the anterior and posterior chambers. ×35. A.I.P. Negative No. 90154.

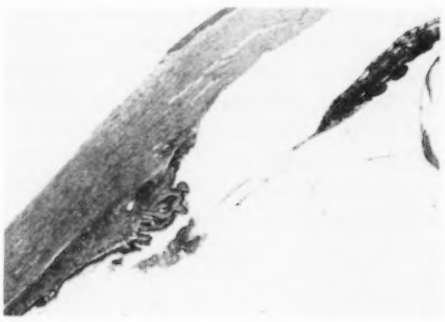


FIG. 12—Traumatic iridodialysis with beginning closure of the coloboma by organizing hemorrhage. ×30. A.I.P. Negative No. 90127.

Secondary glaucoma was a frequent sequel to contusion injuries, and resulted from massive hemorrhage, dislocation of the lens, or synechiae following recurrent nonpurulent endophthalmitis. The optic nerve may early show the effects of this increased pressure; in one case the lamina cribrosa was depressed six weeks after injury. Advanced glaucomatous excavation and atrophy may develop within a few additional weeks if the intra-ocular tension is considerably elevated.

TABLE II

CAUSE OF ENUCLEATION
(PRE-INDUCTION INJURIES)

| | |
|---|---|
| Secondary Glaucoma | 34 |
| Recurrent Inflammation | 16 |
| Recurrent Hemorrhage | 4 |
| Phthisis Bulbi | 3 |

The 57 eyes of this series with nonperforating injuries incurred before induction into the Army are analyzed as a group. The time interval between injury and enucleation varied considerably: the shortest four years, the longest 28 years. The most frequent cause of enucleation was the development of secondary glaucoma, although in many cases it was precipitated by recurrent iridocyclitis or hemorrhages. These eyes were blind in all cases, and prolonged and recurrent pain was the reason for their enucleation. As is to be expected, many of these eyes have the scars of operations, which had been performed to lower intra-ocular tension, to remove cataracts or dislocated lenses, or to repair detached retinas. Thus in these eyes the effect of surgery was superimposed upon the original trauma.

TABLE III

PATHOLOGY OF PRE-INDUCTION NONPERFORATING INJURIES

| | |
|---|---|
| Cataract | 29 |
| Calcified | 17 |
| Dislocated Lens | 5 |
| Chronic Endophthalmitis | 48 |
| Secondary Glaucoma | 32 |
| Hemorrhage | |
| Organized | 8 |
| Recent | 13 |
| Detachment | |
| Retina | 36 |
| Choroid and Ciliary Body | 6 |
| Phthisis Bulbi | 6 |
| Ossification of Choroid | 15 |
| Staphyloma | 6 |

Table III shows the most common pathologic findings in these eyes. Chronic endophthalmitis was the most frequent, with cataract, detachment of the retina and choroid, and secondary glaucoma following in that order. A great variety of degenerative and late inflammatory changes were recorded, including pigmentation and hyalinization of the anterior chamber angle meshwork, atrophy of the iris (Figs. 13, 14), pupillary and cyclitic membranes (Fig. 15), proliferation of the ciliary and retinal pigment epithelium (Fig. 16), atrophy of the ciliary body (Fig. 17),

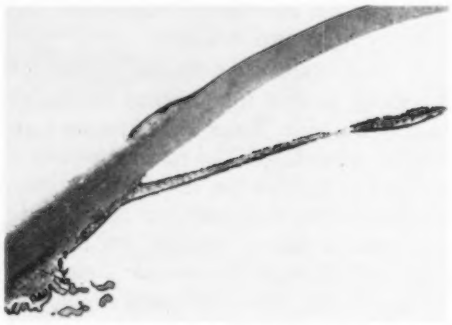


FIG. 13—Peripheral anterior synechia. Atrophy of the iris. ×20. A.I.P. Negative No. 90129.

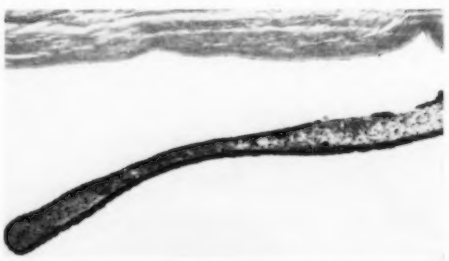


FIG. 14—Atrophy of the iris. Marked ectropion uveae. ×65. A.I.P. Negative No. 90173.

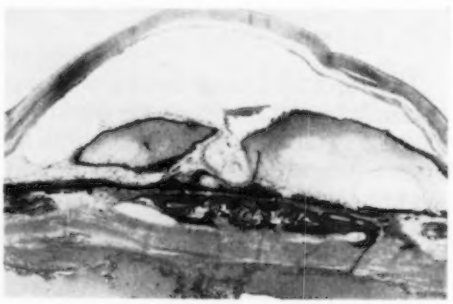


FIG. 15—Ossification of a cyclitic membrane around the encarcerated lens capsule. ×12. A.I.P. Negative No. 90168.

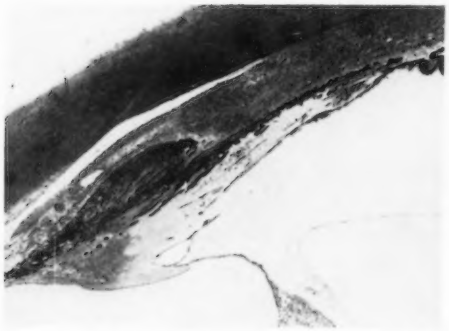


FIG. 16—Proliferated pigment epithelium in a cystic membrane. ×50. A.I.P. Negative No. 90176.

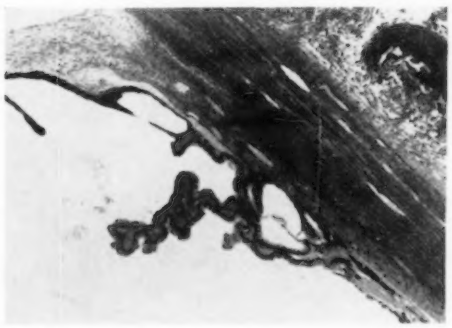


FIG. 17—Atrophy of the ciliary body. ×70. A.I.P. Negative No. 90174.

FIG. 18. Drusen on the lamina vitrea. ×50. A.I.P. Negative No. 90171.

FIG. 19—Ossification of the posterior choroid. ×6. A.I.P. Negative No. 90167.

colloid excrescences on Bruch's membrane (Fig. 18), ossification of the choroid (Fig. 19), hemorrhages and obliterative vascular changes in the retina, and retinal gliosis (Fig. 20) and cystic degeneration (Fig. 21).

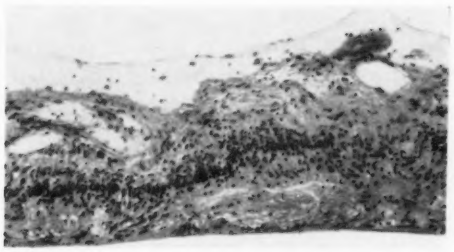


FIG. 20—Atrophy and gliosis of the retina. ×165. A.I.P. Negative No. 90165.

FIG. 21—Cystoid degeneration of the retina. ×120. A.I.P. Negative No. 90144.

The cases in this series that held the greatest interest for us were 20 in which inflammatory reactions followed contusion injuries not complicated by operative procedures. Purulent endophthalmitis was encountered twice, both times in eyes with traumatic laceration of the optic nerve. Within a period of two weeks, purulent keratitis had developed, as a result of exposure or of neurotrophic disturbance. In the first case the purulent reaction extended into the anterior chamber and involved the iris. Here the inflammation was chiefly confined to the anterior segment, although there were some leukocytes in the vitreous hemorrhage and around the vessels in the orbital tissue. The second case exhibited a ring abscess of the cornea, with purulent exudate in the anterior chamber extending into the anterior vitreous and back along the retina. The purulent reaction involved the optic nerve and its sheaths. The mechanism which produced the purulent endophthalmitis is easily comprehended; it undoubtedly proceeded by direct extension from the purulent corneal inflammation and was in no sense a blood-borne infection.

A granulomatous inflammatory reaction was seen only once, and then in the case of a soldier with a positive Kahn reaction. The history was that following contusion of the eye, recurrent inflammation set in and a complicated cataract developed, passing over into phthisis bulbi. Pathologic examination showed dense scar tissue occluding the pupil and forming an adhesion between the cornea, the iris, and the cataractous lens. The ciliary body and its processes were the sites of a granulomatous inflammatory reaction, in which foci of epithelioid cells with occasional giant cells were rather sparsely surrounded by lymphocytes and plasma cells (Fig. 22). The interpretation of this lesion raised certain questions. Was there a quiescent focus in the ciliary body which was stirred into activity by the trauma? Did trauma provoke the granulomatous lesion in an individual with systemic syphilitic disease? It is impossible to answer positively, for the problem is still in dispute in medicolegal spheres.

Active endophthalmitis was manifested in an eye enucleated seven weeks after a gun-powder explosion. There was dense lymphocytic infiltration around Schlemm's canal, with lymphocytes and plasma cells around the vessels of the intrascleral plexus and in the spaces of Fontana (Fig. 23). Chronic inflammatory cells and polymorphonuclear leukocytes clung to the posterior surface of the cornea and to the anterior surface of the iris. Chronic inflammatory cells infiltrated the iris, the ciliary body, the inner layers of the retina, and the nerve head. There was perivascular lymphocytic infiltration in the retina (Fig. 24) and nerve head (Fig. 25), with a few polymorphonuclear leukocytes in the inner layers of the retina. The intensely cellular nature of the inflammatory response is in marked contrast to that seen in a number of other cases, and led us to regard the condition here as a response to infection.

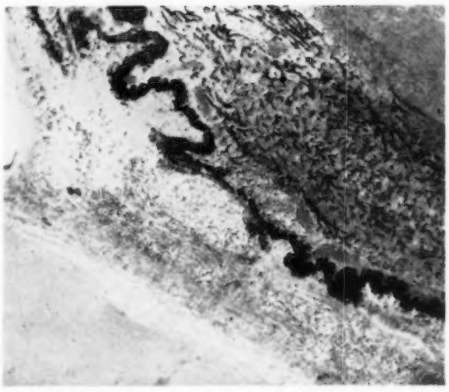


FIG. 22—Granulomatous cyclitis. Nodular aggregations of epithelioid cells in the vascular layer and epithelium. ×95. A.I.P. Negative No. 90148.

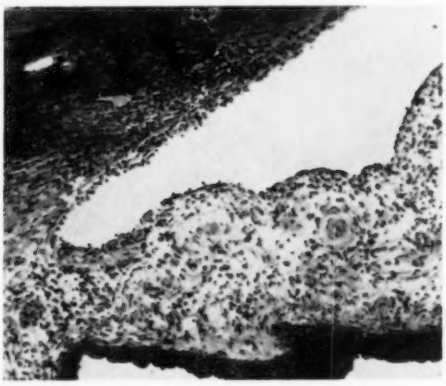


FIG. 23—Active endophthalmitis with lymphocytes and plasma cells in the iris, filtration angle, and spaces of Fontana. Aggregation of lymphocytes around the canal of Schlemm. ×160. A.I.P. Negative No. 90141.

In 16 cases in this series the inflammatory response was definitely a reaction to the tissue trauma and subsequent hemorrhage. These cases of traumatic uveitis appear to be more severe than the inflammations resulting from infections and more frequently lead to enucleation, either because severe inflammation recurs or secondary glaucoma ensues. In this series six eyes were enucleated because of glaucoma, while ten were removed for recurrent iridocy-

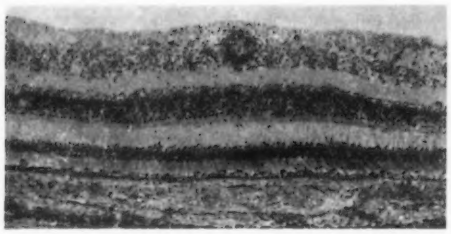


FIG. 24—Lymphocytic periphlebitis in the retina. ×130. A.I.P. Negative No. 94128.

FIG. 25—Lymphocytic perivasculitis in the optic nerve head. ×60. A.I.P. Negative No. 90142.

clitis. The mechanism of production of the inflammatory lesions is not simple. Unquestionably the injury and destruction of tissues can liberate toxic substances which induce an inflammatory response. In the same way, intra-ocular hemorrhage can act as an exciting agent. There are two less likely factors which nevertheless have their proponents, namely, reflex nerve irritation and allergic sensitivity to dispersed uveal pigment or lens proteins. In two of these cases there were enough infiltrating eosinophils to suggest an allergic factor. In the remaining cases the inflammation was predominantly in the anterior segment in 12, in the posterior segment in two. The paucity of cellular response in such traumatic uveitis without penetrating wounds was in contrast to the cellularity of infectious uveitis. Lymphocytes and plasma cells were the dominant cell types, and they were scantily infiltrated through the iris, ciliary body, or choroid. These cells were also noted in the anterior chamber, on the posterior corneal surface, and in the vitreous, particularly around the ciliary processes. The exudative response to inflammation in these cases was more marked than the cellular, and was manifested by the formation of anterior and posterior synechiae, together with pupillary and cyclitic membranes. The pigment layers of the iris, ciliary body, and retina simultaneously exhibited areas of degeneration, with scattering and phagocytosis of pigment adjacent to areas of extensive proliferation of the fixed chromatophores.

## SUMMARY

This report has dealt with 104 cases of nonperforating ocular injuries in soldiers. Forty-seven eyes were injured during combat or training. The interval between injury and enucleation varied from less than one day to two and a half years. In these eyes hemorrhage was the most frequent finding, although nonpurulent endophthalmitis, detachment of the retina, and secondary glaucoma also were often noted. Injury to eyes of 57 soldiers had preceded induction into the Army. The shortest interval between injury and enucleation in this group was four years and the longest 28 years. Chronic endophthalmitis was the most frequent finding, with cataract, detachment of the retina and choroid, and secondary glaucoma following in that order. A great variety of degenerative and late inflammatory changes were recorded.

An inflammatory reaction was present in 20 cases in which no operation had been performed. Purulent endophthalmitis in two of these had resulted from intra-ocular extension of purulent keratitis. A granulomatous inflammatory reaction was observed in the ciliary body and processes of a third. The most striking cellular response was observed in a fourth case, in which there was active endophthalmitis, with dense lymphocytic infiltration around Schlemm's

canal and chronic inflammatory cell infiltration in the iris, ciliary body, retina, and nerve head. This reaction was regarded as a response to infection. In the remaining 16 cases the inflammation was clearly a response to tissue trauma and subsequent hemorrhage, although in two the number of eosinophils in the infiltrate suggested an allergic factor. The inflammatory process was chiefly in the anterior segment in 12 cases, and in the posterior segment in two. The paucity of the cellular response was noteworthy; lymphocytes and plasma cells were the dominant cell types. On the other hand, the exudative response was marked and resulted in the formation of anterior and posterior synechiae together with pupillary and cyclitic membranes.

## DISCUSSION

W. J. B. RIDDELL, M.D., Glasgow, Scotland: In opening the discussion it is very difficult not to become lost in details of particular cases or of particular individuals. When I heard that I was to take part in this discussion I had only the title of the paper and I wondered what I could contribute that might be of interest to bring a slightly different angle to the many problems which arise from this material.

It seems to me that the general military experience in the British Army is precisely similar to that of the American Army, with no racial discrimination in booby trap or grenade, but there is a difference in the British civilian air raids because there destruction was indiscriminate, that is to say, the age group represented the whole population, the sexes were affected equally, and I wonder whether one should not keep this in mind when considering the material at Washington, in that this does respond differently in older age groups than in younger and also there might well be a sex difference. We are aware of the remarkable sex difference in Eale's disease which is predominantly in young men. These are factors which I think should be kept in view.

Another difference between the civilian eye injuries was the enormous excess of glass in the civilian ones. The large majority of eyes in the heavy raids were badly damaged or destroyed by flying particles of glass. The tissue reactions which we saw were mainly intra-ocular hemorrhages. We did learn to what a remarkable extent an eye can withstand a spicule of glass and the glass come out and the eye be repaired. It does require surgery.

One of the difficulties that we had was that these cases weren't emergencies. They were dealt with by general surgeons, and they might lay for twenty-four or forty-eight hours before anybody discovered that there was anything wrong with their eyes or that anything, in fact, could be done for them.

These are some things that struck me when I had the title of the paper.

I then recalled that before the war one of my colleagues, Dr. A. M. Wright Thomson, had wondered whether any effect was to be found when damage was done to the eyes in crush injuries. He is a very careful observer, and negative findings are not always carried out by a careful observer. He got 11 cases of crush injuries. These were mostly young people who got crushed in elevators or between roller bands, that is, their chests and bodies were crushed. They all had subconjunctival hemorrhages. Many of them had subcutaneous hemorrhages around the eyelid, but out of 11 cases there was only one in which one strip hemorrhage was found in the retina. That negative finding occurred to me to be of importance because it requires an injury around the globe or its adnexa to affect the globe itself. About half of these patients recovered and in none of them did any bursa injury, psychiatric or arthritis develop. That point struck me as I was listening to the presentation of the paper by Dr. Roñes.

When I looked at the material before the paper itself was presented an amusing idea struck me and I considered it more carefully. I thought it might add to the knowledge not only in the presentation of this material but frequently in clinical material, if you divided the cases into arbitrary categories of one week, three months, a year, three and one-half years, and so on, and presented that in a table and concealed certain information which might be brought out by a mathematical device. If there is one thing the armies and the government service in both Britain and America inquire into very carefully it is the report. They are always looking for dates and such things. I am quite sure that Dr. Rones knows the day of injury and the precise day of the removal of the eyes. If you count up the total number of days, call the day of injury zero day or V-Day, then ten days later, V plus 10, and 100 days later and 1,000 days later, that is, nothing, 10, 100 and 1,000, you may then use the logarithm of the days, and you get 1, 2, 3, 4 categories in which you can

place precisely the material and thereby spread out the tendency of tabular material to creep up.

I don't know if I have managed to put that across to you. It is a silly sort of idea perhaps you may think, but I thought it out from the table which Dr. Rones has in the text of his paper, and it is a very convenient and useful device for any series of clinical observations stretching over a long period of time. If you do it in any other way, you have an enormous table which nobody can understand and very few people read. That is one thing which struck me as being of value.

Then another matter which came to my mind was the point brought up by my senior assistants, Dr. I. C. Michaelson and Dr. George I. Scott. In those days they were Brigadier Scott and Major Michaelson. They wrote a paper which appeared in the British Journal of Ophthalmology in January of this year, dealing with some 300 or 400 battle casualties. They had 301 cases, and 47 of them were nonperforating, similar to those of Dr. Rones and Mrs. Wilder. They made one point which I think is of interest in considering material of this kind. They suggested that there is a difference between an air-conducted injury and a tissue-conducted one. In general an air-conducted one, of course, affects the anterior segment of the eyes, and tissue-conducted injuries affect the posterior section of the eye. Naturally, of course, you get exceptions to that, but, in general, that appears to come out from their material. In listening to the paper this afternoon I got the impression that the same thing that happened was present in this material to which we have listened this afternoon.

I think these are all points which struck me as being relevant, if I had any real contribution to make, and I think that all that is left for me to do is to congratulate Dr. Rones and Mrs. Wilder on the presentation of their material and on the most valuable material which they have, and finally again to congratulate them on the good use to which they have put that material.

S. RODMAN IRVINE, M.D., Beverly Hills, Calif.: The value of the contributions Dr. Rones and Mrs. Wilder have made to our knowledge of eye pathology resulting from contusion injuries lies particularly in their analysis of the condition of the eyes with respect to the interval of time following injury. An experimental study on animals made by Dr. George Kilgore, who produced varying degrees of contusion injury in monkeys and made an extensive study of the findings at various times after injury. This work was published in the Transactions of the American Ophthalmological Society for 1942. The present authors have made a somewhat similar study on the human eye, taking advantage of the large series of eyes removed following injury, available at the Army Institute of Pathology.

One is impressed by the extensive damage that may occur to the eye as a result of a concussion or contusion injury, and this I believe is not generally appreciated. Clinically there is no doubt but that one is often fooled by the appearance of an eye immediately after such an injury. It is only when recurrent hemorrhages take place and secondary glaucoma develops that the clinician is aware that a major disaster has occurred.

I wonder if the authors would have encountered any particular findings that would lead them to predict that an eye would have recurrent hemorrhages, and how soon blood-staining of the cornea might occur if blood is allowed to remain in the anterior chamber in a glaucomatous eye, and how soon cupping of the disc might be expected. Dr. Rones did mention that cupping occurred in one case about six weeks postinjury, but I wonder if there are other cases where the time interval might have been more definite. These are important questions to the clinician who desires to know how much leeway he has before it is necessary to evacuate the anterior chamber and remove the blood.

It is interesting to me that there was no incidence of sympathetic ophthalmia in spite of there being tremendous disorganization of the ciliary body and pigment throughout the globe. This confirms the observation that a perforating injury is necessary for sympathetic ophthalmia to develop, strengthening the theory of infection through the portal of perforation and detracting from the theory of sensitization to one's own pigment as causing sympathetic ophthalmia. I am aware that cases are reported of sympathetic ophthalmia without perforation, particularly in tumors, but they are so rare I think the burden of proof is on those who report such cases.

Since many of the eyes showed detached retina and marked hemorrhage in the posterior segment I wonder if the authors can venture an explanation as to why the incidence of fibrovascular membrane on the iris is so low. I believe it was mentioned in only one case.

I wonder if Dr. Rones would care to say anything about the incidence of Vossius' ring, which Dr. Trygve Gundersen described so well as occurring following contusion injuries in young people and which does not occur in elderly people. It is a pigmentary disturbance a

little smaller than the pupillary diameter, probably under the epithelium of the lens, and it seems that this series would afford a wonderful opportunity to study the condition.

Regarding case 18, with gunpowder injury, showing rather extensive inflammatory response in the eye, I wonder if this could be explained on the basis of injury to the cornea. Studying the aqueous in rabbits we found that the response in cells and protein in the aqueous can be very marked following inflammation localized in the cornea. For example, we found that it produced much greater response, as indicated by cells and protein in the aqueous, than identical trauma in the periphery of the cornea. If the cornea in this case had been extensively damaged by gunpowder I would expect a marked inflammatory response in the interior of the eye. No mention of the condition of the anterior surface of the cornea was made.

Finally, one naturally considers after reading this paper what benefit the man called upon to treat such injuries can derive from the pathologic studies Dr. Rones and Mrs. Wilder have presented.

Since hemorrhage and secondary glaucoma are the immediate complications, treatment should be directed against these. Such treatment might include complete immobilization of the eyes by snug double eye bandages, keeping the patient quiet in bed, use of ice compresses, use of miotics, and early surgical intervention for removal of blood. It would seem that wide incision and frequent irrigations of the anterior chamber are necessary to accomplish this, and possibly corneoscleral trephining. The question of advisability of removal of dislocated lenses is to be considered. The use of calcium, vitamin K, or any other treatment to inhibit bleeding would seem important.

I wish to congratulate Dr. Rones and Mrs. Wilder on their present study of the material at the Army Institute of Pathology. We all anxiously await the results of their further analysis of the abundant material available to them.

BENJAMIN RONES, M.D., Washington, D. C.: We are deeply grateful to Professor Riddell and to Dr. Irvine for discussing this paper. It is a rather formidable job to have thrown into your lap a paper of this dimension. The reading of it was very much simpler than the task the discussers had in wading through forty-seven detailed protocols and summaries of all this other material. We really do appreciate their attempt to make clinical sense out of this paper.

We also appreciate the audience's attention in the presentation of this material.

With regard to Professor Riddell's discussion, I again wish to emphasize that there were no cases in this series with penetrating injuries of the eyes. These are all nonperforating wounds; consequently, no eyes in this series contained glass, metal, or inorganic substances. There are many more eyes at the Army Institute of Pathology with penetrating wounds, and they will be classified and studied at some later date.

I am very pleased with Professor Riddell's suggestion about the time limitation. However, he must bear in mind that a considerable number of these eyes were removed in field stations and hospitals where the operating surgeons were under great pressure. There was no follow-up on them. We did have considerable difficulty with our timing, and we arrived at the time-spacing relationship on the basis of information that was as accurate as we could get, but not as detailed as a day-to-day notation would signify.

The advantage of this material lies in the fact that one has almost an experimental set of circumstances. If you are working with animals, you produce an injury, then remove the eye in one day, two or three days, or after any length of time desired and then you can study the damage and reparative processes that are present. For the first time we have this set of circumstances in a large series of cases in human eyes, and I think therein is the chief value of this material.

Dr. Irvine asks the question: How can we tell when an eye will have recurrent hemorrhages? From the pathologic basis, one can say that granulation tissue is composed of fresh young blood vessels, and consequently such capillary buds are very likely, under many conditions of strain and stress, to give way and produce another hemorrhage in the eye. How soon blood-staining of the cornea may occur, it is difficult to answer, because that will depend a great deal on the tension of the eye and how many pigment granules are forced to migrate into the corneal stroma.

With regard to the early cases of glaucoma, the earliest one we had in this series was six weeks after injury. There are a number of cases in other types of injury where cupping did occur at a considerably earlier time than that.

With regard to the fibrovascular membranes, actually we mentioned those in 5 cases, and also in many others where it was not mentioned there was description of organizing masses of hemorrhage in the anterior chamber and inflammatory exudates in the pupillary region and over the iris.

# NEW FORCEPS FOR THE REMOVAL OF NONMAGNETIC FOREIGN BODIES FROM THE VITREOUS

HARVEY E. THORPE, M.D.
PITTSBURGH, PA.

Two new forceps are presented. They were designed for the removal of nonmagnetic foreign bodies and parasites from the vitreous. They are applicable when the foreign body is readily seen with the ophthalmoscope or when it can be seen by transillumination through a scleral incision. Their use, of course, presupposes careful x-ray localization for making the appropriate scleral incision. When neither ophthalmoscope nor transillumination are applicable, one must resort to the ophthalmic endoscope.

Figures 1 and 2 show the first type presented as well as its method of application in the eye. This forceps consists of a thin, straight wire moving inside a small calibre tube about 10 cm. long. The movement of this central stiff wire is actuated by the small handle of the forceps under the surgeon's thumb. This opens the jaws of the forceps with thumb pressure. The jaws close automatically and grasp the visible foreign body when thumb pressure is released on the handle. This forceps requires a 4 mm. long scleral incision.

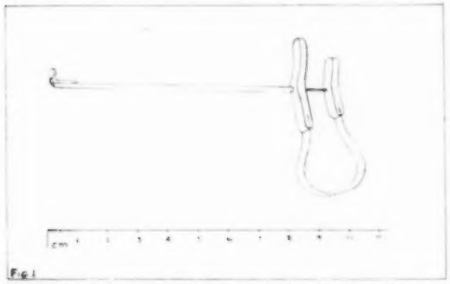


FIG. 1—Self-closing foreign body forceps for use in vitreous.

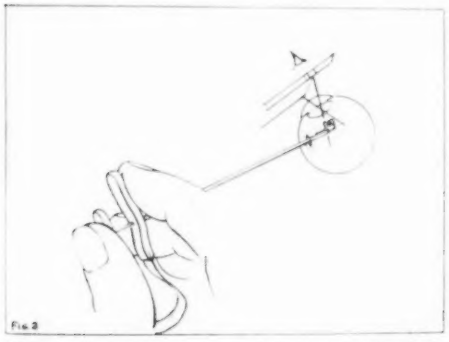


FIG. 2—Method of removal of vitreous foreign body under ophthalmoscopic control with self-closing forceps shown in figure 1.

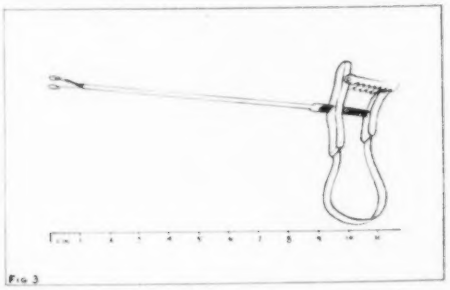


FIG. 3—Grasping type forceps for removal of nonmagnetic foreign bodies from vitreous.

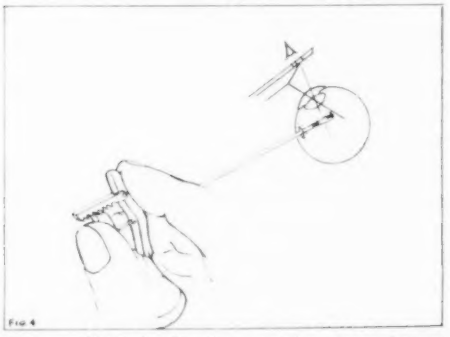


FIG. 4—Method of using grasping type forceps through scleral incision under ophthalmoscopic control. Dots about scleral incision indicate points of surface diathermy application.

The following articles by Drs. Thorpe, Schepens, Berens, and Krimsky, are descriptions of *New Instruments* presented by the authors at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 16, 1946, Chicago, Ill.

Figure 3 shows a grasping type of forceps especially useful when a very small scleral incision such as made by a 2 mm. trephine is preferred. The trephine button can be hinged and the choroid and retina incised with cutting diathermy current. The forceps has 2 small cups which meet and can be used to grasp a shot or other foreign body. The spring blades should be kept closed when introduced through the scleral opening. Pressure on the handle closes the blades and closure is maintained by a small ratchet lock. Incisions made in the sclera through retina must always be surrounded by diathermy application to avoid retinal detachment.

*Note:* These forceps are made to order at Feick Brothers, Surgical Supply Company, 819 Liberty Avenue, Pittsburgh, Pa.

## A METHOD FOR VISUALIZING THE ANTERIOR SEGMENT OF THE GLOBE BY STEREOSCOPIC ROENTGENOGRAPHY

HARVEY E. THORPE, M.D.
PITTSBURGH, PA.

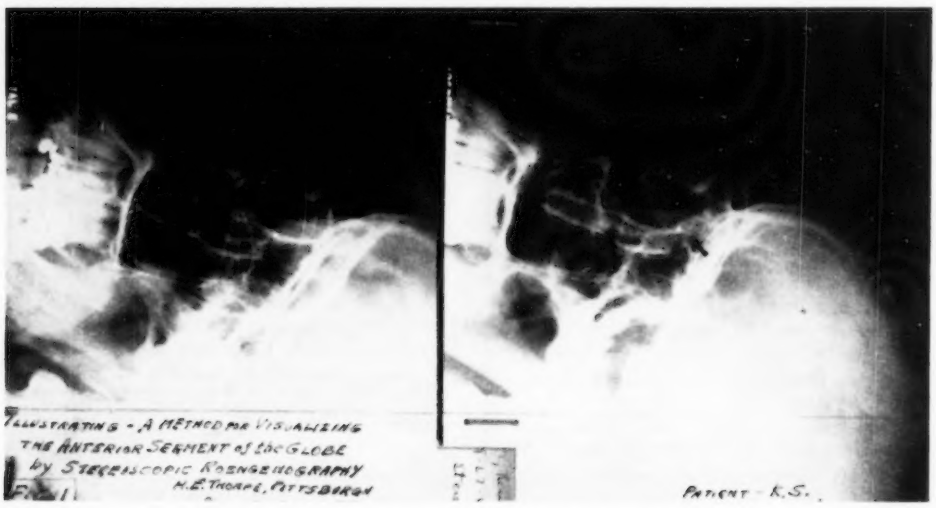

ILLUSTRATING - A METHOD FOR VISUALIZING
THE ANTERIOR SEGMENT of the GLOBE
by STEREOSCOPIC ROENTGENOGRAPHY
H.E. THORPE, PITTSBURGH
PATIENT - K.S.

FIG. 1.—Patient K. S.—Foreign body in upper temporal quadrant of posterior vitreous. Right eye. Arrow points to foreign body. This picture can be viewed with stereoscope.

THE glass contact lens offers an excellent method for outlining the anterior segment of the globe by stereoscopic roentgenography. During the past five years I have employed a modified Comberg glass localizing shell for this purpose. It has enabled me to obtain a tridimensional concept of the position of an intraocular or extraocular foreign body in relation to the anterior segment. To make the picture more realistic the outline of the globe is drawn on the x-ray film by completing the scleral arc of the contact lens (which has a 12 mm. radius).

Stereoscopic *lateral* views are made with the glass shell over the cornea and with the eye focused straight ahead on a distant object. For the postero-anterior view, the eye looks at its reflection in the cassette surface. The postero-anterior view is made in the chin-nose

position (Watters). The glass shell with its 4 lead dots are readily seen. When these are viewed in the stereoscope one gets the impression of seeing the globe with the foreign body in relation to it.

Figure 1 shows a lateral view in stereo which demonstrates a foreign body in the temporal region deep in the vitreous of the right eye. Additional flat localization plates are made in each foreign body case (1 lateral and 1 postero-anterior in Watters' position) for measurement.

*Note:* These x-rays were made by the Department of Roentgenology, Montefiore Hospital, Pittsburgh, Pa.

## A NEW PRISM HOLDER

EMANUEL KRIMSKY, M.D.
BROOKLYN, N. Y.

THE purpose of this prism holder is to facilitate the handling of individual square prisms both for prism training as well as an aid in diagnosis. For this

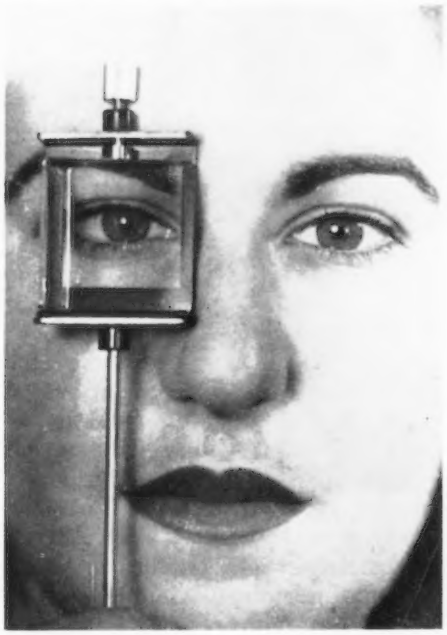


FIG. 1—Prism holder.

the author has provided two units, the single and the multiple prism holders.

The single prism holder is designed for a standard square prism measuring about 38 mm. from one edge to the opposite one. It is designed to hold such prism securely either in a vertical or horizontal position. Beneath the housing proper is a vertical rod which the patient holds flush against his cheek. Such method of holding a prism relieves the patient from keeping the fingers in an otherwise cramped position around the prism; enables the prism to be held in a straight horizontal or vertical position rather than in an undesirable oblique or distorted position; and minimizes the tendency to breakage because prism is properly secured. As an aid in diagnosis, it leaves the hands of the examiner entirely free to hold flashlight with one hand and to operate a supplementary rotary prism using the other hand for the fellow eye. The method of both prism training and use in diagnosis is not within the province of this report.

The multiple prism holder permits the ready insertion of one or more separate square prisms either in the same or in different directions; for example, a base-down prism combined with a base-out prism. The value of such a unit too is readily obvious for it permits the examiner to make his observations such as prism-reflex or cover test while the patient holds the prisms in the desired positions. Hence, such holder is intended to facilitate the examination of ocular deviation showing both vertical as well as horizontal imbalance.

# A NEW OPHTHALMOSCOPE DEMONSTRATION

C. L. SCHEPENS, M.D.
BRUSSELS, BELGIUM
BY INVITATION

I AM glad to bring you the best wishes and greetings of all your Belgian colleagues. In Belgium, we expect American ophthalmologists to promote international cooperation in ophthalmology. Science, medicine in particular, must ignore any sort of frontiers. That is why today, I want to show you a new type of ophthalmoscope and would be glad to hear your criticisms and suggestions about it. I will explain why I made this instrument, what are its different parts, and how it works.

Originally, this ophthalmoscope was designed to facilitate the examination of difficult cases of retinal detachment. In fact, I now use it to examine all sorts of fundus conditions. In dealing with difficult cases of retinal detachment, the most important point is to have a source of light as powerful as possible. This helps to see the retina if the transparent media are cloudy. A strong source of light also helps to see further at the periphery, because, when examining this part of the fundus, the light penetrates into the eye very obliquely and comes out of the eye in the same way, which reduces considerably the luminosity of the ophthalmoscopic image. In Europe, the tendency has been to increase the intensity of the light by using bulbs of a very high wattage. The most powerful now in use is Weve's lamp. The motion picture will show you that my approach to this problem is rather different.

Having secured a strong light, I thought it would be very useful to have a binocular view of the whole fundus, and not only, as is the case with Gullstrand's ophthalmoscope, of its central part. I believe that the new instrument allows one to see, with both eyes, any part of the retina that can be seen by other means, with one eye.

The last difficulty to overcome is to make an instrument which is light, handy and can be used in any position, whether the patient is sitting or lying. How this problem was solved is shown in the motion picture.

My ophthalmoscope is also intended to facilitate, as far as possible, the drawing of the fundus. Gonin and after him, Weve, Amsler, and Arruga insisted on the necessity of making an accurate sketch of the fundus before operating on any case of detachment. I believe this to be of the utmost importance. Perhaps it is still more important that this sketch should be made by the surgeon himself. Special attention should be paid to the following details of the fundus: the retinal holes, or suspected holes, and their relation to the neighboring vessels; the main retinal folds; any hemorrhage or alteration of the retinal vessels; any heaping of pigment; any vitreous bands. The importance of such a drawing is threefold. Firstly, it is the best way, for the surgeon, of knowing thoroughly the topography of the fundus. Secondly, a careful examination of the whole fundus is a great help in making a prognosis, because scattered fundus alterations make the chances of a good result slender indeed, although the holes may be few, small and easy to cover with the diathermy. Thirdly, if the first operation

Patent applied for.

has proved unsuccessful, the drawing made previous to it, is often of great use. After an unsuccessful operation, the vitreous seldom remains clear and the examination of the fundus becomes difficult, but if an accurate sketch has been made to start with, the suspected areas will have been noted, and a second operation can be performed with fairly good chances of success, even though the fundus is not clearly seen.

The new ophthalmoscope is an inverted image one. This may be disturbing for many of you, who are mainly acquainted with the direct method of ophthalmoscopy. There are many reasons for not examining a detachment of the retina with the direct method. Here are the main ones:

1—With the direct method it is always more difficult to see through cloudy media.

2—When using the direct method, the strength of the light cannot be much increased, on account of the heat emitted by the bulb.

3—Making a binocular instrument, which remains handy for use with the direct method, is almost an impossible proposition.

4—The magnification of the ophthalmoscopic image, with the direct method, is too great, which makes it difficult to orientate oneself in the complicated topography of the detached retina.

In my experience, the end results of the operation for detachment of the retina are definitely poorer, wherever the direct method is the only one in use. I hope this will induce some of you to try the new instrument and to suggest improvements on its latest model, but I must warn you that its use requires some practice. There is a definite technic to be mastered. A new technic has also been evolved for its use during the operation, with a special type of electrode,

FIG. 1—The new binocular ophthalmoscope in use.

but this is out of the scope of today's subject.

The motion picture was shown.

## DESCRIPTION OF THE INSTRUMENT

Figure 1 gives a general view of the new binocular ophthalmoscope and shows the way it is used. There are two main components: the illuminant and the headband. The illuminant is fixed on a flexible and balanced arm. Once it has been swung in position, it remains there spontaneously. As a result of this, the observer's right hand is free, which allows him to draw while he actually sees the fundus. The headband is independent from the illuminant, being fixed on the observer's head. In his left hand, he holds a +18.00 D. lens.

Figure 2 shows the illuminant in detail. The ophthalmoscopic image is observed through a rectangular window (7). The use of a red-free filter (8) is particularly advisable during the operation, when using my new transilluminating electrode for surface diathermy.

Figure 3 gives a general view of the headband. It contains four total reflexion prisms with a special adjustment (4), allowing for a variable interpupillary distance and for convergence. The prisms which face the windows (5) and (7) are combined with a +3.00 D. lens. Figure 4 shows a front view of the headband. If a greater magnification of the fundus is required, additional lenses (3) may be slid before the front prisms (8).

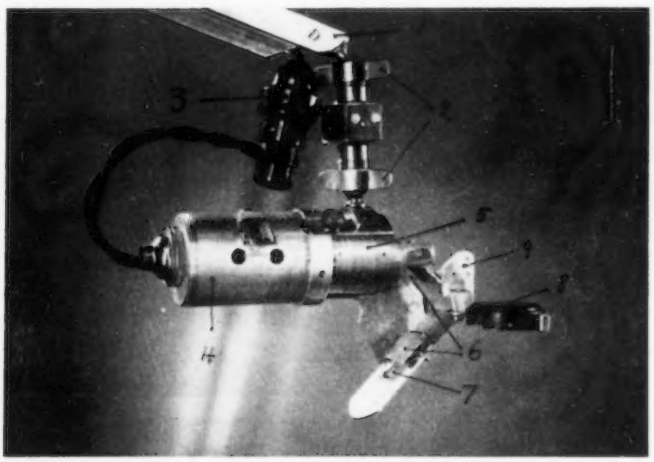



FIG. 2—Illuminant of the new binocular ophthalmoscope.

1. End of balanced flexible arm.
2. Universal joints. A special screw, not seen on this figure, enables operator to unscrew the universal joints and to remove the illuminant from the flexible arm.
3. Electrical connection.
4. Casing containing a 12 volt bulb.
5. Adjustable sleeve containing optical condensers.
6. Front silvered mirrors.
7. Window for observation of ophthalmoscopic image.
8. Red-free filter.
9. Heat insulated handle.

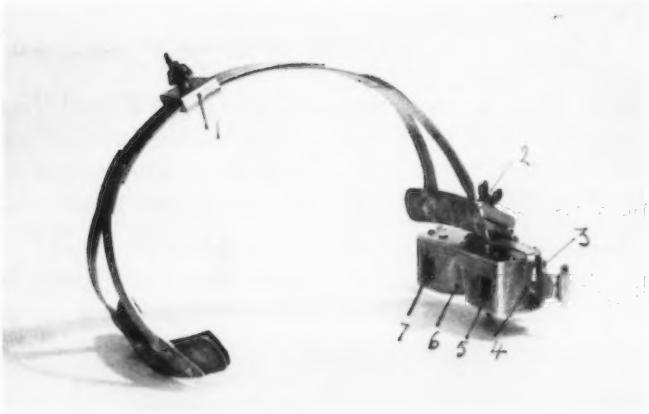



FIG. 3—Headband for binocular ophthalmoscope, general view.
1. Head adjustment.
2. Antero-posterior adjustment for the eye pieces.
3. Sliding additional lenses.
4. Lever for adjustment of convergence.
5. Window for the right eye.
6. Depression for the nose.
7. Window for the left eye.

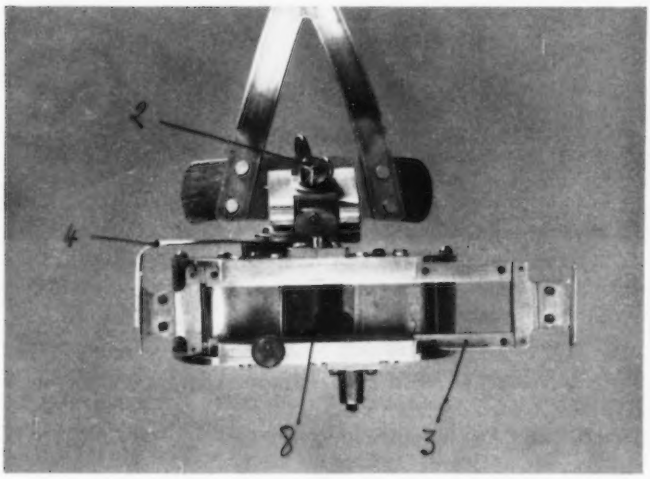



FIG. 4—Headband for binocular ophthalmoscope, front view.
2. Antero-posterior adjustment for the eye pieces.
3. Sliding additional lenses.
4. Lever for adjustment of convergence.
8. Anterior observation window for both eyes.

# AN ILLUMINATED RETRACTOR SPECULUM

CONRAD BERENS, M.D.
NEW YORK, N. Y.

AN illuminated retractor speculum with cord handle and no rheostat, has been developed. In a previous publication,[1] a plastic retractor was described, which could be inserted into a standard flashlight or attached to a cord and rheostat.

Since that time, a way has been found to dispense with the rheostat with the cord handle, thereby making the retractor more useful and less expensive (Fig. 1).

The Arruga Retinal Detachment Retractor has been constructed for use with cord handle so either the regular tip or Arruga tip may be supplied.*

These two illuminated retractors are particularly useful for operations on detached retinas and operations on the oblique extraocular muscles.

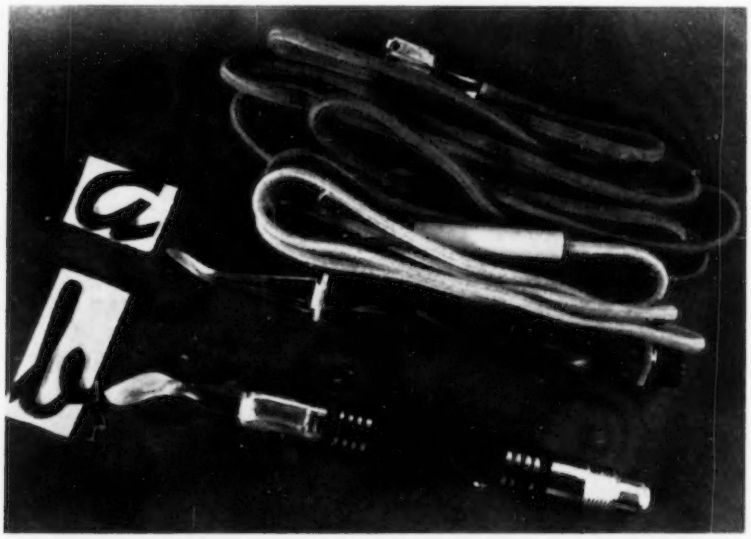



FIG. 1a—Cord handle, plug and switch with author's plastic retractor speculum.
1b—Arruga Retinal Detachment Speculum made in plastic, in flashlight handle.

Aided by grants from The John and Mary R. Markle Foundation and The Ophthalmological Foundation, Inc.

*Made by R. A. Gulden, Philadelphia 24, Pa.

## BIBLIOGRAPHY

1. Berens, C.: An illuminated retractor for eye operations especially for detachment of the retina, Am. J. Ophth., Vol. 17, No. 3 (March) 1944.

# TWO ACCOMMODATING RULES OF OPAQUE PLASTIC

CONRAD BERENS, M.D.
*and*
LUCILLE SOBACKE
BY INVITATION
NEW YORK, N. Y.

Two accommodation rules* made of milk white opaque plastic, and marked with black letters and figures, have been developed.

One rule is 50 centimeters in length, with a carrier (Fig. 1A). This rule is marked in centimeters, inches, diopters, and the mean of the near point of accommodation at different ages according to Duane.[1]

A shorter rule (Fig. 1B) similarly marked, has a string attachment which doubles the length markings in centimeters by replacing the dioptric and inch scales.

The rules are light, easily handled and are pleasant and agreeable for the examiner and patient to touch. The black letters on the white ground make the markings easy to read. Having a ruler with Duane averages for the near point of accommodation for various ages for comparison with diopters, centimeters, and inches makes reference to Duane table unnecessary.

The short rule with the string is particularly convenient for taking near point of convergence, for use in the consultation bag and in orthoptic clinics where the near point of accommodation is determined mainly in young children.

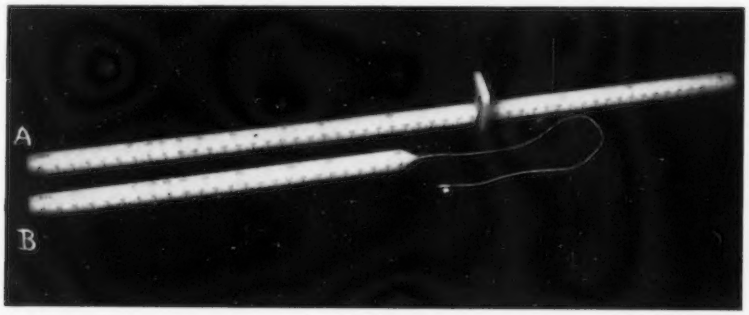



FIG. 1A—Rule with carrier, 50 centimeters in length, marked in centimeters, diopters, and the mean of the near points of accommodation at different ages, according to Duane.
1B—Shorter rule with string attachment marked in centimeters and Duane scale.

Aided by grants from The John and Mary R. Markle Foundation and The Ophthalmological Foundation, Inc.

*Made by R. A. Gulden, Philadelphia 24, Pa.

## BIBLIOGRAPHY

1. Duane, A.: Normal value of the accommodation at all ages, J.A.M.A., 58:1010, 1912.

# THE LUCKIESH-BERENS TANGENT SCREEN ILLUMINATOR

CONRAD BERENS, M.D.
NEW YORK, N. Y.
*and*
MATTHEW LUCKIESH, PH.D.
BY INVITATION
CLEVELAND, OHIO

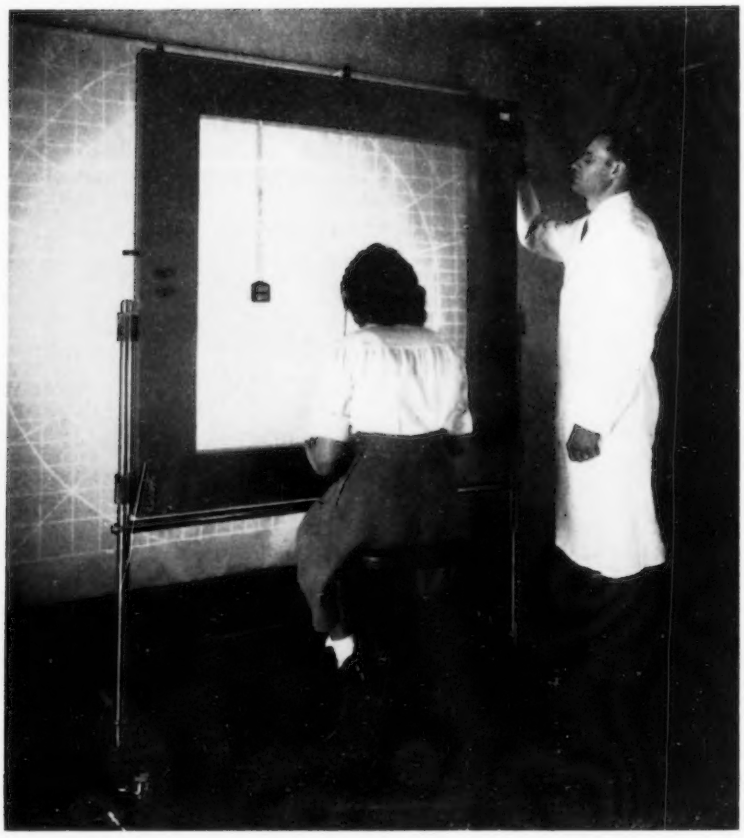


FIG. 1.—The Luckiesh-Berens Tangent Screen Illuminator showing the subject and the light sensitive cells in place.

A REAR view of the illuminator is shown in figure 1 with the light-sensitive cells in place. They swing on an arm from a position on the right side of the hollow square when not in use. The range of illumination on the central region of the screen can be varied from 1 to 100 footcandles. In fact, more than 100 footcandles can be obtained so that as the lamps depreciate in light output, 100 footcandles can always be obtained.

Aided by grants from The John and Mary R. Markle Foundation and The Ophthalmological Foundation, Inc.

In the central 25-degree zone the illumination is uniform within 5 per cent. At a position about at the edge of the hollow square the illumination drops to about 85 per cent of the value in the center of the screen.

The operator is shown adjusting the sliding triple-louver system which varies the illumination from 1 to 100 footcandles. The footcandles are indicated by a meter at the upper right corner near the operator. On the foot near the operator are two switches. Each turns on a set of four 30-watt, 36-inch fluorescent lamps. The eight lamps are used for the higher levels of illumination and four lamps are used for the lower levels.

It was decided to use the new fluorescent lamps of a color-temperature of 4500 degrees. They provide a good compromise in spectral quality between daylight and artificial light.

The wiring is fully enclosed and the little black knobs shown in pairs on the back of the hollow squares are the replaceable starting switches. The back of the hollow square is removable to expose the wiring. The ballasts are contained in the two feet of the device which also have adjustable thumb screws to take care of unevenness in the floor. The complicated wiring rises in the tubular uprights and enters the hollow square near the bottom.

# MULTIPLE TARGET HOLDER FOR VISUAL FIELD STUDY

EMANUEL KRIMSKY, M.D.
BROOKLYN, N. Y.

THE purpose of this simple device is to incorporate in a single rod targets of selective sizes and colors required for visual field study. Another aim is to keep the examiner's hand as much out of the visual field as is possible.

The rod illustrated in figure 1 is thirty inches long, square on cross-section and each surface is 12 mm. wide. It is made of wood stained dull black. Pieces of opaque plastic of different selective sizes and colors are pasted around each end, thereby making eight targets in all. By a mere twist of the thumb the examiner can switch from one target to another or may use the other end of the rod to select more targets.

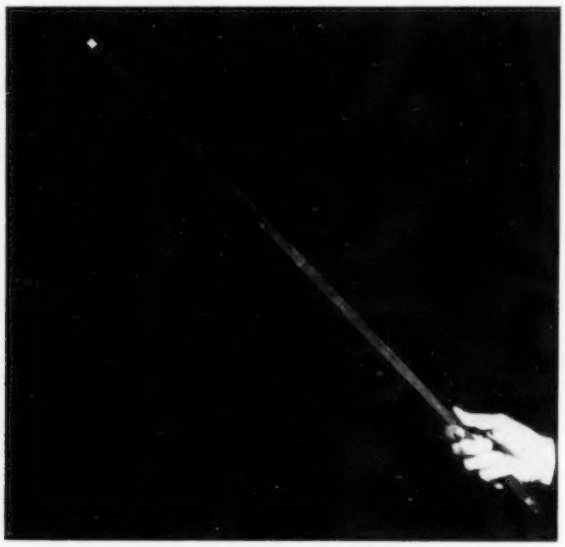


FIG. 1—Multiple target holder.

# COMPENSATING CONVERGENCE AND ACCOMMODATION DEVICE

EMANUEL KRIMSKY, M.D.
BROOKLYN, N. Y.

THE aim in this instrument is to provide a simple, exact and controlled method for the measurement of the near point or angle of convergence as well as of accommodation.

FIG. 1—A view of the convergence-accommodation device.

Convergence measurement is regulated by (a) a movable lighted target to observe the maintenance of the fixational corneal light reflexes objectively, (b) uniform mid-position of such target corresponding to symmetric convergence of the eyes, (c) uniform direction of gaze downwards and forwards corresponding to the natural direction of the eyes for this near distance, and (d) direct readings automatically calculated for the base line connecting the centers of rotation of the eyeballs.

The instrument resembles a simple stereoscope except that the upper half of the viewing head has been removed and the lower half transformed into a sliding or telescopic cheek-rest which

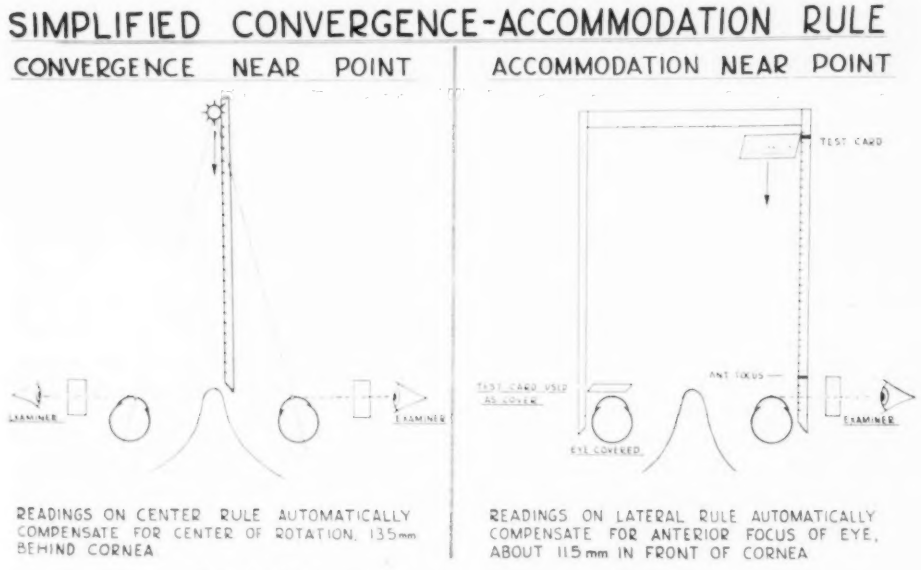

SIMPLIFIED CONVERGENCE-ACCOMMODATION RULE
CONVERGENCE NEAR POINT
ACCOMMODATION NEAR POINT
TEST CARD
EXAMINER
EXAMINER
TEST CARD USED AS COVER
ANT. FOCUS
EYE COVERED
EXAMINER
READINGS ON CENTER RULE AUTOMATICALLY COMPENSATE FOR CENTER OF ROTATION, 13.5mm BEHIND CORNEA
READINGS ON LATERAL RULE AUTOMATICALLY COMPENSATE FOR ANTERIOR FOCUS OF EYE, ABOUT 11.5mm IN FRONT OF CORNEA

FIG. 2—Mechanism of the convergence-accommodation device.

serves to move the head backwards or forwards so as to bring the eyes into a constant or controlled position to correspond to the center of rotation of the eyeballs. The anterior pole of the cornea is used as a fixed guide for such measurements and is observed through a window on each lateral wall of the frame which supports this sliding cheek rest. This window has two vertical hair lines which the examiner aligns with the anterior surface of the cornea. By simple operation of the screw for this cheek rest, this sliding portion can be moved into the proper position. The convergence distances are calibrated on the median bar which is channeled to permit the light carrier to be moved back and forth.

The accommodation unit consists of separate target holders for each eye that can be moved along a separate bar on which readings are calibrated automatically in relation to distance from the anterior nodal point of the eye. Here too, the anterior surface of the cornea serves as a control. Accommodation is tested separately for each eye and the target not in use is brought close to the untested eye to serve as a cover.

# SEPTIC THROMBOPHLEBITIS OF THE NECK

RUSSELL M. DECKER, M.D.
PASADENA, CALIF.

IN an article published in the *Archives of Otolaryngology*, the late Dr. Samuel Iglauer stated that septic thrombophlebitis of the cervical veins constitutes one of the most serious and dreaded complications of infectious processes in the neck. Fortunately, this complication is of rather infrequent occurrence, and the outcome depends in a large measure on its early recognition and the prompt employment of proper surgical and medical procedures.

Dr. Mosher stated in his original article that the most vital structure in the neck from the standpoint of infection is the internal jugular vein.

Septic thrombophlebitis is caused by pyogenic bacteria. The infection invades the vein from without, the phlebitis resulting from a cellulitis of the surrounding tissues. This cellulitis may extend along the course of the vein, so that the phlebitis has every facility for extension.

Dr. Russell Fisher, pathologist at the University of Southern California, stated that the wall of the vein is involved by an inflammatory reaction, characterized microscopically by edema and polymorphonuclear leucocytic infiltration. Progression of the inflammatory mural involvement to the intima serves as the basis for thrombosis and brings the infectious process into direct contact with the actively circulating blood stream. This and subsequent changes in the thrombus which is formed are expressed clinically by the accession of the clinical picture of septico-pyemia with chills, fever, and embolic phenomena. In its nature the thrombosis is reactionary and defensive. The development of an intravenous clot, with its firmness on palpation and the local venous obstruction it causes, makes possible the clinical detection of the venous extension, occasionally widespread, from an originally localized focus.

The degree of involvement of small venules to larger veins must have wide variation, depending chiefly on the magnitude and nature of the primary focus. Beyond doubt, phlebitis of smaller venules in the periphery of a septic lesion frequently occurs in that small degree which is not detectable clinically. The clinical condition of septic thrombophlebitis is dependent upon extension of the process to the larger middle-sized veins, where it can be detected. A detailed knowledge of the anatomical distribution and the direction of flow of the cervical veins should be an aid in correlating the original infection with its septic thrombophlebitic spread.

If the avenue of infection is by direct extension to a vein in contact with an overlying or surrounding abscess or cellulitis, there results, first, a periphlebitis which later in the process results in an endophlebitis.

The etiology is extremely varied and can result from any infection in the neck. The original focus may be any one of the following: acute tonsillitis, peritonsillar abscess, ulcerative lesions of the oral cavity, dental abscesses, pharyngomaxillary space abscess, salivary gland abscess, Ludwig's angina, retropharyngeal abscess, carbuncle, ab-

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

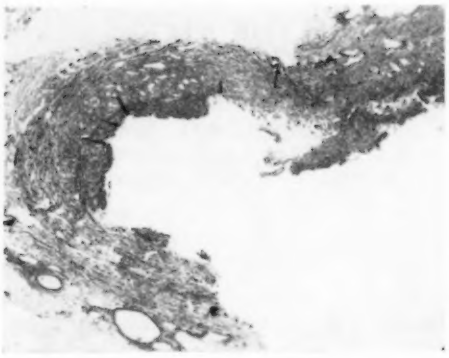


FIG. 1—Septic thrombosis of internal jugular vein. Softened thrombus has largely fallen out. Low power.

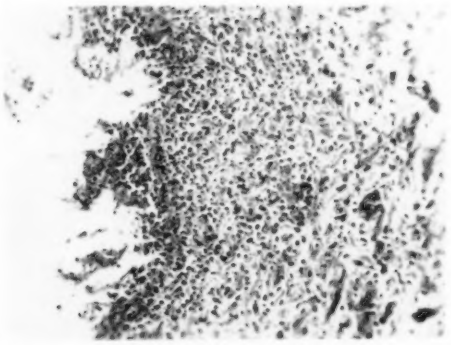


FIG. 2—Septic thrombosis of internal jugular vein. Widespread edema and infiltration of leucocytes in wall of vein. Portion of thrombus attached. High power.

scess of the neck, or osteomyelitis of the mandible and maxilla.

A history can usually be elicited of a mild or severe localized infection in an area drained by the large cervical veins. The outset of thrombophlebitis often manifests itself by a sudden chill accompanied by a high temperature. Recurring chills with high fever may continue for several days, depending upon the phlebitic process. If there is a complete thrombosis present, the chills and fever may subside until there is a breaking down of the thrombus or extension of the thrombophlebitis. One must be careful to differentiate the chills and fever which occur with the initial stage of any acute infectious process from a thrombophlebitis. Occurrence of chills is significant and of considerable importance diagnostically. They constitute one of the most characteristic clinical features of thrombophlebitis, although they may be absent in some cases. The patient may complain only of a fleeting chilly sensation accompanied by sweating. Absence of chills in thrombophlebitis does not mean lowered mortality inasmuch as diabetes or other debilitating diseases may serve as factors in the suppression of symptoms despite extensive disease. Furthermore, a few bacteria entering the circulation at a steady rate may not cause sufficient reaction on the part of the body to produce a chill.

PHYSICAL FINDINGS: The local physical findings vary a great deal depending upon the location of the primary focus of infection and on the amount of involvement. There may be a superficial cellulitis of the neck, face, or scalp. There may be a one-sided abscess of the neck with swelling and tenderness. There may be a tonsillitis, peritonsillar abscess, retropharyngeal abscess, abscessed tooth, Ludwig's angina, or a diffused nasopharyngitis, laryngitis, or other evidence of local infection. As the process develops to the involvement of the blood stream the sepsis develops with marked tenderness and swelling along the course of the internal jugular vein or along its collateral branches. Embolic infections may occur in the lungs, joints, or viscera. In some cases there may be little physical evidence of local infection even in extensive thrombosis of the jugular veins. There may be no edema or tenderness in the neck, and no palpable cord-like formations along the course of the jugular veins. The blood picture is that of severe general sepsis. Blood cultures are indicated and one positive culture is diagnostic in a suspected blood stream infection.

Blood cultures may be negative due to complete thrombosis. The leucocyte count is significant, and should be done daily. It may vary from 20,000 to 40,000. The Tobey-Ayer or Queckenstedt test, if positive, may help. Thrombophlebitis should be differentiated by careful physical and laboratory examinations from malaria, endocarditis, pneumonia, pyelitis and acute infectious diseases. The occurrence of chills and septic fever in a patient who has an apparent recovery from a throat infection requires a careful differential diagnosis.

TREATMENT: The medical treatment is very important and the earlier it is instituted the greater are the chances of limiting a thrombophlebitic process. The procedure to be employed depends upon the etiology of the sepsis and is directed at the primary focus of infection.

Penicillin in large doses should be given. The minimum daily dose in serious infections with or without bacteriemia when caused by resistant organisms should be some 200,000 to 500,000 O. U. units daily. For intermittent intramuscular injections at three-hour intervals, concentrations of 10,000 to 50,000 Oxford units per cubic centimeter of physiologic saline are desirable. Sulfadiazine should be given in conjunction with the penicillin. Dosage should be calculated according to the body weight. A blood level of 11 to 15 mgs. per hundred cc. should be maintained. Sulfadiazine blood levels should be determined daily as well as red and white blood cell counts and urinalysis. Administration of these drugs should be continued until all signs of active infection disappear or as long as the clinical course justifies its use. There is sometimes a masking effect of the chemotherapy, and the classic signs and symptoms of thrombosis are often disguised so as to render a positive diagnosis difficult. The drug may be discontinued after a week or ten days to rule this out. Other supportive medical treatment may be whole blood transfusions along with fluids, vitamins, and sedation. Heparin and dicumerol may be used.

The influence of penicillin and sulfonamides on pyogenic sepsis is profound and many extraordinary recoveries take place. Antibiotic therapy seems to hasten the cure and prevent the danger of local spread of infection and general sepsis. It is a great help but must not be depended upon to take the place of surgical treatment; rather it should be employed as a valuable adjunct to the treatment. Given a case where there is direct extension from a tonsillar, peritonsillar, retropharyngeal, or pharyngomaxillary space abscess, the pus in the primary focus of infection must be surgically drained. If the sepsis continues the carotid sheath on the infected side should be investigated with ligation of the jugular.

In a case of thrombophlebitis where there has been bilateral infection, it may be difficult to determine which side of the neck to explore. There may be a history of more pain, tenderness or swelling on one side than the other. This would lead one to explore that side.

## SUMMARY

1. The original focus of infection should be studied along with the probable course of the thrombophlebitis.
2. The symptoms are those of sepsis associated with local manifestations of infections near or along the course of the veins involved.
3. Physical findings may vary depending upon the location of the primary focus of infection and the amount of involvement.
4. Treatment—Penicillin and sulfonamides are of great value but cannot

replace surgical treatment if an undrained necrotic focus is still present. Extended exposure and drainage of the affected area and ligation of the suspected vessels should be performed if the sepsis continues.

Before the advent of the sulfa drugs and penicillin the prognosis of these cases was grave, but during the past few years, since these drugs have been used, more and more recoveries are being reported.

## DISCUSSION

J. MACKENZIE BROWN, M.D., Los Angeles, Calif.: I should like to compliment Dr. Decker on his interesting review of this vitally important subject.

The sequence of pathologic developments which terminate in thrombosis must be clearly understood before one can institute proper therapy. With the advent of chemotherapy and penicillin, the incidence of this complication has been markedly decreased.

Clinically we can distinguish two types of phlebitis:

1. The periphlebitis which results from suppuration adjacent to the vein, in which case drainage of the abcess is followed by a cessation of the septic picture.

2. The endophlebitis in which suppuration is not necessarily present and in which the local signs of infection may have disappeared. In this type, before the advent of sulfa and penicillin, ligation was the only adequate treatment. Metastatic foci, when they occur, result chiefly from this type of process.

We have observed thrombosis in the facial, anterior jugular, internal jugular, and the superior thyroid veins. In several patients the thrombosis was never demonstrated, and the patients were immediately relieved of symptoms on tying the larger veins in the neck.

Local signs along the course of the veins are infrequent in our experience. Indeed, local signs are often so meager that we are dependent upon the septic appearance, the septic course, and the history, the latter being of the greatest importance. We must remember that fatal sepsis has resulted from a very mild pharyngitis.

As to ligation, it is well to mention what and where to ligate. In pharyngeal cases the infection enters the external jugular vein by way of the anterior and common facial veins. It follows, therefore, that ligation should include the common facial and that the external jugular vein should be ligated distal to it.

Dr. Decker mentions the use of heparin and dicumerol. Before the discovery of these drugs many patients with thrombophlebitis in the head and neck recovered by virtue of the fact that complete thrombosis of the vein occurred, thereby preventing the spread of the infection. I wonder if heparin and dicumerol are not contraindicated in thrombosis due to infection, thrombosis being nature's way of curing these infections. Our experience with these drugs, while limited, would indicate that penicillin and sulfa are just as effective.

I should like again to emphasize that the use of these drugs, penicillin and sulfa, does not eliminate the necessity of surgical drainage if pus is present in a closed cavity.

FREDERICK T. HILL, M.D., Waterville, Maine: This is a concise, well-written paper upon an important subject. There seems to be nothing controversial about it, thereby making it somewhat difficult to discuss.

Septic thrombophlebitis of the neck is always a serious complication. While of less frequent occurrence, it has seemed to be even more serious than thrombosis of the lateral sinus, not because the infection is more virulent but because of the location of the original focus and its proximity to the large vessels, as contrasted with the mastoid and the accessibility of the lateral sinus with the opportunity of jugular ligation well below the thrombus in most cases. While developments in chemotherapy and in antibiotics have greatly facilitated the management of both of these complications, there is the danger of being lulled into a false sense of security and of neglecting necessary surgical measures. Drainage is still essential, and one should not hesitate to explore the carotid sheath with continuing sepsis.

The author wisely advocates combining penicillin and sulfa therapy. Cultures of the original focus might suggest the use of streptomycin. The value of anti-coagulants, such as heparin and dicumerol, should not be lost sight of, when faced with the possibility of retrograde thrombosis, especially in veins about the face.

Much depends upon early recognition and prompt and adequate treatment, both medical and surgical. A history of face, mouth or throat infection in a patient with chills and fever is suggestive, to say the least. Laboratory findings, especially a high leucocytosis, may be confirmatory. Blood cultures are frequently disappointing and reliance should not be placed upon negative reports. The author's point about the

possible subsidence of symptoms with complete thrombosis until the clot breaks down is well worth keeping in mind. Up to this point the reaction has been defensive. Waiting for extension and not realizing the probabilities invites disaster. This may vary from sudden hemorrhage, from erosion of a large vessel, to slower unsuspected embolic phenomena in more distant areas. I recall the postmortem findings of purulent peritonitis secondary to a blind peritonsillar infection.

Developments in blood vessel surgery have provided technical advantages to surgeons dealing with similar conditions elsewhere in the body which may not be available in the neck, owing to the shorter distance between the infected area and the heart, precluding the opportunity of getting ahead of the process. Early recognition then with intelligent use of chemotherapy and adequate drainage becomes essential.

A. L. BECK, M.D., New Rochelle, N. Y.: I wish to emphasize that I have seen infection of the internal jugular vein which at the time of operation had no thrombus at all, but having resected a piece of it for microscopic examination, the walls were found to be infiltrated with polymorphonuclears, with some erosion of the intima, and without thrombus formation. I called attention to that some years ago.

Then the other extreme. I have seen a large section of an internal jugular vein, extending from the level of the digastric muscle downward into the neck, completely disintegrated and gone, and its position outlined by a thrombus; this without visible pus.

I wish to emphasize that you cannot depend on the local signs in these cases. With thrombosis of the internal jugular following thrombosis of the lateral sinus with mastoiditis, you very often can feel a cord-like bogginess along the course of the internal jugular vein. That is chiefly because the neck tissues themselves are not infected and one can palpate deeply. In cases of neck infection, however, there is inflammatory swelling of the tissues of the neck and of the lymphatics, and finger palpation elicits no local signs which point to the possibility of thrombosis of the vein; therefore, one has to rely entirely upon the general systemic symptoms, such as sepsis and dehydration.

Speaking of dehydration, that is a very important part of the medical treatment. Whenever I get high concentration of sulfadiazine, say, for instance, over 15 or 20 milligrams per cent, I suspect that I have not controlled the dehydration completely. With good control of dehydration my percentages rarely go above 7 or 8 milligrams per cent. If at any time it should approach 20 milligrams per cent, I should suspect dehydration with deficient kidney elimination, and in such cases the probabilities are that the kidneys will be severely damaged, and you will have hematuria, crystal formation, and possibly anuria.

One very important sign or indication of thrombosis or infection of the vein is the occurrence of a distant inflammation, so-called metastatic inflammation. The outbreak of inflammation in distant parts of the body, may be as far distant as the feet. If that condition exists or takes place, you may be fairly sure that you have a blood-stream infection.

One thing that I have tried to stress in the past has been that there may be no abscess formation and still there may be inflammation of the vein. Sometimes there is marked inflammatory swelling of the sheath itself, and on one or two occasions I have had to snap small bleeding vessels in the sheath itself.

It also should be remembered that the phlebitis may take place initially in other veins in the neck, such as those that Dr. Mackenzie Brown mentioned. Dr. Iglauer reported a case that began with thrombosis in the anterior jugular vein.

In conclusion, it is absolutely necessary to find and drain the primary focus of infection. The use, now, of the antibiotics and the sulfonamides has introduced a factor which makes evaluation of the condition very much more difficult and it is more difficult to determine the optimum or best time for surgery.

RUSSELL M. DECKER, M.D., Pasadena, Calif.: Thank you, Dr. Brown, Dr. Hill and Dr. Beck, for discussing my paper.

In regard to Dr. Brown's question of giving heparin and dicumarol in the presence of septic thrombophlebitis, I was discussing this matter the other day with Dr. Alvin Foord, from the Pathology Department of the University of Southern California. He feels that it is difficult to determine which is the greatest factor present. On one hand the thrombus is reactionary and protective in its nature. It is nature's response to infection and attempt to wall off the infective process. On the other hand, by propagation, it can become pernicious and extend upwards or downwards, as you know. Dr. Foord feels that only after more cases have been reported and the results evaluated can this question be answered.

In regard to Dr. Hill's reference to streptomycin, until the present time, the drug has been difficult to obtain, and because of this, its use has been limited. Not until more research has been done can we recommend its use.

I will agree with Dr. Beck, the worst types of cases have few local symptoms, especially where there is an endophlebitis which develops without a complete thrombosis present.

I wish to reemphasize that the primary focus must be drained, and that penicillin and the sulfonamides will greatly help but they must not be relied upon to take the place of surgery when indicated.

# OTOLARYNGOLOGICAL ASPECTS OF WRY NECK

J. H. Maxwell, M.D.
ANN ARBOR, MICH.

The purpose of this paper is to direct attention to some of the otolaryngological aspects of wry neck and to discuss in some detail one of the more serious of the acquired types of this deformity which concerns the otolaryngologist; namely, dislocation of the atlanto-axial joint.

The differential diagnosis of wry neck must embrace both the congenital and acquired forms. Anomalies of the cervical spine and base of the skull, and the so-called "congenital torticollis," produced probably by ischemic contracture of the sternomastoid muscle, comprise the vast majority of the congenital types. Acquired types of the deformity include those due to various diseases of the cervical vertebrae, trauma and inflammatory reactions in the soft tissues of the neck, and to such conditions as occupational habits, and even attitudes assumed to compensate for imbalance of the extraocular muscles.

Cases of torticollis due to inflammatory processes in the soft tissues of the neck are the ones more commonly encountered by the otolaryngologist.

The so-called "acute wry neck" is usually a trapezius myositis produced by or associated with an acute upper respiratory infection or exposure to cold. The head is held rigidly but the torticollis is slight. Movements are limited by pain. Ethyl chloride spray, or in more severe cases injection of novocain into the tender cervical edges of the trapezius muscle, followed immediately by active full range motion frequently affords prompt relief.

Lymphadenitis and deep cervical abscesses also produce torticollis due to muscle spasm. The kinesiology of the cervical musculature is very complex, and in cases of muscle spasm due to inflammatory processes the individual muscle actions may be difficult to evaluate. The sternomastoid, the trapezius, the levator scapuli, the splenii, and scaleni muscles may be involved. In most instances of deep cervical abscess the sternomastoids are not in spasm. Torticollis due to spasm of the deep cervical muscles developing during a cervical adenitis indicates periglandular inflammation and frequently forebodes the breaking down of a gland by suppuration. In these cases, as in others of muscular origin, the head is tilted towards the side of the lesion and the chin is rotated to the opposite side and elevated. Complete range of motion is possible, but limited by pain.

Atlanto-axial dislocations may on occasion be of vital interest to the otolaryngologist. These dislocations may be partial or complete, unilateral or bilateral, and may occur with or without fracture. When fracture is present, it usually involves the odontoid process. Corner[3] emphasized the importance of the loose ligaments of the atlanto-axial joint. Laxity of these capsular ligaments permits the gliding motion of the atlas upon the axis whose insecure ovoid articular facets slope downward, outward, and forward in oppositely inclined planes. Corner[3] also stated that, due to their adaptation to rotary movement of wide excursion, they are the

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

Professor of Otolaryngology, University of Michigan Medical School, Ann Arbor, Mich.

most vulnerable of any of the spinal joints when violence is applied obliquely to the head. Besides the two lateral joints there is a third mesial joint between the anterior surface of the odontoid process and the articular surface on the posterior aspect of the anterior arch of the atlas. The importance of this latter joint in complete dislocations or in dislocations associated with fracture, lies in the possibility of compression of the spinal cord between the displaced odontoid and the posterior arch of the atlas.

Atlanto-axial dislocations, as reported by many authors, may occur with or without trauma. Dives into shallow pools, falls producing head and neck injuries, and automobile accidents have been mentioned as the traumatic etiologic factors in many cases. Stimson and Swenson[7] commented on the frequent mildness of the initiating trauma, such as a quick turn of the head or a twist of the neck while stretching in bed. Badgley[1] has recently treated one patient who sustained a unilateral atlanto-axial subluxation while turning his head quickly to catch a thrown ball. Stimson[8] stated that the more severe types of displacement are usually due to "flexion plus compression of the neck between the head and thorax" to which might be added a twisting force.

Spontaneous atlanto-axial dislocations have been reported by many observers who have encountered these lesions complicating cases of nasopharyngitis, cervical adenitis, rheumatic fever, and scarlet fever. The exact mechanism of these dislocations is not known although various theories have been proposed to explain the relationship between the weakened joint and the local or systemic infection. Berkheiser and Seidler[2] have noted that the majority of the nontraumatic subluxations are of the anterior type. They suggested that flexion of the head in recumbency predisposes to this type of dislocation.

The clinical and roentgenographic findings are:

1. Pain and disability in the severe cases, especially those with fracture (Stimson[8]). The occipital pain may be due, as suggested by Fitzsimmons,[4] to pressure upon the second cervical nerve as it crosses the lamina of the axis behind the superior articular process.
2. Torticollis. In an anterior unilateral dislocation, the head will be tilted toward the side of the lesion and the chin will be turned to the opposite side. The chin will be depressed. Corner[3] states that the "side to which the chin is directed is that on which the transverse process of the atlas is rotated backward. The side to which the head cannot be rotated is that which is, or is only partially, dislocated." It may be possible to rotate the head to some extent in the direction that increases the deformity, but any effort to rotate the head toward the midline is blocked and causes pain. There is no muscle spasm on the short side. Stimson and Swenson[7] noted that there is usually palpable spasm of the muscles on the long side and that the affected vertebra on the long side is tender. If the displacement is bilateral, there will be flexion of the head (Stimson[8]).
3. The spine of the axis may be prominent and slightly off the midline away from the side of the anterior dislocation. Great prominence of the spine suggests the likelihood of odontoid fracture.
4. Decrease in the size of the cavity of the nasopharynx due to the prominence of the lateral mass of the atlas.
5. Evidence of injury to the spinal cord. This is rare except in severe

traumatic cases with fracture. Injury to the spinal cord may be immediate and fatal, or may be delayed and cause paralysis or even sudden death many days or weeks after the injury due to a sudden turn of the head. These accidents are usually associated with a cracked odontoid process.

6. The radiographic signs may be visualized to best advantage in stereoscopic films made in lateral projection and also in anteroposterior projection taken through the open mouth. The lateral projection may demonstrate an upward tilt of the posterior arch of the atlas toward the base of the skull and forward displacement of the anterior arch of this vertebra. The anteroposterior view may demonstrate the overriding of the articular process of the atlas on that of the axis on one or both sides. If the odontoid is fractured, a diagnosis may be made from this film.

The differentiation between complete and partial atlanto-axial dislocations is often very difficult. Corner[3] stated that "the distinction is difficult to make in some cases as the complete dislocation differs from the partial only in the quantity of its symptoms, not in the quality."

In differential diagnosis one must consider the types previously mentioned. In cases of torticollis due to deep cervical abscess the tilt and rotation of the head are comparable, but the chin is elevated. Evidence of adenitis or fascial space abscess will be present. In congenital torticollis there is fibrosis and shortening of the sternomastoid muscle. Again the chin is elevated. In cases due to arthritis of the cervical spine, there will be limitation of motion in all directions and no characteristic position of the head can be described. In those cases due to an habitual attitude resulting from a position assumed at work, one maintained to overcome diplopia or to compensate for a scoliosis, there is no muscle spasm and no limitation of motion.

The treatment of atlanto-axial dislocations varies considerably with different orthopedic surgeons. Manipulation under anesthesia, prolonged traction utilizing a head halter or a Crutchfield clamp, and open reduction have been used. In general the orthopedic surgeons advise immediate treatment.

## CASE REPORTS

Case 1. J. C. No. 525644, female, age 10. This patient was admitted to University Hospital on May 13, 1943, complaining of stiff neck which developed during an attack of severe sore throat two months previously. Since the onset of the wry neck, the patient's head had been turned to the right and she had been unable to move it toward the left. She gave a history of some swelling in the side of the neck during the acute sore throat, but this had disappeared. Examination revealed the head tilted toward the left side with the chin turned toward the right and depressed. There was no palpable spasm of the sternomastoid muscles. It was possible for the patient to extend the head and rotate it to the right slightly but it was impossible for her to turn her head toward the left. Roentgenograms demonstrated an atlanto-axial dislocation or subluxation. Headhalter traction was used for three weeks and then a Thomas collar was applied and left in place for two months. Improvement was gradual and full range of motion was obtained after a period of six months.

Diagnosis: Spontaneous subluxation of the atlas on the axis secondary to acute cervical lymph adenitis.

Case 2. J. S. No. 546227, male, age 14. This patient was admitted to University Hospital on April 21, 1944, complaining of a stiff neck. He stated that an operation had been performed on his right mastoid in January of 1943 and that another operation had been performed on the same mastoid about a month later. The stiff neck apparently developed after the second mastoid operation although the history of the details of the development of this deformity was most unsatisfactory. In November, 1943, headhalter traction and, later, tongs traction were used which served to reduce the dislocation, but the correction could not be maintained. A Minerva jacket cast had been applied.

FIG. 1a—Case 1—Lateral view showing the upward tilt of the posterior arch of the atlas against the occiput.

FIG. 2a—Case 1—After reduction—Normal position of posterior arch of atlas.

FIG. 1b — Case 1 — Anteroposterior view taken through the open mouth demonstrating the lateral displacement in the atlanto-axial joint.

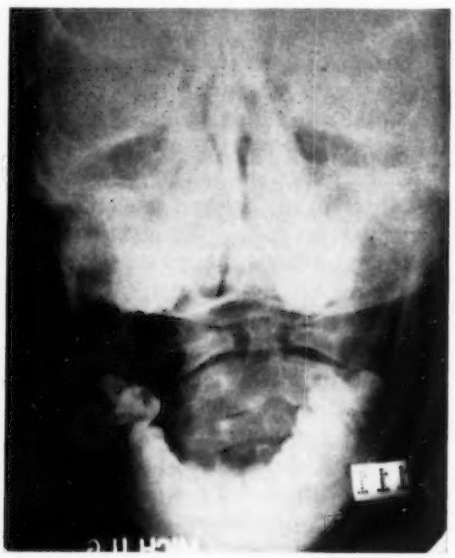


FIG. 2b—Case 1—After reduction—Normal alignment of atlas and axis.

Examination demonstrated a very marked torticollis with the chin greatly depressed and turned slightly to the left. The head was bent forward to a marked degree, and there was flattening of the cervical spine. Clinical examination suggested a right-sided forward dislocation of the atlas on the axis, but roentgenograms indicated a bilateral forward dislocation.

FIG. 3—Case 2—Lateral view demonstrating the marked forward displacement of the atlas and the flexion of the head.

A decision was made by the orthopedic surgeons to do a fusion of the occiput, the first, second, and third cervical vertebrae. On October 7, 1944, the patient was admitted to the hospital for operation, but the procedure was postponed because of an upper respiratory infection. In January, 1945, when he returned to the hospital, the clinical examination suggested some lessening of the deformity. There was no evidence of any nerve injury. Roentgenograms again demonstrated the same condition as previously described. It was decided at that time to observe the patient and postpone operation. There is no record of a return visit to the clinic since that date.

Case 3. S. M. No. 86653, female, age 6. This patient had a right acute suppurative otitis media and mastoiditis for which a right complete mastoidectomy was performed under ether anesthesia on April 7, 1942. The child was thin and weakened after a three week's acute illness which started with an attack of bronchopneumonia. The mastoid was extensively pneumatized and was the seat of a diffuse coalescent suppurative process. The operation was without untoward incident and the patient was apparently in excellent condition when she left the operating room.

At eight o'clock on the evening of the operation the patient rather suddenly became cyanotic, gasped weakly for breath, and was practically pulseless. The temperature was 100° F. She presented the picture of acute circulatory failure. She was given adrenalin, artificial respiration, and oxygen. After one-half hour her condition had improved remarkably. She was conscious, the pulse was rapid but was regular and of good quality, and she complained only moderately of pain in her operated ear. She was rather drowsy, however, and it was noted that respirations were a little labored. She held her head rather stiffly with the chin depressed and turned away from the side of the operation. The following morning her general condition was excellent and remained so during the uneventful healing of her mastoidectomy wound. The torticollis persisted and on April 25, just before her discharge from the hospital, roentgenograms of the cervical spine were ordered. The lack of muscle spasm, the painful block to rotary movements of the head to the right, and what seemed in this child's very thin neck to be an unduly prominent spine of the axis, suggested the possibility of an atlanto-axial dislocation. Roentgenograms, however, failed to demonstrate any definite abnormality in the upper cervical spine. One month later, after complete healing of the mastoidectomy wound, the torticollis remained unchanged. Roentgenograms of the cervical spine taken at this time revealed a subluxation of the atlanto-axial joint. (Fig. 4). After consultation with the orthopedic surgeons the patient was advised to enter the hospital for the application of headhalter traction. During a period of one week's procrastination, there seemed to be some improvement in the wry neck, after which the patient was reluctant to enter the hospital for treatment. On June 18, another orthopedic consultation was arranged and on that same day check up roentgenogram studies of the cervical spine were made. While she was on the x-ray table, the patient suddenly became able to move her head freely and the films which were then taken demonstrated a normal cervical spine. (Fig. 5). On clinical examination it was found that the head was held in normal position and capable of full range of motion without pain.

FIG. 4a—Case 3—The upward tilting of the posterior arch of the atlas and the forward projection of the lateral mass of the atlas producing a decrease in size of the nasopharynx are visible.

FIG. 5a—Case 3—After reduction—Normal relationship between the posterior arch of the atlas and the spine of the axis. Normal appearance of the nasopharynx.

FIG. 4b—Case 3—Overlapping of the articular processes on the right and shift to the right of the atlas upon the axis are demonstrated in this view.

FIG. 5b—Case 3—After reduction—Normal relationship between the articular surfaces of the atlas and axis. Normal position of the odontoid process.

## COMMENTS

Dislocations and subluxations of the atlanto-axial joint are conditions with which the otolaryngologist should be familiar and which he should consider in his differential diagnosis of cases of wry neck. Frank[5] discussed spontaneous lesions of this type in 1936 and reported a case due to retropharyngeal abscess. He urged otolaryngologists to concern themselves with this lesion which may complicate inflammatory conditions in the throat and neck but which he could not find discussed in any otolaryngological journal. Included in Fitzsimmon's[4] four reported cases, there was one 11 year old girl who had such a dislocation after a mastoidectomy. Hess, Bronstein, and Abelson[6] reported two cases of spontaneous atlanto-axial dislocations in the pediatrics literature. They suggested that avoidance of over-rotation of the child's head during operations on the mastoids or neck glands, and the prevention of rotation of the head by large neck dressings on children with suppurative foci in the neck would serve as prophylactic measures. A wry neck associated with cervical adenitis, pharyngeal abscess, or acute tonsillitis should prompt consideration of altanto-axial dislocation in the differential diagnosis.

The cases presented here suggest other aspects of the condition which are worthy of the otolaryngologist's consideration. Since spontaneous dislocations secondary to pharyngeal infection are more apt to occur in children than in adults, and since many of the operations done by the otolaryngologist are on children who have acute infections in the pharynx, cervical glands, or the mastoids, the surgeon should consider this lesion as a postoperative complication. Assuming that the infection has weakened the capsular ligaments of the lateral atlanto-axial joints by one or more of the pathologic changes which have been suggested by various authorities on the subject, the otolaryngologist should look for methods of preventing the occurrence of this type of dislocation. Forceful or sudden rotation of the head during the muscular relaxation in anesthesia in a patient whose capsular ligaments are weakened by infection might easily produce the dislocation. The sudden turning of the patient's head by the anesthetist in case of vomiting, or the marked rotation used to expose the operative field during a mastoidectomy or surgical drainage of a cervical abscess, particularly if there is slight flexion of the head produced by a pillow or pad under the occiput, may serve as the initiating trauma. These factors can be obviated by instructing the anesthetist regarding over-rotation of the head, by avoiding the use of a pad under the head, and by placing a sand bag under the cheek during operations which require turning the head to one side. Otolaryngologists who have seen wry neck develop rather frequently after a mastoidectomy probably have seen cases of atlanto-axial subluxation that have proceeded to spontaneous reduction. In such cases radiographs should be taken and an effort should be made to determine whether any demonstrable joint derangement represents a complete or partial dislocation. In case 3, the fortunate spontaneous reduction may have indicated that the lesion was a partial dislocation. However, the positive roentgenographic findings and the sudden attack of circulatory failure experienced by the patient a few hours after the operation, which may have been due to a quick movement of the head producing pressure on the spinal cord, indicated that there was a lesion of serious potentialities.

## REFERENCES

1. Badgley, Carl E.: Professor of Orthopedic Surgery, University of Michigan Medical School. Personal communication.
2. Berkheiser, E. J. and Seidler, Ferdinand: Nontraumatic dislocations of the atlanto-axial joint, J.A.M.A., 96:517-523 (Feb. 14) 1931.
3. Corner, Edred M.: Rotary dislocation of the atlas, Ann. Surg., 45:9-26 (Jan.) 1907.
4. Fitzsimmons, Henry J.: Four cases of the unilateral rotary displacement of the cervical spine, Interstate M. J., 22:983-988 (Oct.) 1915.
5. Frank, Ira: Spontaneous (nontraumatic) atlanto-axial subluxation, Ann. Otol., Rhin. & Laryng., 45:405-411 (June) 1936.
6. Hess, Julius H., Bronstein, I. P. and Abelson, S. M.: Atlanto-axial dislocations unassociated with trauma and secondary to inflammatory foci in the neck, Am. J. Dis. Child., 49:1137-1147 (May) 1935.
7. Stimson, Barbara B. and Swenson, Paul C.: Unilateral subluxations of the cervical vertebrae without associated fracture, J.A.M.A., 104:1578-1579 (May 4) 1935.
8. Stimson, Barbara B.: Injuries to the cervical vertebrae, New York State J. Med., 42:615-619 (April 1) 1942.


## DISCUSSION

W. E. GROVE, M.D., Milwaukee, Wis.: As I was reading Dr. Maxwell's paper the thought occurred to me that this paper takes us into a field of practice which we otolaryngologists do not ordinarily consider an allied field, namely, orthopedics. We have an interest in neurology, in neurosurgery, in neck surgery, in plastic surgery and in problems of the thorax. But hitherto most of us have considered the field of orthopedics and orthopedic surgery as more or less alien to our own. This paper illustrates the point that even in the field of orthopedics there are problems in which we share a common interest. To me, however, it teaches a far more important point and that is, that before we are otolaryngologists we must be practitioners of medicine and surgery in its broader sense. In the over-specialization of medicine we are so apt to lose sight of the patient as a whole and quickly dive into our own narrow field of practice. Is there any good reason why the otolaryngologist should not take a careful and comprehensive history? Why he should not use an ophthalmoscope, a stethoscope or a blood pressure machine? If our specialty is losing caste as has been intimated in various quarters in the last few years is it not because we ourselves are to blame and because of the fact that in practising our own specialty we have at times lost sight of the broader aspect of medicine?

While the paper reports three cases of atlanto-axial dislocation, an orthopedic subject, it also points out that a torticollis of less serious implications is due to inflammations in the pharynx, in the fascial spaces and in the soft tissues of neck. With this type of wry neck we are probably more conversant than with the orthopedic variety. But both types have certain common characteristics. They occur in the young much more frequently than after adolescence. Dr. Maxwell"s three cases were at ages 6 to 14. In a collection of cases of spontaneous dislocation of the atlanto-axial joint compiled from the literature in 1940 by Drs. Wilson, Michele and Jacobson,[1] twenty-eight in all, the ages ranged from 2 to 62 but only 4 of these were over 12 years of age. Secondly, the causative factors in both types of the condition are the acute upper respiratory infections, lymphadenitis, suppurative or otherwise, mastoid infections, scarlet fever, rheumatic fever, acute articular rheumatism, parotitis, fascial space suppurations and focal infections. In this series of cases the time between the predisposing infection and the appearance of the torticollis was given as ranging from one day to one month.

How the soft tissue infection in the neighborhood of the affected neck muscles operates to produce a spasm in these muscles is not hard to understand but how these various infections operate to produce a spontaneous dislocation of the atlanto-axial joint is not quite clear. The most plausible pathologic explanation of the production of this spontaneous condition seems to me to be that of Berkheiser and Seidler who think that this dislocation is caused by an overdistention of the bursae surrounding the atlas, induced, no doubt, by the infectious process. This is a very excellent and timely presentation.

1 Wilson, M. J.; Michele, A. A. and Jacobson, E. W.: Spontaneous dislocation of the atlanto-axial articulation, including a report of a case with quadreplegia, J. Bone & Joint Surg., 22:698-707 (July) 1940.

GORDON F. HARKNESS, M.D., Davenport, Iowa: When the Secretary asked me to discuss wry neck in relation to otolaryngology, I mistakenly assumed it would be general discussion of the subject, including spasmodic torticollis. With this phase my own clinical experience was so limited that I wrote ten of my good friends in private practice, since unusual cases gravitate to teaching clinics, and found their experience coincided with mine. It was practically nil if one excluded manifest inflam-

matory neck conditions, myositis and the like. I also anticipated the essayist might discuss that mysterious maze, the connections of the vegetative nervous system with the vestibular apparatus in association with torticollis. I was relieved when I received Dr. Maxwell's paper to find that he was limiting his discussion to a syndrome known to some as Grisel's disease.

To properly appreciate his essay I think the otolaryngologist should keep in mind types of wry neck which Dr. Maxwell is not discussing. The congenital and parturitional types present at birth, associated with breach and posterior occiput presentations and forceps deliveries, manifest a sternomastoid hematoma and fibrosis with shortening of the muscle. In spite of traction trauma I can find here no record of dislocation of the atlas-axis joint. This to me is corroborative evidence that preceding infection of the upper cervical region is a fundamental antecedent to the syndrome under discussion.

The head tilt due to extraocular muscle imbalance is a pseudo-torticollis. It is never present at birth, comes with the development of visual fusion, is a compensatory effort by nature, and the head can passively be brought to a normal position. The chin more often does not turn away from the side of the head tilt. Even if the ocular imbalance upon ocular examination is not recognized, simple occlusion of one eye will bring a return to a normal position; yet I once had a little patient in whom this simple test was overlooked in a well-recognized orthopedic clinic, and neck surgery was advised.

Structural changes due to disease, the occupational factor, and the manifest inflammations with myositis are without the province of this discussion. Foci of infection, other than the fundamental antecedent in Grisel's syndrome, have not established themselves in any chronological order. Finally, the typical spasmodic torticollis of the adult exhibits a variety of forms depending on the cervical muscles involved. There is a definite muscle spasm, not always easily defined with the deeper muscles. In the sternomastoid and trapezius types, the chin is more often elevated and markedly away from the side of the head tilt. A few cases have come to autopsy, and the central pathology has varied. Even granting its existence, there are cases which show marked accentuation with emotional stresses. Here the psychogenic factor so predominates that recovery may take place with only psychiatric treatment.

Returning to the syndrome under discussion, it is not a spasmodic torticollis per se. With some exceptions it occurs during childhood and early adolescence. But how and why? Is it because ligaments in the young are more easily distended; can myositis and muscular contraction wear down ligament efficiency? Is there a preceding effusion? Does this or a reduction of calcium content in the arch of the atlas weaken the ligamentous attachments These may be debated, but the fact remains that there is always a preceding infection in the cervical region, generally followed by a quiescent interval before the joint luxation occurs. It is to be remembered that the atlanto-axis joint is a three-point suspension. In order to subluxate, two points must give or we have a fracture.

What are some of the clinical signs leading to a suspicion of atlanto-axis subluxation prior to x-ray confirmation? I would ask Dr. Maxwell to answer a few questions. Is not the head tilt, accompanied by a greater degree of flexion in anterior dislocations than in spasmodic torticollis? Is not the chin less elevated and turned to the side opposite the head tilt in a much less degree than in other types of torticollis, excluding ocular psuedo-torticollis? In any of your cases was there an actual muscle spasm present? This syndrome under discussion to me does not mean muscle spasticity. In all of your cases could you turn the head to some degree beyond its fixed position, but found it locked in attempting to return it to the normal? Might this not be a good lead? R. C. Martin reports a case where, following a mastoid operation, the head tilt was to the opposite side, thereby putting the sternomastoid on the stretch. That also seems a good lead.

In your cases how much interference with opening of the mouth has been present? Does it have significance? In your cases did all show a narrowing of the nasopharyngeal space due to the pushing forward of the anterior ramus of the atlas. This is, of course, significant. In your third case with the postoperative collapse and rapid recovery and x-ray substantiation of a subluxation, does not this rather argue against an interference with spinal canal patency? Might a carotid sinus or something else have been the responsible factor? Finally, there is the question of the psychogenic factor in a child. Do you think that could have been a part of this picture in your third case, in spite of x-ray substantiation of the presence of and recovery from a subluxation?

I want to thank Dr. Maxwell for the honor of participating in the discussion of his essay. We all know that any presentation from the Department of Otolaryngology of the University of Michigan always means something worthwhile to take home. If we will take home and practice the precautions to which he has called our

attention, his contribution will not have been in vain.

Thank you.

ANDERSON C. HILDING, M.D., Duluth, Minn.: I am not going to take much time. I would only like to ask a question. I had such a case as this about ten years ago in a girl ten or eleven years old. She showed all the typical signs which Dr. Maxwell has given—the wry neck, the head tilt, the displacement of the spine posteriorly, the change in the pharynx, etc. She was turned over to an orthopedic surgeon and she got well. The condition showed up after mastoid operation, and I must confess I didn't know at that time that there was such a thing as atlanto-axial dislocation due to local infection. She walked around for some little time before it dawned on me that something was seriously wrong. In this girl the pneumatization was about as extensive as I have ever seen. The sigmoid sinus was so completely surrounded by cells that one might almost say it was contained within a conduit. Dr. Maxwell said that in his last case there was extensive pneumatization. My question is this, do you think that extensive pneumatization is a causative factor in the development of atlanto-axial dislocation?

JAMES H. MAXWELL, M.D., Ann Arbor, Mich.: I wish to thank the discussants for their kindness in participating in this discussion.

First, in regard to Dr. Grove's question about the weakening of the joints during infection. This is a moot question. It seems difficult to me, to explain the particular weakening of the atlanto-axial joints, or ligaments around these joints, by infection. It would seem that other joints might be involved also. Of course, we do know that the capsular ligaments are especially loose, and perhaps they may be rendered more susceptible to weakening by neighborhood infection. It has occurred to me that continued displacement of the neck due to muscle spasm might eventually wear down these ligaments and produce a subluxation. Thus perhaps we are dealing with a condition that is partly mechanical.

I was interested in studying two or three normal patients on whom we had taken x-ray films. After having the patient voluntarily turn his head to one side in a greatly exaggerated position, over-riding of the articular facets was produced and the odontoid was seen to be a little off the midline. In none of these cases, however, did we note on the x-ray film the characteristic upward tilt of the posterior arch of the atlas against the occiput, or the narrowing of the nasopharynx.

Dr. Harkness has just now given me his questions, and I am sure that there will not be time to answer all of them; but I will make an attempt to cover a few of them briefly. He asks: "Does the head tilt, accompanied by a certain degree of flexion, become more marked in the anterior dislocations than in spasmodic torticollis?" The answer is yes. By "spasmodic torticollis", he may mean one of two things: either the recurrent twitching of the head that is thought by many neurosurgeons to be on an organic basis and by many orthopedic surgeons to be on a psychogenic basis; or he may mean the acute wry neck that is very commonly due to trapezius spasm. Here there is much less tilt of the head, as Dr. Harkness suggests. As you know, the treatment is, in acute cases, manipulation or, as has been suggested by some orthopedic surgeons, forceful full range of motion after spraying the region of the trapezius muscles with ethyl chloride until light, superficial anesthesia has been produced. Some orthopedic surgeons have injected novocain into the trapezius muscles with excellent results.

Dr. Harkness asks if the chin is not less elevated and turned to the opposite side and the head tilt less in pseudo-torticollis. The answer is yes. The thing that I wish to bring out as one of the most important differential points is that in all muscular cases the chin is elevated, but in subluxation or dislocations of the atlanto-axial joint the chin is depressed.

Dr. Harkness also asks whether or not in these cases presented the head could be turned to some degree in the direction increasing the deformity. In each case that was possible, but there was a definite block and that block was associated with pain when an attempt was made to move the head toward the normal position.

In none of the cases do I recall that there was interference with opening of the mouth. I am sorry that I cannot answer that question.

Regarding the narrowing of the nasopharyngeal space, this finding was definitely noted in one of the cases. That was case 3. In case 2, the one in which there was suspected bilateral dislocation, examination of the nasopharynx was attempted but not accomplished because the patient was uncooperative. There was, however, definite roentgen evidence of narrowing of the nasopharynx.

Dr. Harkness asks about the clinical findings that would produce suspicion of this lesion. The one mentioned particularly in the third case, where the subluxation was suspected before the x-ray was taken, was depression of the chin. Then, of course, in that case also there was decrease in the size of the nasopharynx.

Dr. Harkness asks whether or not the postoperative collapse, rapid recovery and x-ray substantiation of a subluxation might argue against an interference with spinal canal patency. Personally I am inclined to feel that it rather indicates it. Of course, the signs of pressure on the brain stem and medulla vary with the acuteness of the condition although they are characteristic if the circulation is impeded. This is probably a more significant factor than actual mechanical pressure upon the nervous tissue in many instances. If the circulation is interfered with, a temporary anoxia may develop and produce changes in blood pressure and pulse rate, and respiratory disturbance of the Cheyne-Stokes type. In this case, however, that particular type of respiratory disturbance was not noted.

In the third case as to the question of the psychiatric factor, I do not know. It certainly looked typically organic. There might have been some functional factor in this girl's condition but both the clinical and the radiographic findings indicated very definitely that there was an actual organic lesion.

In regard to the frequency of occurrence of this condition I will agree that it is rare. I did find two other cases, however, one reported to me recently by one of the neurosurgeons. After an operation on a child for brain tumor the neck was dislocated. Whether it was due to rotation of the head during the operation or during the anesthesia, I do not know, but there was roentgen evidence of joint displacement. I have seen one additional case occurring after mastoidectomy.

Dr. Hilding raised the question about the extensive pneumatization of the mastoid. I cannot answer that question, although there may well be some connection, since in a very extensively pneumatized mastoid one may remove the posterior aspect of the tip and produce some inflammatory reaction in the deep cervical muscles, particularly the splenius capitis, which may go into spasm and produce wry neck. If we are to consider that mechanical factors play a part in those cases which develop spontaneously, a subluxation might be produced by such prolonged muscle spasm.

Thank you.

# DEAFNESS OF PSYCHIC ORIGIN AND ITS RESPONSE TO NARCOSYNTHESIS

HERMAN SEMENOV, M.D.
BEVERLY HILLS, CALIF.

AN accurate differential diagnosis of deafness depends upon careful otologic examination, an intelligent interpretation of hearing tests, and a precise knowledge of the physiology and pathology of the ear. Audiometry has proved to be the most dependable method of determining hearing acuity; and Crowe, Guild, and Polvogt[2] after meticulous audiometry and microscopic examination of serial sections of temporal bones have scientifically correlated audiograms with the pathology of the ear. In one-fourth of the cases of deafness they could find no histologic lesions, either in the middle or inner ear, and concluded that such cases either have no visible morphologic changes, or else have an ill-defined disturbance of the central auditory pathways—something in the nervous system that may interfere with the perception of sound.

Hearing is a mental process, and a perceptive deafness may be due to a disturbance in the inner ear, auditory nerve, or brain. Central or functional suppression of hearing is the unknown factor. But how often does it cause virtual deafness, and under what circumstances, and how can it be diagnosed and treated? These are some questions that I shall try to discuss.

Hearing tests under sodium pentothal seminarcosis have greatly facilitated the diagnosis of functional deafness. Prior to the use of this technic it was quite difficult to diagnose many such cases. After a careful analysis of the results obtained at Hoff General Hospital, an Army center for the rehabilitation of the deaf, we concluded that one-tenth of the cases (12 per cent of 500 consecutive admissions) had psychogenic deafness. Martin[9] and Rosenberger and Moore[10] have published preliminary reports concerning this material, and in this discussion I should like to make some additional observations. Truex[13] and Knapp[7] working at Deshon General Hospital, having access to similar material, subsequently arrived at similar conclusions. Walter Work[14] at Borden General Hospital was among the first otologists to employ pentothal for the diagnosis and treatment of deafness, but to Grinker[5] goes credit for the first recorded case, treated early in the course of the war.

## MATERIAL AND METHODS

The material on which this study is based consists of cases of binaural deafness with 30 decibels or more of hearing loss.* All deaf patients receive a series of repeat audiograms to establish the accuracy of their responses and this precaution uncovered many cases of functional deafness. Whisper and spoken voice tests were performed in a quiet place, soundproof chambers were

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

Work done while in the Army of the United States, Hoff General Hospital, supplemented by further work at Letterman General Hospital, and observations made at various other military hospitals.

* An average in decibels of the tones 512, 1024 and 2048. The per cent of hearing loss (Sabine[11]) was calculated according to the A.M.A. accepted method.

Symbols used: Wv—whisper voice (15 ft. normal); Sv—spoken voice (20 ft. normal); pp Sv—quiet conversational voice; ff Sv—loud conversational voice; db—decibels; R—(o) right ear; L—(x) left ear; MT—membrana tympani; OMCC—otitis media chronic catarrhal; OMCP—otitis media chronic purulent; WIA—wounded in action.

used for all AC and BC audiograms, including binaural "free field" pure tone and speech perception.

Prior to the beginning of this work I availed myself of the opportunity of visiting the Bureau of Standards and through the courtesy of Dr. Guy Cook and Dr. R. K. Cook, had the intensities of my voice measured with a noise level meter. My whisper, using functional residual air at the end of expiration, varies from 32 to 38 db.; my pp conversational voice varies from 40 to 60 db.; and my loud ff voice is in the neighborhood of 75 db. These measurements were made at a distance of 4 feet, and by using the inverse square law with the conversion factor for decibels, it is possible to predict the intensity at any distance. My figures were submitted to Dr. Harvey Fletcher of the Bell Telephone Laboratories, and he found them acceptable. I have repeatedly checked my voice against known degrees of deafness in patients and find it well within 20 db. of the audiogram, often as close as 10 db., and sometimes within 5 db. The same intensity of whisper and spoken voice, carefully standardized, was used by all members of the staff at Hoff Hospital.

One hundred twenty-three cases were selected for this report including 20 cases of malingering and simulation, 22 controls (cases of severe organic deafness) and 81 cases of psychogenic deafness. Pentothal was administered to 97 cases, including the 22 controls. Deaf patients were routinely interviewed by a psychologist assigned to the Otological Service and the problem cases were examined by a psychiatrist.

*Description of Narcosynthesis.* Our patients got to know each other, and the first dramatic cure made quite an impression on the others—they all wanted "a shot in the arm." Thereafter it became a matter of introducing those who needed the cure to those who had it. After several preliminary interviews with the psychologist, the patient walks to the operating room wearing his hearing aid. He is told that he will receive treatment and be cured of his deafness. Sodium pentothal (2½ to 5 per cent solution) is injected intravenously, while he counts backward from 100 to 99, 98, etc. It must be given slowly, about 0.1 gram per minute, for deaf patients have a tendency to go to sleep. The psychotherapeutic level is the twilight zone just between sleep and awakening. He becomes incoherent with 0.25 to 0.8 grams, may have reached a count of 50, is carried in a light so-called "babbling" state during the interview which may require 20-30 minutes of seminarcosis.

Under the quieting effect of the sedative, many repressions come to the surface. The ideas have a strong emotional basis (fear, anxiety, antipathy, feeling of guilt, etc.) and the turmoil of childhood, family life, and current situations intermingle. Information of diagnostic importance is obtained when the patient starts recounting these isolated memories, reliving them, becoming excited, and sometimes violent. The release of pent up feelings, "mental catharsis," usually is of therapeutic value and is known as abreaction. The reorientation of the ego and effect of the sedative may be lumped together under the term narcosynthesis.

Some speak spontaneously; others must be primed with questions or descriptions of a desired situation, repressed memories, or sound effects which test the presence of hearing (electrical recordings of combat noises). The doctor remains an observer unless the situation requires him to play an active role. The patient is aware that a doctor is present, must have confidence in him and needs to be supported by the belief that he has had ear treatment.

The hearing aid is removed when the patient starts responding to conversation. A loud voice is used, then a softer voice, and finally a whisper, testing each ear separately. The subject-matter must be of interest to the patient, and well-aimed remarks will demonstrate the threshold of hearing. Whisper and spoken voice tests, audiograms, and recorded speech perception tests were used with some degree of accuracy, but one must arouse and remind the patient all through the test. Such readings are fairly reliable, and sometimes more accurate in the hypnotic state than in the posthypnotic state. When good hearing is found, the patient is told that his hearing is normal, and that he will hear from now on. He is assured for 2-3 hours after awakening, is susceptible to suggestion for a day or two following the narcosis. In a few instances, when he becomes conscious of the fact that he is hearing, he may suddenly revert to a deafened condition. There may be an element of unresolved hysteria, or of conscious malingering in such cases, but one thing is certain, the otological diagnosis is established, and thereafter the psychiatrist continues with the treatment.

*Observations on Conversion Hysteria.* Everyone has seen a deaf individual take advantage of a situation and "turn a deaf ear" when it suits him. A similar reaction, carried to a psychopathologic state, is known as conversion hysteria. The term, conversion state, may be used and it appeals to me because in most instances there are few if any outward manifestations of hysteria other than the deafness. The true conversion states are apt to have an initial organic basis, the symptoms continuing long after the pathologic condition has disappeared. (Case 1.)

*Case* 1. *Psychic Deafness, Conversion State.* A 26 year old sergeant was wounded in action, November, 1944, and admitted to Hoff General Hospital three months later. He was injured by the blast of an exploding shell, had shrapnel wounds of the arm, and a ruptured right ear drum which bled for several days.

Otoscopic examination: normal ear drum on both sides, perforation healed. The audiograms were repeated six times and varied as much as 48% in the right ear and 17% in the left ear. Following is a record of his hearing:
Right ear 2/15 wv, 6/20 sv, audiometer loss 52%-71%-93%-97%-100%-94%
Left ear 2/15 wv, 7/20 sv, audiometer loss 82%-83%-82%-83%-97%-80%
His binaural loss for speech was 62 db. Vestibular function was found to be normal.

He was placed on the aural rehabilitation program, given a hearing aid, taught lip reading, and was rated as very good at it. He had typical symptoms of severe combat reaction: headaches, nervousness, tremulousness, and anxiety, and eventually was given pentothal (0.625 Gm.). He fell asleep, aroused a few minutes later, and began to mutter. Under seminarcosis his hearing was found to be normal; he responded to a soft spoken voice, revealed a strong religious conflict and war neurosis.

He was told that his hearing would now be cured. Ethyl chloride was sprayed upon his ear. He was told that from now on he would hear normally. First a loud tone of voice was used, gradually the voice was lowered, finally he was addressed in a soft conversational voice, from the opposite of the room, and from his responses it was evident that he was hearing. He relaxed and for a period of two hours told of many things that were disturbing him.

This sergeant was a hard working, conscientious farmer prior to entering the Army. He left school in the 12th grade, was active in community and church work, was married, had a wife and baby and on joining the Army, he became a good soldier and leader of men. His platoon followed him with devotion, and when his men were killed or maimed he took the matter seriously. His religious conscience bothered him a great deal and he was tormented with the faces of the enemy that he had killed, or buddies of his that had been killed through some imagined neglect on his part. He was in a fox hole when an artillery shell crashed down. His buddy was killed, and he found himself completely deaf.

He was reassured and told that he was forgiven for all the wrongs that he had done, that the war was over, that he could hear and that his men were safe now. His anxiety disappeared, and when the effects of the pentothal had worn off he was overjoyed to have regained

his hearing. He left the hospital at the end of three weeks with normal hearing in both ears and in excellent emotional condition. Comment: A study of the audiograms in this case reveals: (1) Variation in hearing acuity, (2) A flat curve, (3) The spoken and whisper voice tests do not coincide with audiograms.

There was no cause for the deafness other than a mental conviction that he could not hear. Whether this is shell shock, or some other condition, is immaterial. One thing is certain, it is not malingering. The character of this individual, the presence of great strain, combat fatigue, nervous tension, and initial deafness, is enough to explain this case. It is obvious that the blast of an exploding shell, blood oozing from the ear, and sudden temporary deafness offered a basis for conversion hysteria.

*Case 2. Pure Hysterical Deafness.* A 34 year old WAC became hard of hearing one month after entering the Army. Six months later she was totally deaf, unable to hear even with a powerful hearing aid, and the only way of communicating with her was by writing notes. She developed pseudo-lip-reading ability, was quite cooperative and willing to help the doctors. Otoscopy and caloric tests were normal. She had contracted visual fields, diminished corneal reflexes, and areas of anesthesia. After narcosynthesis she regained her hearing. She was the type who liked the center of the stage, was unhappy, and tense and anxious since her mother's death. Comment: This is the text-

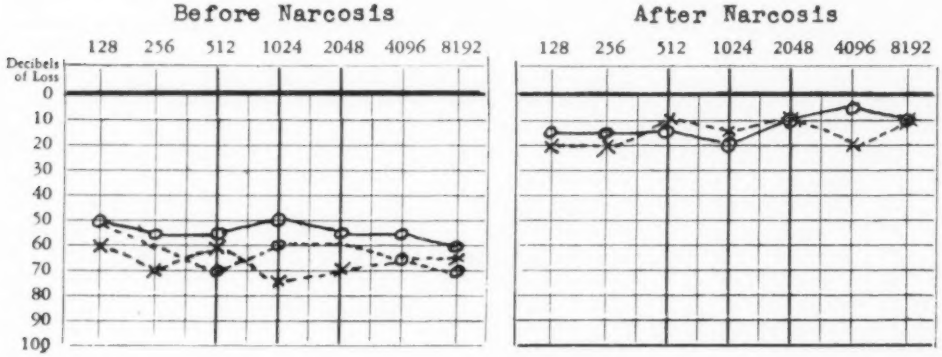



FIG. 1.—Case 1.

| | Wv | Sv | Audiometer loss | | | |
|---|---|---|---|---|---|---|
| Rt. | 2′ | 6′ | 52% | 71% | 97% | 100% |
| Lt. | 2′ | 7′ | 82% | 83% | 83% | 97% |

| | Wv | Sv | Loss |
|---|---|---|---|
| Rt. (o) | 15′ | 20′ | 2% |
| Lt. (x) | 15′ | 20′ | 1% |

Age 26: PURE CONVERSION STATE: WIA by shell blast, bled from rt. ear, still has tinnitus. MT is healed, caloric is normal, lip reading good. Narcosynthesis reveals severe war neurosis, strong religious conflict.

In the 1,500 cases at Hoff General Hospital, functional deafness was diagnosed in one per cent of cases prior to the use of pentothal, after that the figure rose to 12 per cent.

The notion that deafness of psychic origin is an extremely rare condition may be due to the fact that so little is known about it. The literature usually describes grotesque cases of hysterical deafness, cases in which the findings are so extraordinary as to preclude the possibility of any other diagnosis. (Case 2.) In actual practice, however, such cases are the exception rather than the rule. book case, was seen once in 75 cases of psychic deafness.

Functional deafness, in the majority of the cases, is built around actual ear disease, inflammation, injury or partial deafness, and this nucleus of half-truth interferes with exact otological diagnosis. More than one otologist has suspected malingering or hysteria in such cases but had no way of proving it, at least none that would satisfy him. These are the cases that a pentothal interview may uncover, and one may estimate the percentage of loss due to or-

ganic pathology and that due to the functional overlay. (Case 3.)

*Case 3. Organic Deafness with Functional Overlay.* A 23 year old AAF accountant with 21 months of duty confined to the U.S., had otitis media as a child which resulted in a large central perforation in the left MT, with an atrophic right MT and reddened promontory. He gave a history of poor hearing since childhood which had become worse since entering the Army. Examination showed a mixed type of deafness with inconsistent audiograms and discrepancy between the spoken voice and audiometric loss.

The spoken voice used in this test has an intensity of 55 db. at 5 ft., and at 3 ft. it is 60 db. It will be noted that this patient had audiometric losses which preclude hearing at the distances indicated. Repeated audiograms were performed and slight fluctuations were encountered. Because of these inconsistencies this patient was given pentothal. Under the sedative it was found that he could hear a whisper across the room. Audiograms and hearing tests upon awakening revealed good hearing in the right ear with a slight improvement in the left ear.

He had a true organic deafness in both ears, but there was at least 64 per cent of functional

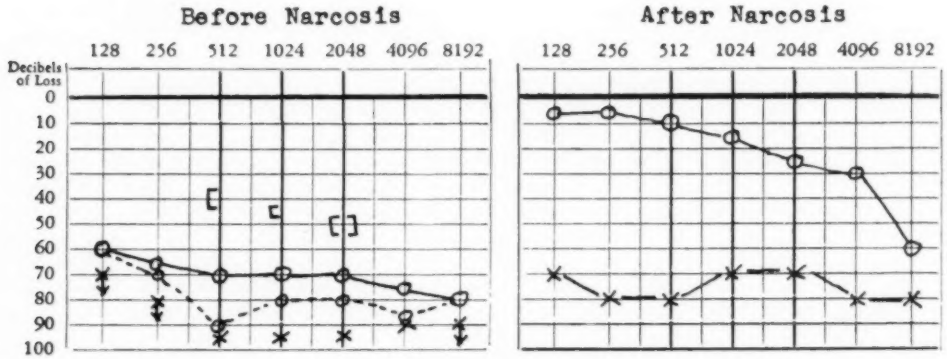



FIG. 2 — Case 3 —

| | | Wv | Sv | Loss | Wv | Sv | Loss |
|---|---|---|---|---|---|---|---|
| --------- | Rt. (o) OM CC | 0 | 5′ | 77% | 15′ | 20′ | 13% |
| --------- | Lt. (x) OM CP | 0 | 3′ | 100% | 0 | 3′ | 85% |

Deafness 12 years, chr. otitis media bilateral—large perforation left MT. Under seminarcosis he can hear Wv across the room with rt. ear. Conversion state.

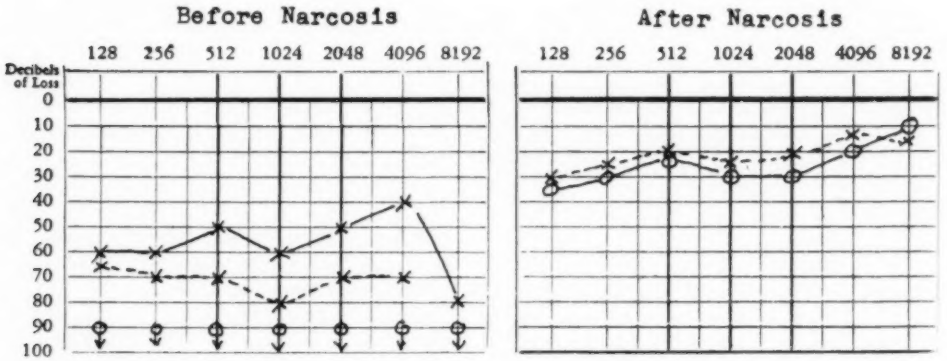



FIG. 3 — Case 4 —

| MT | Wv | Sv | Loss | | Wv | Sv | Loss |
|---|---|---|---|---|---|---|---|
| Rt. scarred (o) | 0 | 0 | 100% | OM CC | 10′ | 20′ | 13% |
| Lt. atroph. (x) | 0 | 0 | 31% | OM CC | 15′ | 20′ | 6% |

Age 26, paratrooper, poor hearing since parachute jump 2 years ago, a year later had otitis media for 6 mo., exposed to much artillery fire for 5 mo., tinnitus, vertigo; has his own hearing aid, good lip reader. Under pentothal hears pp Sv @ 15′ Mild chr. OM with functional overlay, anxiety conversion state.

overlay in the right ear. Comment: This is the type of case that is easily overlooked.

*Case 7. Conversion Hysteria, Fear and Guilt Complex.* A 20 year old corporal (colored) with 3 years of military service, worked as a blaster prior to induction, and after much blasting would notice that he could not hear for several hours. After entering the Army he became quite "ear conscious" and stated that explosions on the infiltration course had damaged his hearing. He was shipped overseas and shortly after arrival in the combat zone became totally deaf. He was returned to the U. S. and gave a vague history of the aggravation of the deafness "can't be sure." He was suspected of malingering because of the unexplained development of total deafness, was given pentothal, and soon began babbling about the dangers of the infiltration course, displayed a great deal of emotion and fear, eventually revealed his guilt as an extortionist and blackmailer. He used his rank to make soldiers under him "pay off" for returning late on pass, playing with crooked dice, and going to red light districts. He was voluble, called out to his victims in high emotion while under the pentothal, argued with them, but showed no signs of hearing. Finally the psychologist, hearing planes flying over the hospital, grasped the patient's arm and shouted

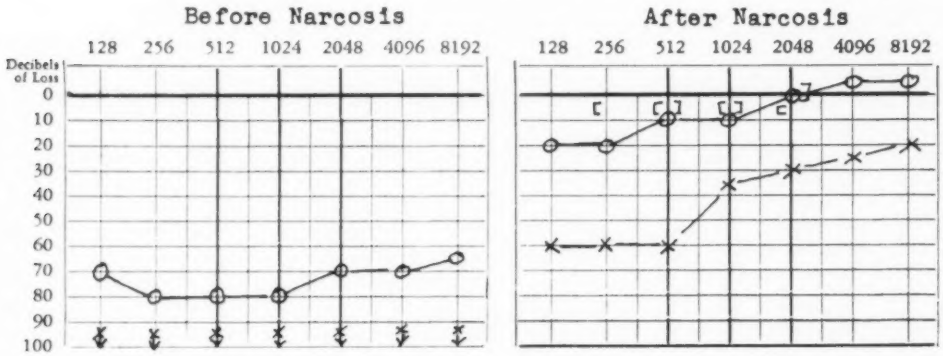



FIG. 4 — Case 5 —

| | | | Wv | Sv | Loss | | Wv | Sv | Loss |
|---|---|---|---|---|---|---|---|---|---|
| Rt. | (o) | ------- | 0 | 0 | 88% | ------ | 15′ | 20′ | 0% |
| Lt. | (x) | OM CP | 0 | 0 | 100% | OM CP | 2′ | 10′ | 30% |

Age 20, shell concussion produced large perforation left MT. He can not hear a loud shout. WIA 18 May, narcosis 18 July, final audiogram 23 July, '45. Under narcosis hears normally. Chronic otitis media with functional overlay, Conversion.

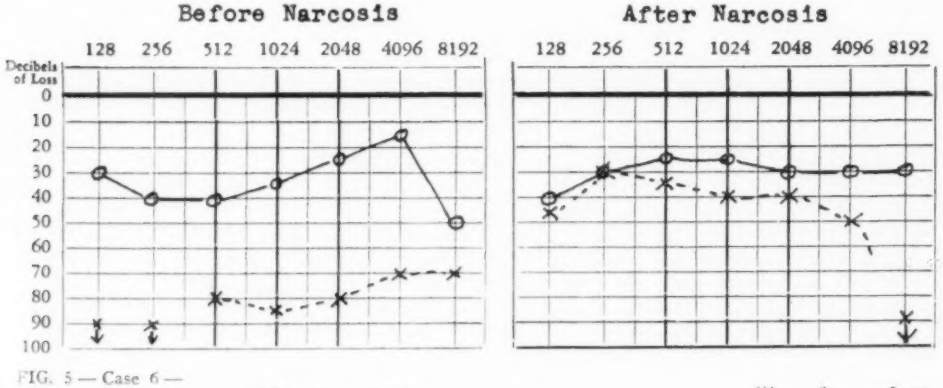



FIG. 5 — Case 6 —

| | | | Wv | Sv | Loss | | Wv | Sv | Loss |
|---|---|---|---|---|---|---|---|---|---|
| Rt. | (o) | ------- | 5′ | 9′ | 18% | ------ | 5′ | 20′ | 12% |
| Lt. | (x) | OM CP | 0 | 0 | 99% | OM CP | 2′ | 15′ | 27% |

Age 33, colored, 15 mo. Leyte, 5 mo. combat, explosion ammunition dump, bilateral otitis media, chronic central perforation left MT. Left hemicrania, numbness, vertigo. Narcosynthesis with violent reaction—fought Japs. Overjoyed when recovered his hearing. Functional overlay left ear only. Conversion state.

"Planes!" whereupon the patient jumped off the bed, ran for cover, cried out "Planes, planes, I hear them!" This was the first evidence that he could hear. He was told that his sins were forgiven, that he could hear, and this psychotherapy soon restored his hearing to a nearly normal level.

### DIAGNOSIS

The chief purpose of my discussion is to call attention to the differential diagnosis, describing as clearly as possible the symptoms and signs which indicate the use of pentothal or other psychiatric measures. Most significant are:

1. Obscure or sudden onset of perceptive deafness
2. Complete absence of hearing in both ears
3. Total absence of hearing in one ear; severe deafness in the other
4. Flat audiograms
5. Variation of audiograms

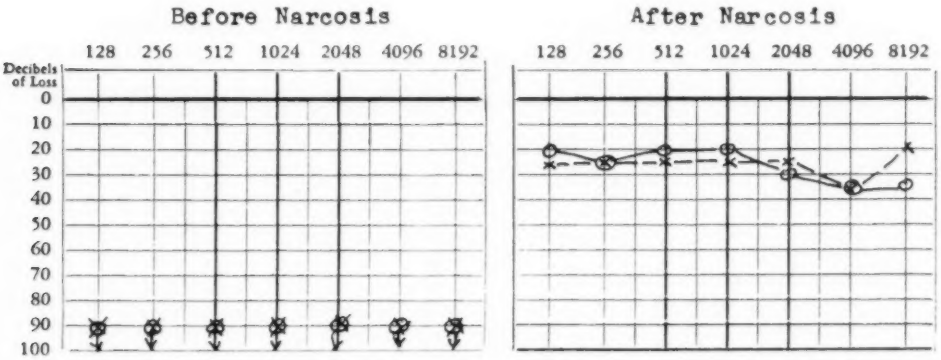



FIG. 6 — Case 7 —
Total deafness bilateral
Audiometric loss 12%

Age 20, colored corporal, indefinite "can't be sure" deaf for about a year. Under narcosynthesis reveals guilt complex, extortion and blackmail, fear of infiltration course and combat. Caloric reaction normal. Conversion hysteria.

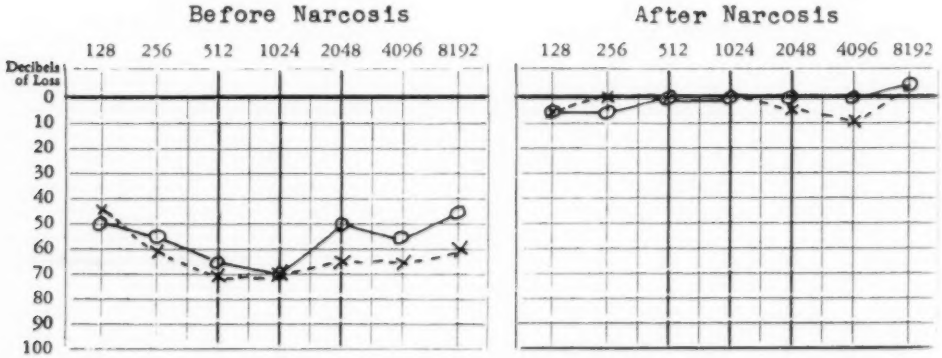



FIG. 7 — Case 8 —

| | Wv | Sv | Audiom-loss | | Wv | Sv | Audiom-loss |
|---|---|---|---|---|---|---|---|
| Rt. | 0 | 1′ | 60% | - - - - - | 15′ | 20′ | 0% |
| Lt. | 0 | 1′ | 76% | - - - - - | 15′ | 20′ | 0% |

Age 18 yrs., 9 months in the army, poor hearing for 8 years, had "scarlet fever" with otitis. Unable to hear a loud shout at 2 ft. NP: sick headaches, epilepsy petite mal, worried about mother who has cancer. Deafness became worse following tonsillectomy. Narcosynthesis produced dramatic result. Pure conversion hysteria.

6. Disproportion between hearing tests.
7. Normal vestibular responses
8. Binaural deafness, pathology only in one ear
9. Severe shock or emotional distress prior to onset
10. A sudden improvement in the hearing clinches the diagnosis.

The sum-total of findings must be evaluated, and reliance on any one finding is erroneous. The diagnosis of functional deafness is always difficult, especially when it is associated with an underlying organic ear disease, and frequently, although not always, acoustic trauma. Suspicion is aroused when unusual complexities, inconsistencies and

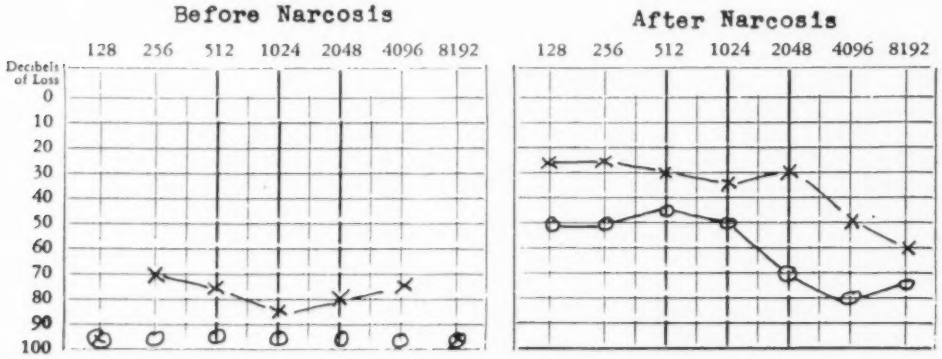



FIG. 8 — Case 9 —

| | Before Narcosis Wv | Sv | Audiometer loss | After Narcosis Wv | Sv | Audiometer loss |
|---|---|---|---|---|---|---|
| Rt. (o) | 0 | 0 | 100% | 15′ | 20′ | 54% |
| Lt. (x) | 0 | 0 | 93% | 15′ | 20′ | 18% |

Age 23, farmer, poor hearing since a child, shell struck house and he was unconscious several days, "stone deaf" for 2 weeks. M.T. normal, caloric normal. N.P.—tinnitus, headaches, tubular vision, tremulous. Narcosynthesis—quick response, much reaction, hearing returned. Interval between injury and treatment 6 months. Conversion hysteria, severe.

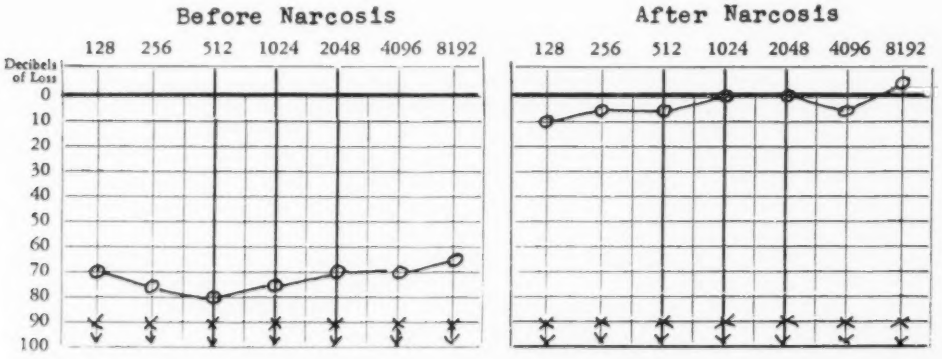



FIG. 9 — Case 10 —

| | Before Narcosis Wv | Sv | Loss | After Narcosis Wv | Sv | Loss |
|---|---|---|---|---|---|---|
| Rt. (o) ---------- | 0 | 0 | 85% | ---------- | 15′ | 20′ | 0% |
| Lt. (x) OM CP ---- | 0 | 0 | 100% | OM CP ---- | 0 | 0 | 100% |

Ten mo. combat. Saw female Nazi kill his buddy, rescued 16 wounded comrades at great personal danger, was bombed and strafed at St. Lo by his own planes, tank finally hit by a British rocket bomb, comatose for days, total deafness. Left mastoidectomy in childhood, left labyrinth hypoactive. Severe anxiety and hearing improved after narcosynthesis but developed amnesia which subsided. Conversion state—Organic Basis.

contradictions appear. In doubtful cases one must evaluate the possibilities of:

1. The emotional changes of organic deafness
2. Loss of hearing due to pure hysteria
3. Conversion states superimposed on organic ear disease
4. Malingering and simulation
5. Encephalopathy.

*General Appearance:* Mental suffering is characteristic of organic deafness. The average patient with hysterical deafness is not agitated. He looks and acts like an ordinary case of deafness. He is apt to be inconsistent, may respond only to a shout and then be seen playing cards or listening to the radio under circumstances which would require better hearing. He is not perturbed by the inconsistency and makes no pretenses one way or the other, unlike the malingerer who is very careful to avoid suspicion.

Patients with organic deafness complain bitterly of their disability — the

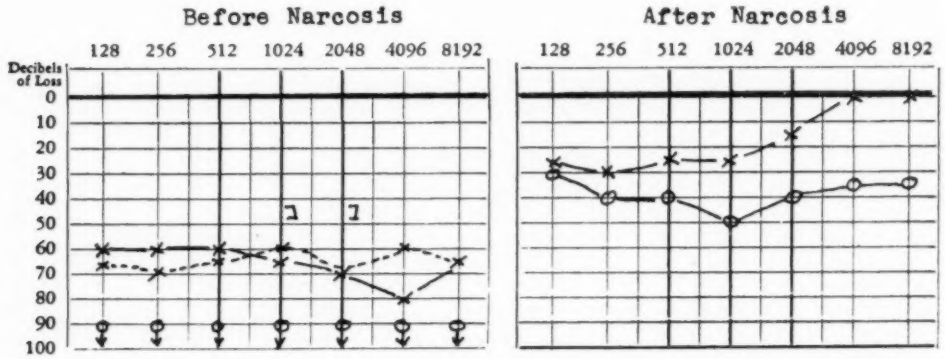



FIG. 10 — Case 11 —

Age 34, "ears closed up—never reopened" after rifle practice, constant tinnitus. MT opaque bilateral. Caloric normal. The onset is dubious—not sufficient acoustic trauma to cause a total deafness in one ear and severe deafness in the other. Seminarcosis reveals anxiety, fears shells and test hops in planes, hears soft spoken voice, conversion state—functional overlay on partial organic deafness.

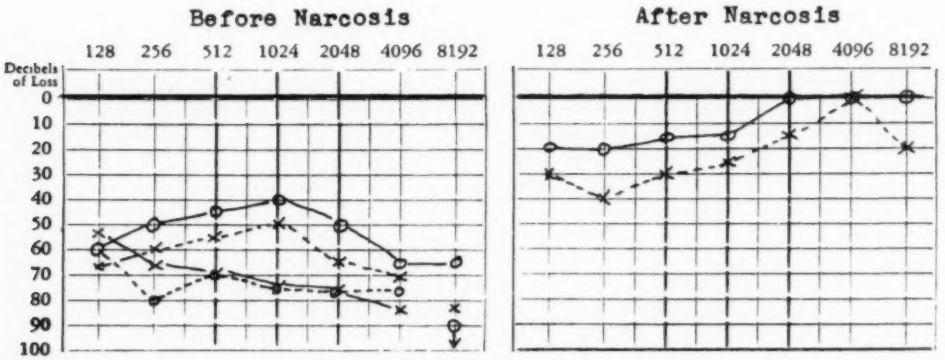



FIG. 11 — Case 12 —

| Wv | Sv | Audiometer loss | | | Actual hearing | Wv | Audiom. |
|---|---|---|---|---|---|---|---|
| 10′ | 15′ | 45% | 52% | 61% | Rt. ear (o) | 15′ | 2% |
| 5′ | 15′ | 61% | 84% | 73% | Lt. ear (x) | 15′ | 2% |

Age 28, farmer, fell off truck, headaches, vertigo, backaches, nervous. Under narcosis reveals violent reaction, persecution complex, remained in coma 24 hrs., answers to whisper. Was an excellent psuedo-lip-reader. Note wide fluctuations and inconsistencies.

hysterical cases are less concerned and seem to take the disability for granted. Some are quite placid and seemingly not very tense; others are dull and apathetic; and still others are nervous, show tension and tremors.

*History:* A doubtful history often suggests the diagnosis: rapid onset of deafness without obvious cause, or obscure and sudden aggravation of a partial deafness. A history of deafness following a head injury or acoustic trauma is laden with possibilities of hysteria. A history of great anxiety or nervous tension at the time of the onset is significant.

A study of 50 cases shows that 36 per cent gave a history of prior partial deafness up to 19 years in duration. A history of acoustic trauma occurred in 19 cases (38 per cent) and of this number 8 cases (16 per cent) had been wounded in action by a shattering blast, while 11 cases (22 per cent) were exposed to the noise of weapons, often of doubtful intensity.

*Tinnitus and Vertigo:* Tinnitus and vertigo are common complaints in psychic deafness. Tinnitus occurred in 20 per cent and vertigo in 8 per cent of cases.

*Audiograms in Functional Deafness:* The character and shape of the audiogram often suggests the diagnosis. The audiometer was not in existence during the last war, when so many cases of hysterical deafness were studied, and as a result, the literature makes no mention of this subject. The audiometer measures the variations which characterize hysteria with speed and accuracy, and the fluctuations can be observed as consecutive curves are plotted. Practically all cases of psychic deafness have flat audiometric profiles, such audiograms also occur in organic deafness, but are quite constant in hysteria. An audiogram with a classical high tone loss or good bone conduction speaks against the possibility of hysteria.

As a rule repeated audiograms performed on successive days may show variations of 10 to 70 per cent, the average being about 30 per cent. Fluctuations appeared in more than half of the cases and when this is taken together with the shape of the audiogram and the degree of loss, which usually is quite profound, it is almost pathognomonic.

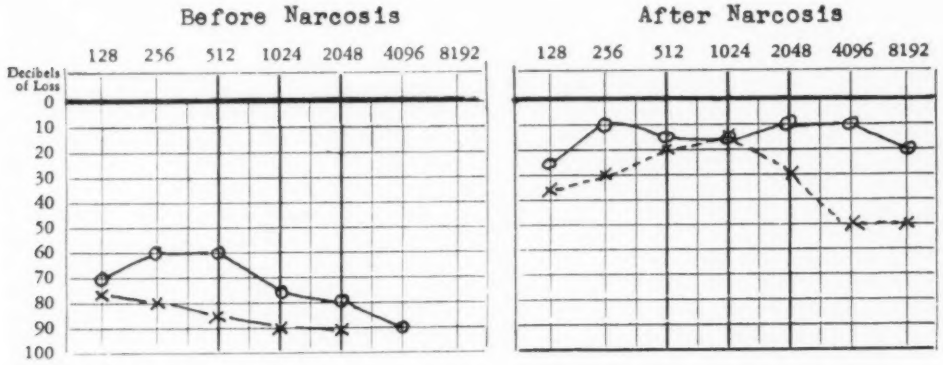



FIG. 12 — Case 13 — Sv 0/20 bilat. Rt. 88%, Lt. 100% Sv 20/20 Rt. 0% Lt. 10%

Age 25, poor hearing for 16 years, WIA concussion shell. Under pentothal reveals typical combat anxiety and neurosis—sees the Germans that he has killed, sees dead buddies, worries about his wife, his buddy can not sleep because enemy might sneak up on them and pt. would not hear them. Organic deafness with functional overlay.

*Severity of the Deafness:* It is difficult to delineate the precise disability because it is inconsistent, but I have taken 50 cases and attempted to define the amount of deafness in order that something concrete may be described.

| BETTER EAR | |
|---|---|
| Moderate Deafness (30-50 db.) | 34% of cases |
| Severe Deafness (50-85 db.) | 46% of cases |
| Total Deafness (85 db. or more) | 20% of cases |

RESULTS AFTER NARCOSYNTHESIS
50 CASES

| | Better Ear | Worse Ear |
|---|---|---|
| Normal hearing | 46% | 16% |
| Mild deafness | 30% | 26% |
| Moderate deafness | 24% | 30% |
| Severe deafness | 0% | 14% |
| Total deafness | 0% | 4% |

As has been stated, all of the cases had binaural deafness and the above figures are based on the hearing in the better of the two ears. A study of the worse ear shows much greater losses. In 50 per cent of the cases the worse ear was totally deaf, i.e., either had no hearing at all or showed a loss greater than 85 db.

There is a severe degree of deafness in 66 per cent of cases. The most striking cases are those with symptoms of total binaural deafness—20 per cent of cases. In hysteria the audiograms indicate more loss than the voice tests. The spoken voice portrays the actual disability. Just why the audiometric loss is greater than the spoken voice loss is not clear, but I presume that an instrument affects suggestible individuals. These two methods of testing must be used together and elimination of either one is a great mistake.

The power of suggestion is evident when it comes to the use of hearing aids. Some prefer very powerful 90 volt batteries, while others reject instruments and do not admit any assistance in hearing with them. A striking success with an instrument when one would not expect anything to be very effective is quite characteristic of hysteria.

*Vestibular Tests:* Vestibular responses are objective and beyond the control of the patient, therefore, when signs of an inactive labyrinth exist after three minutes of mass douche (68° F) an organic lesion is present. (Case 14.) The vestibular responses remain active in functional deafness and normal caloric reactions are obtained.

A spoken voice test, using a calculated intensity of sound, may be performed during the caloric douche. The water masks one ear and well-timed questions concerning the existence of vertigo and nausea may suddenly develop a "will to hear." Sometimes a malingerer after being confronted with this test may admit his error.

*Case* 14. *Inner Ear Deafness, Bilateral, Sudden Onset.* W. G., age 38 years, took "aspirin" for a severe headache and awoke with vertigo and deafness. The left ear is totally deaf, the right shows loss of 77 db. He has great difficulty even with a powerful hearing aid, is practically totally deaf.

Vestibular tests reveal a dead labyrinth on the left, the right is hypoactive.

Because of the total deafness, sudden onset, and possibility of a functional overlay, he was given 0.6 Gm. of pentothal intravenously. No psychiatric symptoms were uncovered, and in spite of intensive suggestion, his hearing remained unchanged. Comment: This case illustrates the importance of vestibular tests in the differential diagnosis.

*Voice Changes and Speech Defects:* The voice is of normal quality, although shouting may develop. As a rule speech defects do not appear in functional deafness. This is a fairly reliable sign but not infallible, especially in cases with a functional overlay. (Case 15.)

*Case* 15. *Partial Nerve Deafness, Speech Defect, Functional Overlay.* A 19 year old infantryman, 12 months of service, 6 months in the

SWPA, entered the Army with a diagnosis of defective hearing and a past history of hearing trouble while in school.

He had a speech defect for the sounds of H, S, D and T; presented a dull apathetic appearance, and was completely deaf. Hearing in both ears was reduced to 1/20 Sv; with a 60 db. loss bilateral. His hearing loss varied from 33 to 68 per cent on successive tests. He was given pentothal and after narcosynthesis his hearing improved. He appeared much brighter, more alert, and on one occasion when I was walking behind him, I spoke to him in an ordinary tone of voice and he turned around immediately, something he could not have done before. He was able to hear a whisper at a distance of ½ foot and a spoken voice at a distance of three feet. His final audiometric loss was 30 db., showed a high tone loss consistent with his speech. It is evident that he had an old partial deafness, cochlear type, with speech defect prior to conversion hysteria.

*Other Signs of Neurosis:* The significant findings which may be encountered occasionally are: corneal anesthesia, tubular vision, glove anesthesia of extremities, stuttering, vasomotor disturbances, reddening of the hands, etc. These findings are not always present but are helpful when they do occur; on the other hand they may be coincidental. They often differentiate psychic deafness from malingering and are not present in the latter condition. Tubular vision was seen in only 2 cases. One of the signs which is supposed to be characteristic of hysterical deafness is anesthesia of the external ear, but this rarely is seen.

## RESULTS

*Results Obtained with Narcosynthesis:* A dramatic restoration of hearing occurs in 90 per cent of the pure functional cases. In cases mixed with otologic pathology there was a remarkable improvement up to the level of the pathology.

Hearing was restored to useful levels in 76 per cent of the cases selected for treatment, which includes many cases with organic lesions. At this level the patient hears ordinary conversation at normal distances. This requires satisfactory hearing in one ear, a loss of acuity of not more than 30 db. in the better ear, or a response to the whisper voice at distances beyond 3 feet, or spoken voice 20/20.

An average improvement of 55 db. was obtained in cases of pure hysterical deafness, and a gain of 42 db. was obtained in cases with an underlying organic lesion.

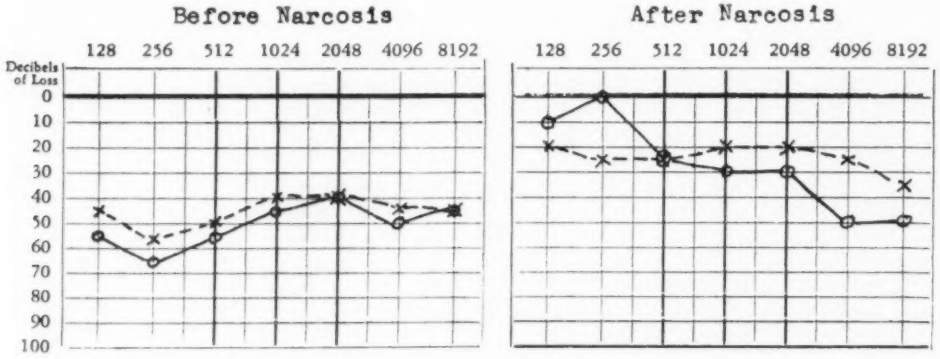



FIG. 13 — Case 15 —

| | Wv | Sv | Audiometer loss |
|---|---|---|---|
| Rt. | 0 | 1½′ | 37%-40%-62%-36% |
| Lt. | 0 | 1½′ | 52%-33%-68%-52% |

| Postnarc. | Wv | Sv | Audiom. |
|---|---|---|---|
| Rt. ear (o) | 3″ | 2′ | 16% |
| Lt. ear (x) | ½′ | 3′ | 7% |

Age 19: Speech defect for letters h-s-d-t. Functional overlay. 6 mo. SWPA. Long standing organic deafness with conversion state.

Hearing was restored to normal in both ears in 16 per cent of cases. These are the pure hysterical cases. Since the better ear is used in hearing it is important to note that in 46 per cent it was normal. The moderate degree of deafness that remained in 24 per cent of the cases, may be attributed to underlying organic deafness, residual hysteria, or malingering.

*Malingering and Simulation:* Malingering is one thing and hysterical deafness is another. One is a deliberate deception and the other is a disease. Both exaggerate existing otologic disturbance. All three conditions, ear disease, hysteria, and malingering, may coexist in the same patient but they are specific entities and must be regarded as such. The picture is confusing, but this does not alter the facts. Malingering occurs quite frequently and must be dealt with in a precise and scientific manner. Twenty cases of binaural simulation were encountered in a relatively short time and are mentioned here simply to clarify the picture of hysterical deafness.

The malingerer uses salesmanship, is cautious, tries to avoid inconsistencies, can be intimidated, and often retreats with excuses.

Inconsistencies appear sooner or later, and the otologist must confront the suspect with a discouraging insistence on accuracy of response, repeat as many tests as are indicated. Pentothal rarely is necessary, can be used as a last resort, is not foolproof; but anyone who is anxious to take it is not an ordinary malingerer. The chances are that he is convinced of his deafness, and in such cases we are dealing with a borderline condition. One of the first cases treated with pentothal was suspected of being a malingerer. He turned out to be an hysteric and developed a violent abreaction, emerging from the narcosynthesis with good hearing. The true malingerer resembles a normal person under pentothal anesthesia. (Case 16.)

*Case* 16. *Malingering.* This 36 year old soldier with 2½ years of military service was a salesman prior to entering the Army He was inducted with normal hearing and attributed his deafness to an airplane engine which ruptured his ear six months after entering the Army. He had evidence of an old otitis media. Both ear drums were retracted, thickened, inflamed and scarred.

Right ear: Wv 1 ft., Sv 4 ft., audiogram 89% loss.

Left ear: Wv 6 in., Sv 3 ft., loss 94%.

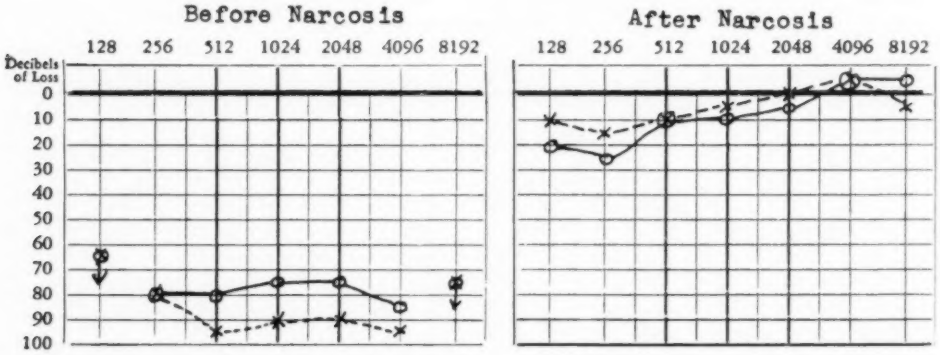



FIG. 14 — Case 17 —

| | | | | |
|---|---|---|---|---|
| Rt. | Sv 0/20 | 74% loss | Sv 20/20 | 0% loss |
| Lt. | Sv 0/20 | 86% loss | Sv 20/20 | 0% loss |

Age 19, salesman prior to induction, noise of motors on test block caused tinnitus, army service only one year. This much deafness from acoustic trauma is inconsistent. Suspected of malingering—he had no tremors, reflexes were normal, no tension or fatigue. Under pentothal hearing proved to be normal.

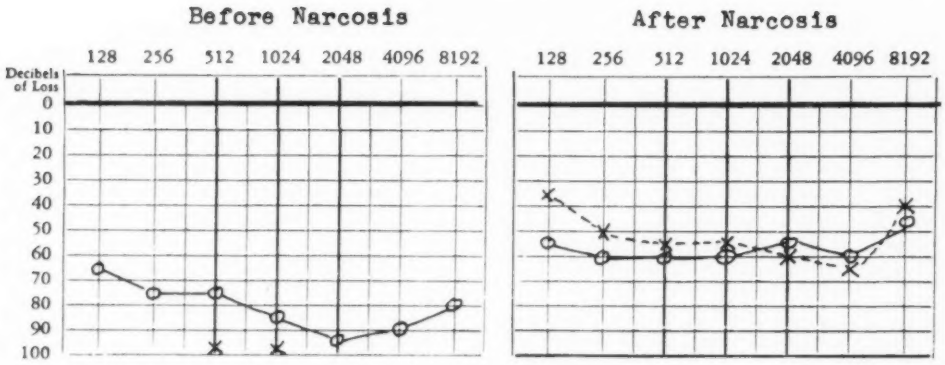



FIG. 15.—Case 18.—

| | Wv | Sv | Audiom. loss | | Wv | Sv | Audiom. loss |
|---|---|---|---|---|---|---|---|
| Rt. (o) | 0 | 1′ | 96% | 68% | 10′ | 20′ | 53% |
| Lt. (x) | 0 | 1′ | 100% | 100% | 2′ | 15′ | 57% |

Age 26, colored, poor hearing since a child, army 2½ years, deaf past 6 mo. MT, ET, Caloric normal. Under pentothal hears pp. Sv @ 10 ft. Upon awakening denies hearing. Later admits improvement. Malingering established. Hosp. 5 mo. ZI duty.

These findings are inconsistent. The audiometric loss precludes hearing either the whisper or spoken voice at the distance which he indicated. He seemed too cheerful and happy for a deaf man. He was given pentothal (0.75 Gm.) and it was found that under semi-narcosis he could hear the spoken voice at a distance of 10 ft. He was confronted with the facts; returned his hearing aid, and could hear very well without it.

Auto-suggestion plays a part in this triple combination. A partial deafness leads to malingering which in turn leads to auto-suggestion and thus by steps a condition similar to psychic deafness is reached. The power of suggestion can be reversed in those who are not severe psychoneurotics, and hearing is thus restored. I have seen such cases begin with a routine physical examination and go from one extreme to the other and back again.

*Control Cases:* Twenty-two cases of organic deafness, equally divided between otosclerosis and typical inner ear deafness, were given pentothal. These cases had anxiety and other neuroses. Their hearing tests and audiograms were consistent with ear pathology. Good bone conduction was present in the cases of otosclerosis. Hearing tests made before and after intensive psychotherapy was used failed to show any worth-while improvement. These patients, however, seemed to feel better and thought that they could hear better, but there was no objective change in the deafness. Not a single dramatic return of hearing appeared. These cases established once and for all that it takes an otologist to make the differential diagnosis. It must be remembered that 90 per cent of deafness is organic, that anxiety and tension and hysteria may exist in anyone, deaf or otherwise, but more often in the deaf. These findings alone are not to be trusted as signs of psychic deafness. (Cases 19 and 20.)

## DISCUSSION

Psychogenic deafness can be divided into two types: (1) pure psychic deafness, and (2) organic ear disturbances with a functional overlay. Type one occurs in about one-third of cases, and type two in two-thirds of the cases.

The incidence of psychogenic deafness is relatively high in rehabilitation centers which deal with marked bilateral deafness, and, in 500 consecutive

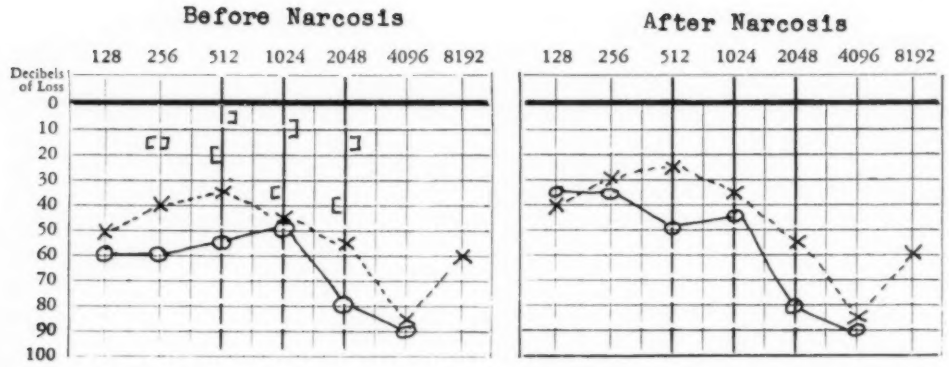



FIG. 16 — Case 19 —

| | | | | |
|---|---|---|---|---|
| Rt. | — very large perforation MT | Wv 0/15 | Sv 0/20 | 64% |
| Lt. | — very large perforation MT | Wv 0/15 | Sv 2/20 | 44% |

Age 32, explosion booby trap, bilateral chronic otitis media since, speech loss 44-52 db., given pentothal without results. Has a mixed deafness with good bone conduction. CONTROL.

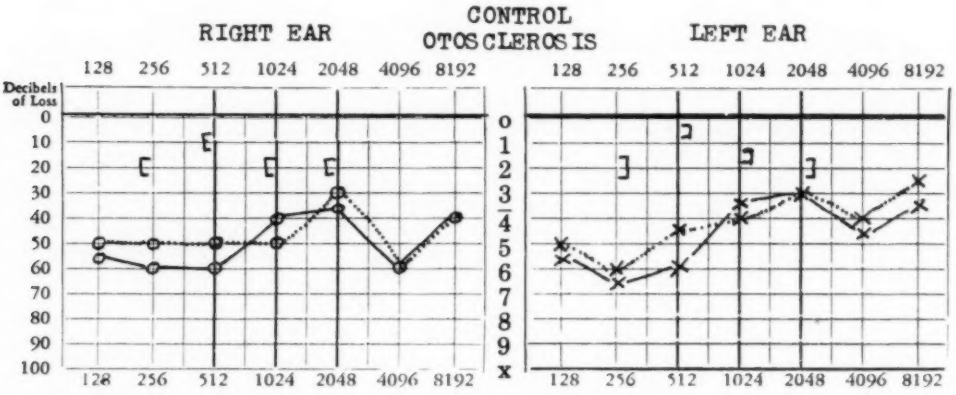



FIG. 17 — Case 20 —

Age 24, progressive deafness 2 years, tinnitus 7 mos., slight tension—nervous, MT normal, hyperemic promontory. Given pentothal, no change in hearing. (Before ————, After . . . . . .)

admissions, was found in 54 cases. In a casual survey of 1,500 ordinary otologic cases, under my care in three other general hospitals, I saw it only four or five times. This corresponds to what one may see in private practice where all sorts of minor hearing defects are encountered.

Conversion states occur in cases of chronic otitis media, and I have no doubt that this is true of otosclerosis as well. A significant number of individuals develop psychic deafness in everyday life, and while it is obvious that such conditions appear more frequently under the stress of war, it is well to remember that there is much anxiety and latent hysteria in peacetime.

I have seen at least 3 cases in civil life. One was a kleptomaniac who was deaf for speech, but heard the tones of the audiometer normally. She liked music, and the pure tones of the audiometer interested her, but she did not care to hear what people had to say, and for obvious reasons. The second case is that of a young man whose mother married an Indian which caused much comment

in the community and resulted in a sudden and complete deafness on his part. The third is the case of an elderly lady who immediately after being told of her husband's death developed a sudden and total deafness which has persisted for many years.

Shambaugh[12] mentions the case of a student nurse who recovered from her deafness when she gave up nurse's training. Kopetzky[8] mentions the psychogenic overlay in elderly persons with presbycusis. Fromm[4] recently reported the case of a ten year old boy who developed otitis media at 2½ years of age, became a deaf-mute and for eight years thereafter showed no signs of hearing or speaking. Fromm improvised hearing tests, and concluded that this was indeed a case of psychic deafness, that the boy was neither totally deaf nor pretending to be so, that he did hear sounds which interested him and did not hear sounds which he feared or disliked. Froeschels[3] recently reported 3 other cases and stated that he had seen 12 cases of psychic deafness in children. There are many other reports in the older literature and there is no need to review them.

Pseudo-lip-reading may be detected by silent tests. Individuals with functional deafness appear to be good lip readers in 50 per cent of cases, the remainder are only fair at it. The psychology of this condition is due to the fact that seeing and hearing re-enforce each other, as I have determined by some personal experiments. It is interesting to note that motion picture sound technicians take this into consideration when recording for the movies. It is necessary to add that doubtful cases of deafness should not be given lip-reading and placed on a program suggesting permanent disability until it is certain that they actually have organic deafness. Suggestible individuals remain deaf in such an atmopshere.

I first began to believe that much deafness might be psychogenic after observing the methods used in acoustic training. In this work an effort is made to re-educate the residual hearing by acoustic stimulation, music, speech, and sounds. Much time is spent with patients by workers in this field, and their interest, attention and enthusiasm is of some psychotherapeutic benefit. On the other hand, a number of individuals have made claims, and have cases to prove it, that special acoustic vibrations cured deafness. There can be little doubt that they chanced to find an hysterical deafness and by force of personality and much noise did effect a restoration of hearing. The same applies to other bizzare methods of treating deafness. Atkinson[1] cities such an experience.

Whether psychic deafness is due to inhibition, suppression, or mental block, is a matter which psychologists may decide. All we need to know as otologists, is that there is a subconscious interference with the perception of certain intensities of sound, regardless of whether it enters the ear by air conduction or bone conduction, and from the fact that both routes are impaired we conclude that a mixed, or, in the vast majority of cases, a perceptive type of deafness is present.

Individuals with psychic deafness fail to comment on sounds of strong intensity and seldom show reactions to it. They tolerate hearing aids with excessive volume. The shrill tones of the audiometer may be turned on to full intensity and permitted to remain there for considerable period of time without causing any apparent annoyance. Hurst[6] found it quite impossible to waken two patients who were suffering from total hysterical deafness by shouting within a foot of their heads.

Noise is undesired sound and we normally are not aware of hearing a certain amount of it; and it seems that

psychic deafness is a similar process exaggerated to a pathologic degree. A soldier deafened by concussion finds that he cannot hear and ceases trying to listen. When he makes no further effort to listen there may be changes in cerebration which suppress the perception of sound.

Acoustic trauma sustained by soldiers who are under the strain of constant fatigue, anxiety and tension is more apt to be associated with concomitant psychologic deafness. Deafness caused by high explosives occurred in 20 per cent of the cases that were sent to rehabilitation centers. One-third of the cases of severe blast deafness had a psychic overlay. Fear of noise and falling are instinctive even in infants and it seems to me that "normal" men may develop deafness with psychologic complications when a shattering blast rends earth and sky. Rupture of the ear drum occurred in most of the cases, and in 60 per cent there was complete healing of the perforation. The rest had chronic otitis media, but in spite of this their hearing was restored to satisfactory levels. The combat cases are mentioned frequently because they are interesting and instructive, but the majority of cases of functional deafness appeared behind the lines and one-third were at home in the States.

According to Zilboorg,[15] who made a review of psychiatry in the Soviet Army, hysterical deaf-mutism was observed more frequently than any other war neuroses. They could only communicate by writing notes, had absolute mutism, and were unable to produce the slightest sound. (Our soldiers, at least were articulate, which may be a G.I. peculiarity.) It is interesting to note that psychic deaf-mutism appeared in 50 per cent of their cases of concussion deafness. This figure is similar to what we found, except that we had no mutes. Giliarovski[15] apparently drew a distinction between wartime and peacetime, indicating that in peacetime psychic deafness may exist alone.

They gave much thought to differential diagnosis by objective tests, and in the tradition of Pavlov, various workers (Slivko, Strelinkhin, Mizrukhin[15]) developed conditioned reflexes to sound in patients with functional deafness and thus established the diagnosis. Zalkind[15] utilized the cochleo-palpebral reflex, striking a gong at a distance of three feet, and noted that the patient usually closed his eyes or moved his head. This observation contradicts Hurst,[6] and I presume that the intensity of the sound is a source of error. In any case of total deafness whether due to organic disease or hysteria, the auditory motor-reflex is absent according to Hurst. If the reflex is obtained and the patient says that he can hear nothing at all, he probably is malingering, but as long as he admits that he can hear something, the test does not help much in distinguishing between organic deafness, hysterical deafness and malingering. In partial deafness, therefore, whether organic or hysterical, as long as a sound can be heard at all it may produce a reflex. The question of malingering was not mentioned in the abstract by Zilboorg. Hurst[6] remarks that psychic deafness when associated with mutism is easy to treat. The hearing returns when speech is restored. Ioffe[15] studied 114 cases of psychic deaf-muteness, reported 70 per cent complete recoveries, and indicated 86 per cent satisfactory recoveries. According to Zilboorg[15] they used alcohol, but favored ether as a psychotherapeutic drug, and reported good results in the second stage of anesthesia (excitement). The deaf-mutes started to talk in this stage, were kept talking till out of the ether, and when conscious they started laughing, crying and trembling. Their speech was imperfect at first, and

78 per cent of cases went through a period of stammering.

A high rate of cure has been reported by many observers at home and abroad. The prognosis is excellent in pure hysterical deafness, in patients with intelligence and a normal background devoid of serious neuroses. It is not so good in life-long neurotics, those who cannot adapt themselves to the world about them, and the unintelligent. I have seen cases subside spontaneously and others persist for years when treatment was denied.

The psychic element often interferes with evaluation of otologic procedures, either produces failures or unusual results which are contrary to the principles of physics. Such inconsistencies are apparent in reports of the fenestration operation. Potent suggestion sometimes causes a remarkable improvement in the other ear. On the other hand failures may develop in neurotic individuals who have subconsciously developed a functional overlay or a conversion state. The solution to these problems is difficult, but pentothal may help, at least one should consider the possibilities. This applies to many otologic conditions. Our problem is to recognize the functional element and to be aware of its existence. Severe deafness requires more than a mere routine ear examination. Psychic deafness may run a protracted course and there is no way of knowing how many remain deaf for life. The importance of correct diagnosis is evident from the high rate of good results in treated cases. These are not new ideas, they have been known for many years and many otologists have expressed their opinions on this subject. The deaf are highly suggestible, show anxiety and neurotic traits.

When organic pathology is present, the functional overlay is more resistant to treatment, but we have succeeded in treating it. Even when pure hysterical deafness disappears, the patients find it difficult to hear at times, and act like people who are thinking about something else until one jolts them a bit.

The diagnosis of functional deafness* is primarily an otologic problem. The otologist is the one who is first consulted and he must make the initial differential diagnosis, and if he states that a cure is possible, the patient will believe him, and thus be more amenable to psychotherapy. The psychiatrist cannot evaluate the picture without the otologist, and if left to his own devices, attempts to treat frank cases of nerve deafness and cases of otosclerosis because of associated psychic disturbances, and in every instance where this was done, the most intensive psychotherapy, with and without pentothal, was a failure as far as actual hearing restoration was concerned, although the patients thought that they heard better. The otologist, on the other hand, is able to weigh the facts which fit into the patterns of deafness, and by examination of the ear, differential hearing tests, air conduction, bone conduction, spoken and whisper voice, repeated audiograms, and vestibular tests he is able to rule out the organic cases. Familiarity with these procedures and the fundamentals of psychiatry leads to segregation of the otogenic and psychogenic factors in deafness. Whether or not the psychic disorders are amenable to treatment is a matter which the psychiatrist is in a better position to determine.

* The term, functional deafness, simply indicates that deafness is not due to organic ear disease, but may be due to malingering, hysteria, etc. The term, hysterical deafness, indicates that a true neurosis is present. The term psychic deafness is used in the sense that the disability may be psychologic. I find myself using this term because it is short and may be applied to cases which lack definite signs of hysteria. Psychosomatic disorders produce peripheral organic dysfunction (diarrhea, hypertension, etc.) and even morphologic changes (gastric ulcers, etc.), therefore this term cannot be used for deafness which is caused by a central denial of sensation rather than a pathologic change in the ear.

Team-work is conducive to the best results in diagnosis and treatment, but there is much that the otologist can do with psychotherapeutic measures, and he should not hesitate to use them. He must realize, however, that unstable individuals develop other neurotic disturbances and that for permanent results the patient must gain insight which only a trained psychiatrist can give. There are some conditions which respond to simple measures and others which require prolonged psychotherapy.

Pentothal is particularly suitable for the management of functional deafness. Other technics, such as psycho-analysis, require the writing of notes and this has obvious disadvantages. The use of hypnotism has proven to be difficult. Hurst[6] and other workers attempted to use it but without success. I recently compared a case of hysterical blindness and one of hysterical deafness treated one day apart by the same psychiatrist. He used hypnotism on both patients, cured the hysterical blindness within 15 minutes but failed after 45 minutes of effort in the treatment of the hysterical deafness. Deafness erects a barrier, interferes with communication, and hampers conversation. The use of intravenous pentothal breaks these barriers, is rapid and effective.

Ten thousand cases of deafness have passed through the Army rehabilitation program. It is estimated that 200,000 will apply to the Veterans' Bureau. If one in ten may be psychogenic it is estimated that anywhere from 0 to 20,000 cases of functional deafness may be seen by the Veterans' Bureau. The problem in long standing cases will be more difficult, especially where compensation is involved.

## CONCLUSIONS

Most deafness is organic in origin, but a significant number of individuals develop a functional overlay, become an unnecessary burden to themselves and the community, and as with all psychiatric disorders, there is an increased incidence due to the stress of war.

Experience in an Army center for the rehabilitation of the deafened, indicates that 1 out of 10 cases of severe binaural deafness with 30 db. or more of hearing loss may be psychic in origin. The majority of such cases appear to be due to a subconscious inhibition of the perception of sound. The otologic findings in typical cases are those of a severe perceptive type of deafness. Total deafness occurred in 20 per cent of cases. Organic ear disease was present in 60 per cent of cases. Suspicion is aroused when repeated audiograms show variations, or when there is an inconsistency between the ear pathology, the audiogram and spoken voice tests.

The use of narcosynthesis has resulted in a dramatic improvement of hearing in 76 per cent of cases. Experience with 81 cases has demonstrated the fact that the differential diagnosis must be made by otologists, that a knowledge of the pathology of the ear is a necessary prerequisite, that precise hearing tests are mandatory and that a final and conclusive diagnosis cannot be made until the hearing has been restored to a useful, or satisfactory, if not normal level.

The audiogram is flat, and the hearing loss by audiometry is greater than by voice tests. Both methods of testing must be employed. Audiograms must be repeated in every case of deafness to determine the accuracy of response.

The otologist, when a doubtful case of severe or total deafness is encountered, must pass everything in review and search for signs of a functional disorder, attempt to discover some discrepancy and, having found one, must then look for others. In doubtful deafness, without obvious evidence of hysteria, the diagnosis must be guarded. The differential diagnosis between mal-

ingering and hysteria is sometimes quite difficult, but can be accomplished in most instances. Both conditions may coexist, but they are not synonomous.

Having seen cases of functional deafness in private practice, and having seen many more in the Army, I am of the opinion that the study of this condition is not only a problem of considerable importance in war veterans, but also in the population at large; that classical text-book cases of hysterical deafness are exceptional; that the most common cases are those with an underlying organic ear disease, otitis media, or partial deafness; that such conditions add to the complexity of the diagnosis; and that the pentothal interview has facilitated the evaluation of the functional overlay in such cases.

I have seen some cases subside spontaneously and known of others that persist for many years, and believe that bizarre reports of cures by methods which are inconsistent with the known facts of otology, are in all probability manifestations of this ubiquitous disorder.

## REFERENCES


1. Atkinson, Miles: Psychogenic factors (hysterical and emotional), Tr. Am. Otol. Soc., pp. 260-266, 1937.
2. Crowe, Samuel J., Guild, Stacy R., and Polvogt, Leroy M.: Observations on the the pathology of high tone deafness, Bull. Johns Hopkins Hosp., 54:315-379 (May) 1934.
3. Froeschels, Emil: Psychic deafness in children, Arch. Neurol. & Psychiat., 51:544-549 (June) 1944.
4. Fromm, Erika O.: Study of a case of pseudo deaf-muteness (psychic deafness), J. Nerv. & Ment. Dis., 103:37-59, 1946.
5. Grinker, Roy R. and Spiegel, John P.: War neuroses, Blakiston Co., Philadelphia, 1945.
6. Hurst, Arthur F. and Miller E.: Discussion on functional deafness, J. Laryng. & Otol., 55:393-404 (August) 1940.
7. Knapp, Peter H.: Emotional aspects of deafness, Unpublished manuscript, (February) 1946.
8. Kopetzky, Samuel J.: Psychosomatic factors in loss of hearing, in Diseases of the nose, throat and ear, Jackson and Jackson, W. B. Saunders Co., Philadelphia, p. 277, 1945.
9. Martin, Norvil A.: Psychogenic deafness, Ann. Otol., Rhin. & Laryng., 55:81-89 (March) 1946.
10. Rosenberger, A. I. and Moore, J. H.: Treatment of hysterical deafness at Hoff General Hospital, Am. J. Psychiat., 102: 666-669 (March) 1946.
11. Sabine, Paul E.: On estimating the percentage loss of useful hearing, Tr. Am. Acad. Ophth., p. 179-187 (March-April) 1942.
12. Shambaugh, George E., Jr. and Roberts, W. S.: Chronic progressive deafness including otosclerosis and diseases of the inner ear, Arch. Otolaryng., 35:291-331 (February) 1942.
13. Truex, Edward H., Jr., Unpublished manuscript and personal communications, (July) 1946.
14. Work, Walter: Personal observation made by the writer (February) 1945.
15. Zilboorg, Gregory: Some aspects of psychiatry in the U.S.S.R., Am. Rev. Soviet Med., 1:562-575 (August) 1944.


## DISCUSSIONS

GORDON D. HOOPLE, M.D., Syracuse, N. Y.: Dr. Semenov has called our attention to a malady of hearing and a possible treatment for the same about which most of us know little. I have had no experience with narcosynthesis. Perhaps it is best that one of the discussers of Dr. Semenov's paper falls into that category, for an unbiased comment is thus made possible.

As I reviewed the paper, I found several points which I think shou'd be stressed. A psychogenically deaf person must not be confused with a malingerer. This seems to me to be important. These I believe are distinct entities and any attempt to place the psychogenically deaf in the same class with the malingerers and to apply the methods of handling the latter to the former will only end in failure and disappointment.

Diagnosis of the psychogenically deaf is not easy, and the possibility of its presence must be kept constantly in mind or cases will be overlooked. The history or the attitude of the patient may reveal some things which should make the examiner aware of this possible diagnosis. These patients present a binaural deafness of marked degree, but irregularities in the hearing tests, such as inconsistent findings in repeat audiograms or uncorrelated pure tone

and speech perception tests should put one on guard.

I did not see the audiograms Dr. Semenov has shown prior to this morning. I had considerable experience in testing the hearing of soldiers in this war, and one thing that stood out as I saw them was that most of these men complained of exposure to gun fire or blast injury, and their audiograms showed a typical dip at 4096. In Dr. Semenov's cases there was no dip at 4096, in any instance. Most of them had flat curves. This is another inconsistency which should put the examiner on guard, particularly if there is that history.

There are usually no speech defects, as Dr. Semenov remarked, though there is complaint of a marked degree of deafness. Only when there is organic otologic disease of some standing with overlying functional deafness should speech defects enter the picture.

I admit with some chagrin that prior to the war I had never made the diagnosis of uncomplicated psychogenic deafness in the course of my practice. Within the past two months I have seen 2 cases and I discovered them only because I was alive to the possibility of their existence. I am certain we should all find some of these patients in our practices if we are on the lookout for them, but I wish to emphasize it is my belief we shall find no such numbers in civilian practice as might be indicated by this presentation of the military picture.

Dr. Semenov has told us he encountered the psychogenic deaf in 12 per cent of 500 consecutive readmissions to an aural rehabilitation hospital. Those who have had experience with military life know, with its frustrations and unnatural environments, there are many more psychogenic factors in such an existence than in civilian life, though goodness knows, what with the meat shortages, atomic bombs, and the women—God bless them—the possibilities of functional disorders in ordinary life are rapidly mounting. In our complex world we shall see an increasing number of these patients, but for most of us they will be only occasional. If we can discover them and then treat them as successfully as Dr. Semenov indicates in this presentation, we shall owe a debt to him and to other workers in this field for calling this condition to our attention.

EDWARD H. TRUEX, M.D., Hartford, Conn.: I congratulate Dr. Semenov on this excellent presentation and the comprehensive nature of his observations on functional deafness.

All of us in the military services who had contact with these cases were deeply impressed by the large numbers that were encountered and by the dramatic results of therapy.

It is worthy of note that in every case psychic trauma is the precipitating factor, usually associated with or preceded by aural symptoms, which serve as the focal point for the conversion phenomenon.

Psychic trauma is not measurable other than in terms of the reactions that it induces and the nature of these reactions is dependent upon the emotional development and stability of the individual affected. Dr. Semenov has cited cases (and there are many of them) who developed functional deafness in this country, far from the stress of combat and with little or no apparent cause. In this connection the important fact to bear in mind is that a circumstance or situation which is quite benign for most of us may be physically catastrophic for others with an occult or manifest neurasthenia.

The conversion phenomenon itself is really an escape mechanism of subconscious origin wherein the mind directs the body to accept punishment of a lesser magnitude in order to avoid or to escape punishment of greater magnitude. The combat soldier, for example, accepts deafness in order to escape the emotional turmoil of impending death, and the trainee in this country accepts deafness, for example, to escape the lashing tongue of his top sergeant which to him is more severe than deafness.

The treatment of functional deafness, or of any functional abnormality, is three-fold:

1. It necessitates a purging of the subconscious mind in order to bring to the surface the causative factors.

2. A removal of the conversion symptoms through suggestion and elucidation.

3. The development of insight to establish for the patient the relationship between the cause and symptoms.

Dr. Semenov has not brought out forcibly enough the importance of this third category of treatment. As he has shown, it is relatively easy in most cases to penetrate the crust of hysteria through the medium of suggestion with the aid of barbiturates and produce a symptomatic cure. To insure the permanency of the cure and the prevention of conversion to other organs of the body, it is absolutely imperative, in my opinion, that a complete understanding of the dynamics of the symptoms be given to the patient. This is, indeed, a laborious task for a well-trained psychiatrist, but believe me, as he stated, many patients will not accept it unless it comes from the ear doctor. Many patients are inclined to regard the psychiatrist's methods as a quasi-fraudulent endeavor.

One other point that I wish to make bears upon the incidence of psychogenic deafness. The figures that Dr. Semenov has given represent those cases in military centers that were recognized. I believe that many others escaped detection, even some among those who were considered normal after pentothal therapy. The absence of an abreaction under narcosynthesis does not rule out conversion symptoms.

To illustrate my point, I wish to cite very briefly one case. It is that of a twenty-seven year old captain in the Rangers. His history upon admission to the hospital in October of 1945 was not unusual. Bilateral hearing loss having resulted from a shell burst which momentarily rendered him unconscious. This occurred in September, 1944. He had been wearing a hearing aid with reasonable satisfaction since February, 1945. This was eight months prior to admission to the hospital, but since the onset the hearing loss had been getting progressively worse. On examination, both ear drums were thickened, deeply scarred, and immobile. He reacted normally to caloric tests. (Slide) His hearing loss, however, as you can see here, was approximately 78 decibels for speech reception and about 95 decibels in the left ear in the pure tone speech range. He had no hearing on the right side.

During the first two or three pentothal interviews, carried out by the psychiatrist, no unusual reactions could be elicited. Many factors, however, indicated a functional component in his hearing deficiency. We thought that he did have an organic hearing loss and that he had a functional overlay. During the third interview under barbiturates, he abreacted clearly, giving vent to hate, fears and conflicts that went back to his early childhood.

Strong suggestive therapy was used along with faradic stimulation over the mastoids, but it was ineffective. After sixty hours of psychoanalytic interview and during the eighth session under pentothal, some three months after entering the hospital his hearing was suddenly and dramatically restored to normal.

Again my congratulations to Dr. Semenov. Thank you.

W. L. POST, M.D., Joplin, Mo.: I feel that this paper is very timely. Those of us who were in a position to see psychiatric cases in the service will realize that especially. As for myself, I spent eighteen months with the Air Forces Hospital for Teaching Psychiatrists, and for that reason I have a different viewpoint from that of the essayist and the following discussers.

We naturally had a select group of psychiatric cases, but none of them presented what the essayist has so far dealt with, predominant ear symptoms. Our patient load was not large, but we covered everything from simple fatigue to the most intricate of psychiatric conditions. That was necessary in a teaching hospital. Naturally, I formed some conclusions, as every patient who made a complaint, and every patient who was seen by a ward officer to have some evidence, whether he complained of it or not, of a hearing defect was sent to the Ear and Throat Department, evaluated, and then was followed through receipt of the definite psychiatric treatment, which was based on narcosynthesis, as you may know, if you have read the writings of Dr. Grinker and his colleagues in "Men Under Stress", which I may recommend to any of you who wish. It is a very interesting and easily read book on the psychiatric background of this problem.

We found a great many minor hearing losses, particularly in those cases where the battered ego was trying to recede from reality. This is not a true conversion and these patients showed all of the other psychiatric symptoms either of anxiety state or of simple depression. When their problem was explained to them through psychiatric therapy, we found that practically no ear therapy was necessary, excepting reassurance and simple explanation of mechanisms, but the psychiatrist had first to get over to them their true or basic problem. That is the place where narcosynthesis is invaluable, as Dr. Semenov has so very ably shown you. I feel that we must remember this, because I am finding psychiatric causative factors in veterans today coming to my office with hearing difficulties. I am also seeing the same difficulties in industrial cases, particularly blast injuries.

It is too bad this paper does not go into other ear symptoms. We saw pseudo vertigo in severe conversion states. We also had a great deal of otalgia following blast injuries, which again was cleared by proper psychiatric therapy and by reaching the problem through narcosynthesis. I must again emphasize the real cure is not just proving to the patient that he really can hear, but his problem must always be worked out with him on the level of the underlying conflict; otherwise he will not receive sufficient ego support to return and become a permanently useful citizen. His conflict will break out somewhere else. Maybe he will lose the use of an arm. Thank you.

STEWART WOLF, M.D., New York: I thought that it might be of interest to the members of the Academy to know of similar findings in

the case of partial blindness, particularly with pericentral and central scotomata and constriction of the visual fields. In the Ninth General Hospital, stationed in the South Pacific, where prisoners, having been released from three years of imprisonment, under the Japanese, were seen, Dr. Frank Constantine, of this Academy, recognized optic atrophies secondary to something which went on during the three years of imprisonment. In many individuals, however, there occurred visual field defects, both central and peripheral without evidences of abnormality of disc on ophthalmologic examination. These patients were managed similarly to the patients with hearing defects which Dr. Semenov described. Many of those who displayed no visible nerve lesion regained normal vision under sodium amytal.

Others with visible lesions of the optic nerves showed improvement in their visual fields after the injection of sodium amytal. It would appear that some had purely functional visual defects while others had functional disabi'ity superimposed upon a structural degeneration of fibers.

Thank you.

CHAIRMAN BROWN: Dr. Semenov, will you please close? I have a question here from Dr. Vietch, of Toronto, "How can you be sure that a case is not clear who, after narcosis, decides further deception is not feasible?"

HERMAN SEMENOV, M.D., Beverly Hills, Calif.: This is a very good question, and as I said when I started there are many things in the written text which I could not mention at this time because of the time limit. One can be reasonably sure of hysteria when a definite emotional abreaction is obtained under pentothal. The malingerers are pretending and ordinarily do not show such reactions.

I want to thank Dr. Truex and Dr. Hoop'e, Dr. Post and Dr. Wolf for their very kind discussions.

## ALUMNI DINNERS

Alumni Dinners will be held on October 13, 1947. All groups wishing to sponsor Alumni Dinners please notify W. L. Benedict, M.D., Executive Secretary, 100 First Avenue Building, Rochester, Minnesota.

Because of the limited facilities of the hotel, it will not be possible to hold all dinners in the Palmer House. As many groups as possible will be assigned to the Palmer House, and the hotel will be glad to assist secretaries of those groups which cannot be accommodated in the Palmer House in making arrangements for dinners at nearby hotels.

Announcement will be made in the TRANSACTIONS of all dinners which have been arranged for if notice is sent to the Executive Office before the July-August issue goes to press.

TRANSACTIONS

American Academy of Ophthalmology and Otolaryngology

*Official Publication of the American Academy of Ophthalmology and Otolaryngology*

Published under the direction of the Editorial Board

WILLIAM L. BENEDICT, Rochester, Minnesota, *Editor*

W. HOWARD MORRISON, Omaha, Nebraska, *Associate Editor*

HENRY L. WILLIAMS, Rochester, Minnesota, *Associate Editor*

Printed by Douglas Printing Company, 108 North 18th Street, Omaha 2, Nebraska

All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, the American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

# Editorial

## Section on Instruction

Attendance at the instruction courses last year was the largest in the history of the Academy. Members were given the opportunity to attend at least twice as many courses as were available in past years.

The reason for this was that half of each day was devoted to either otolaryngology or ophthalmology, and the program was held for four days instead of three. Of course, this growth in attendance entails planning for more instructors and more courses to keep pace with the increased interest in the program.

The secretaries are grateful to those instructors who so willingly and unselfishly have given their time to make this program successful. The membership also has been loyal and helpful in constructive criticism, and we know that with combined help, we shall continue to have an excellent instruction program.

Four full days will be given the program again this year. The section on ophthalmology will be held Monday, Tuesday, Wednesday, and Thursday afternoons. Tuesday, Wednesday, Thursday, and Friday mornings will be devoted to otolaryngology.

Since the majority of members wait until the time of the meeting to secure tickets for the courses, there has been some difficulty in the past in obtaining these at registration time. Also, programs were published so late that members were not given time to place advanced orders. This year the program has been completed for this issue of the TRANSACTIONS, so there will be about six months in which to procure tickets in advance.

Will you please cooperate by sending your orders to the central office as soon as possible. Requests will be filled in the order of the applications received.

It is quite obvious that it will be impossible to fill all orders for specific

basic courses. Attendance in these courses necessarily must be limited. Consequently, only a fraction of the membership may attend these courses each year. We shall attempt to improve this situation by repeating many of the basic subjects each year, so that if they are not available during the current year, application may be made for the next year. In other words, basic courses will be annual presentations, known by their titles. Instructors may change from year to year.

There are three definite advantages in selecting courses in advance:

1. Time will be saved and congestion avoided at the time of registration.
2. The applicant will have sufficient time for reading.
3. In the event that certain basic courses have been filled for the current year, the applicant may apply for the desired courses the following year.

The objective of the instruction courses is to provide men in each specialty with information regarding certain advances in these fields, to review standard clinical subjects, and to provide a review and incentive to future study in certain subjects dealing with the basic sciences. We are sure there is something of interest in these courses for all of our membership.

We urge you to procure tickets as soon as the program is announced. Be on time for the courses. Help the secretaries with constructive criticism regarding the instruction section. Let them know what subjects you would like included in the program. They will welcome all suggestions regarding courses and instructors.

DEAN M. LIERLE, M.D.

## Diversion Club

The Diversion Club of the Academy is planning an all expense three day post Academy vacation at the French Lick Springs Hotel, French Lick, Indiana. The group will leave Chicago on Friday evening, October 17th, and remain in French Lick until Monday afternoon, October 20th. This is an ideal resort for a post convention vacation. Golf, skeet shooting, bridge, horseback riding and hiking are among the many activities which may be enjoyed, also many natural scenic spots within a radius of a few miles may be visited.

The exact cost of the trip has not been worked out, but this detail will be published in the next issue of the TRANSACTIONS.

Reservations can be made for only one hundred. If you wish to take this vacation please write Dr. William L. Benedict, American Academy of Ophthalmology and Otolaryngology, Rochester, Minn.

C. H. MCCASKEY, M.D.

## In Memoriam

Joseph Henry Allen.....Pasadena, California
September 8, 1946

R. B. Boucher.....Vancouver, Canada

George Freiman.....Brooklyn, New York
January 3, 1947

Robert Gewanter.....Long Island City, New York
November 10, 1946

Matthew G. Golden.....Brooklyn, New York
November 11, 1946

Morris Louis Harris.....Brooklyn, New York
November 28, 1946

Thomas Otis Klingner.....Springfield, Missouri
January 7, 1947

Robert K. Lambert.....New York, New York
November 3, 1946

Walter Ivan Lillie.....Philadelphia, Pennsylvania
February 21, 1947

Edward R. Neeper .....Colorado Springs, Colorado
November 21, 1946

Thomas Hall Shastid.....Duluth, Minnesota
February 15, 1947

Samuel Anise Shoemaker.....Orlando, Florida
November 6, 1946

# AMERICAN ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES — 1946 MEETING

The seventh annual meeting of the American Association of Eye, Ear, Nose and Throat Society Secretaries was held on Wednesday evening, October 16th, at the Palmer House, Chicago, during the convention of the American Academy of Ophthalmology and Otolaryngology. The Academy again extended its courtesy to the Secretarial Group by providing an excellent dinner for the members and guests.

The meeting was called to order by the president, Dr. Paul C. Craig, of Reading, Pa. Dr. Craig expressed the Society's appreciation of the dinner given by the Academy and welcomed the guests present. These included Drs. William L. Benedict and Carl H. McCaskey, of the Academy's official family, and 21 E.E.N.T. specialists from South American countries who were in Chicago to attend the Academy session and the first Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology.

Business matters discussed were as follows:

1. Treasurer's Report.

The treasurer's report was read and accepted.

2. Graduate Study Courses.

Dr. Craig discussed the subject of Graduate Study Courses and announced the appointment of a committee to work with Dr. William L. Benedict, Executive Secretary-Treasurer of the American Academy of Ophthalmology and Otolaryngology, on the various phases of this question and to report its recommendations at the next meeting of the Secretaries' Association. The committee is composed of Dr. J. E. Landis, of Reading, Pa., and Dr. W. Howard Morrison of Omaha, Neb.

3. Central Clearing Houses for Information Regarding Programs.

It was announced that the director of each district will collect all available information regarding society programs from the various societies in his district and will release this information to any secretary upon request.

4. Training Course for Secretaries.

Announcement was made of the decision of the Executive Council to provide such a course at the next meeting of the American Academy. All secretaries, whether members of the Secretaries' Association or not, will be welcome to attend this course. The various problems facing the secretaries of E.E.N.T. societies will be considered and solutions proposed. General discussion and interchange of ideas and suggestions will follow. A number of the secretaries present expressed their interest in such a course and their desire to attend. It is to be understood that this project must not interfere in any way with any Academy activity. The course will be held at a time and place which will insure against any such conflict. Further announcement to all secretaries is to be made in the near future as the plan matures.

5. Election of Officers

The nominating committee, composed of Drs. Sam H. Sanders of Memphis, and S. B. Freeman of Buffalo, announced its choice of officers for the coming year, as follows:

President-Elect: Dr. William E. Keith of Kansas City, Mo.

Secretary-Treasurer: Dr. Kenneth L. Craft of Indianapolis, Ind.

There were no nominations from the floor and the above nominees were duly elected.

6. Election of Directors

Dr. Craig, speaking for the Executive Council, then announced the appointment of the following district directors:

District No.

1. . Dr. Burton Lovesey, Boston, Mass.
2. . Dr. S. B. Freeman, Buffalo, N. Y.
3. . Dr. L. A. Brown, Atlanta, Ga.
4. . Dr. Edley H. Jones, Vicksburg, Miss.
5. . Dr. D. S. DeStio, Pittsburgh, Pa.
6. . Dr. William Mann, Chicago, Ill.
7. . Dr. F. D. Hurd, Great Falls, Mont.
8. . Dr. Barton E. Peden, Seattle, Wash.
9. . Dr. Moacyr E. Alvaro, Sao Paulo, Brazil

7. Dr. William L. Benedict, Executive Secretary-Treasurer of the American Academy, was then introduced by Dr. Craig. Dr. Benedict welcomed the guests present and spoke briefly upon Academy activities. Also, he stressed the importance of carefully planned activities and programs to insure the success of local E.E.N.T. Societies and meetings. He mentioned the Kansas City Ophthalmological and Otolaryngological Society as one of the outstanding local societies of the country and one whose policies and activities might well be adopted as a pattern by other similar groups. Dr. Benedict said that the local societies should act as connecting links between the Academy and its membership throughout the country and asked for fuller cooperation along this line from the local societies. In speaking of the future of the American Academy, Dr. Benedict emphasized that although the Academy is rapidly growing to huge and perhaps unwieldy proportions, it would be very unwise to divide the Academy into Eye and Ear, Nose and Throat units or to break it down along any other lines. The life of the Academy, he said, depends upon the solidarity of its various component parts and must have the combined support of all groups to conserve its strength and to insure its continued success. He stressed the importance of the Secretaries' Association in promoting the welfare of the Academy and requested support from the Association in Academy affairs.

8. Dr. Craig then introduced Dr. C. L. Jackson of Philadelphia, Secretary of the Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology. Dr. Jackson spoke upon ways and means of improving Pan-American relations and advocated fuller collaboration between Latin-American and United States E.E.N.T. societies. He then introduced the Latin-American guests present, many of whom elaborated upon his remarks and stressed the advantages of closer relationship among the E.E.N.T. societies of the various Pan-American countries.

Dr. Jackson then showed a series of interesting colored films snapped during his recent visits to South American countries. A number of the Latin-American guests present were seen in the group pictures. The series also included views of the many beautiful scenic spots visited by Dr. Jackson.

Dr. Craig expressed the society's appreciation of Dr. Jackson's share in the program and invited the guests to attend future meetings of the Association.

Dr. Craig then inducted into office Dr. Sam H. Sanders of Memphis, Tenn., the new president of the Association.

The meeting was then adjourned.

KENNETH L. CRAFT, M.D.
*Secretary*

# THE FITTING OF HEARING AIDS

RAYMOND CARHART, PH.D.
EVANSTON, ILL.
*and*
EVA A. THOMPSON, LT. (jg) USNR
PHILADELPHIA, PA.

THERE are varied approaches to the fitting of hearing aids. At one extreme is the view that individualized fitting is unnecessary — that an instrument of proper construction will meet all usual needs. At the other extreme is the philosophy that each hearing loss is a unique entity calling for an especially chosen instrument. Between these limits lie many shades of opinion.

## SYSTEMS OF FITTING

Modern methods for selection of hearing aids include: (1) a thorough study of the individual and his auditory impairment, (2) laboratory tests of his performance with hearing aids, and (3) training in use of the instrument selected. The emphasis is upon an integrated sequence in which every procedure serves a definite clinical purpose. An audiologic approach of this type leads to more effective selection of hearing aids and greater satisfaction in their use. For example, 94 per cent of the patients issued hearing aids at the Philadelphia Naval Hospital are using their instruments with satisfaction.

The remainder of this paper deals with principles in audiology which underlie effective selection of hearing aids.

## CLINICAL ADVANCES

The past three years have offered exceptional opportunity for progress in audiologic theory and practice. A brief evaluation of this progress is pertinent here.

In 1943 and 1944, the Army and Navy established their centers for Aural Rehabilitation.* All centers were in military hospitals. Remembering that the military programs for aural rehabilitation processed about 14,000 patients, one realizes the opportunity which existed to clarify procedures for fitting of hearing aids. The clinical feasibility of different methods could be given ample check. New research findings could be assessed and could be adapted to clinical requirements. As these things were done, one fact emerged with great clarity: namely, *when decisions and recommendations are based on careful analysis of each patient's problem, gratifying efficiency in hearing aid performance and insightful success in hearing aid use are the usual results.*

## RECENT RESEARCH ADVANCES

Wartime research made important contributions to our understanding of hearing aids and the principles involved in their selection.** One contribution

---

The following papers by Dr. Carhart, Lt. Thompson, Dr. Westlake, and Mr. Carter were presented at the meeting of the Committee on the Conservation of Hearing, October 13, 1946, Chicago, Ill. A paper presented by Dr. Kinney entitled "Public School Hearing Program and Demonstration" (The Cleveland Hearing Conservation Program) was published in the November-December, 1945, TRANSACTIONS.

Northwestern University School of Speech.
Philadelphia Naval Hospital.

*The Army centers were at Borden General Hospital (Chickashee, Okla.), Deshon General Hospital (Butler, Pa.), and Hoff General Hospital (Santa Barbara, Calif.). Recently, the three Army programs have been combined and moved to Walter Reed General Hospital (Washington, D. C.). The Navy's program for aural rehabilitation was established and has remained at the U. S. Naval Hospital (Philadelphia, Pa.).

**The most pertinent research was conducted, under the auspices of NDRC, at Harvard University and at Central Institute for the Deaf.

was detailed electro-acoustic analysis of hearing aid performance. Another contribution was the development and standardization of improved speech tests.[4] For example, the spondee word lists (which were made available in several forms) allow rapid determination of the threshold for speech reception. Again, several tests (notably the PB 50 series) allow measurement of the patient's ability to discriminate finer elements of speech.

A third contribution was a series of psycho-physical studies directly concerned with the use of hearing aids.[3] The intensity levels at which loud sounds became intolerable sounds were explored at Central Institute for the Deaf. It was discovered: (1) that persons with hearing loss have thresholds of intolerance which are similar to those of normal listeners, and (2) that tolerance for loud sounds can be increased by appropriate training. Another study, conducted at Harvard University, gave experimental test to the principle of complementing the audiogram. The conclusion which emerged is that complementation with either uniform amplification or with a six decibel rise per octave is more effective than amplification which mirrors the audiogram. While clinical experience indicates that further research on this question is needed, the foregoing conclusion is having an important and helpful influence on thinking regarding the selection of hearing aids.

## METHODOLOGIES OF AUDIOLOGIC ANALYSIS

Careful fitting of hearing aids, in both civilian and military situations, calls for an audiologic analysis composed of seven steps. The progression covered by these steps is:

1. Thorough medical and audiometric study of the patient.
2. Decision regarding the feasibility of a hearing aid and the type of instrument to be specified.
3. Procurement of an individual earpiece whenever air conduction fittings are to be tested.
4. Preliminary selection, through appropriate screening, of a manageable number of fittings which are particularly promising for the patient.
5. Detailed testing to discover the help which the patient receives with each instrument surviving the preliminary selection.
6. Recommendation, on the basis of test results and the patient's special problems, of the instrument best suited to him.
7. Auditory training designed to adjust the patient to the instrument chosen.

With the foregoing summary in mind, each step may be considered more fully.

A. *Otologic and Audiometric Study.* Hearing aids offer electro-acoustic prosthesis. Consequently, a hearing aid can never rightly be recommended until after general physical examination and careful otologic study have ruled out the feasibility of both medical and surgical treatment.

Furthermore, other criteria must be satisfied before fitting procedures should be undertaken. For one thing, the person must have a degree of loss which lies within the range where hearing aids give promise of useful compensation. Except in special circumstances, the loss over the vowel range (512-2048 c.p.s.) should average 30 decibels or more. Conversely, patients with extreme impairment do not receive enough amplification to obtain full satisfaction from an instrument. Generally speaking, patients whose 512-2048 c.p.s. averages for the better ear lie between 45 and 75 decibels are most effectively helped.

A second criterion is that the pattern and type of hearing loss must favor effective use of hearing aids. On the whole, cases of conductive loss obtain more gratifying results than cases with a predominantly perceptive component. Similarly, cases with uniform impairment tend to get more help than cases showing irregular audiometric curves—although the latter often do astonishingly well.

A third criterion is that language comprehension and the ability to discriminate the phonetic elements in speech must be adequate for ordinary social situations. It sometimes happens that the hearing problem includes more than mere decrease in auditory acuity. One example is the child with congenital deficiency in audition, who has failed to learn how to interpret spoken language. Again, we have cases whose deficiencies are predominantly in the central nervous system. Finally, there may be concomitant involvement of both the central mechanism and the ear. Under any of these three circumstances, measurement of hearing loss with the pure tone audiometer does not fully indicate the severity of the practical handicap. Many cases falling into these categories find hearing aids relatively useless.

Obviously, full exploration of auditory capacities is necessary to judge the practicability of recommending a hearing aid. As a minimum, *this exploration must include both pure tone tests and standardized speech tests.* One of the outstanding facts confirmed by experience in military programs for aural rehabilitation is that both types of tests are essential to sound procedures for hearing aid fitting. Neither one alone gives information even distantly comparable to that yielded by a combination of both.

Regarding pure tone audiometry, we need merely state that a careful survey by both air conduction and bone conduction are necessary. The usual precautions in technic and in maintenance of equipment must be rigidly observed.

Regarding speech audiometry, more must be said. Modern procedures for speech tests have their experimental basis in the work of the Bell Telephone Laboratories[5] and of the Psycho-Acoustic Laboratory at Harvard.[4] Modern procedures have their clinical basis in the speech reception technic introduced in 1942 by Hughson and Thompson.[6] Stated briefly, these tests present standardized samples of speech at controlled intensities. Test materials are administered by live-voice or from phonograph records. The patient either repeats or writes down what he hears. Depending upon the test materials and the technic, one may obtain from the patient's responses a measure either of acuity for speech or of capacity to discriminate phonetic elements.

In the opinion of the writers, monitored live-voice is the most practical method for presenting speech tests in the typical clinical situation. The live-voice method requires special equipment—as, in matter of fact, do all controlled speech tests. The basic units in the equipment for measurement by monitored live-voice are shown in figure 1. The tester, who must be trained to talk at a constant level, speaks into the microphone. The resultant electrical impulses are made more intense by the pre-amplifier. To insure that his voice is at the proper intensity, the tester observes the monitoring meter which measures the impulses as they leave the pre-amplifier. If necessary, the tester modifies the strength of his voice so as to produce a signal of proper level. The electrical impulses then pass through an attenuator with a range of at least 110 decibels. By means of this attenuator, the impulses can be reduced by any specific number of decibels. The electrical impulses are then transmitted through

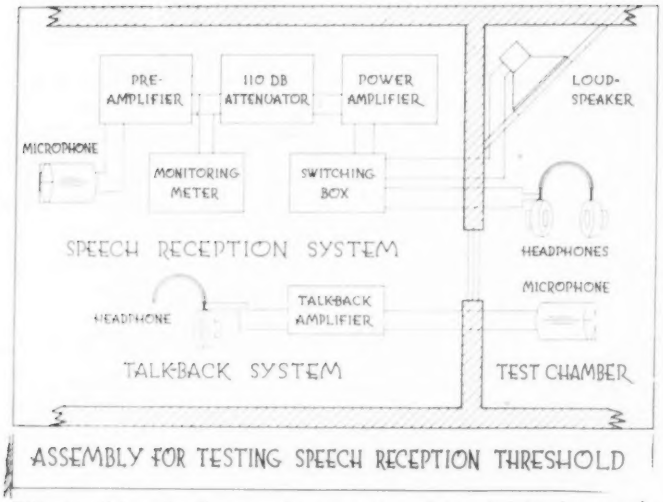



FIG. 1.—Schematic diagram of equipment used in administering speech tests by monitored live-voice. (From *Archives of Otolaryngology*).

a power amplifier to a loud speaker or a set of headphones.

The equipment allows one to present speech tests at any desired intensity above either normal threshold or above the patient's own threshold. Thus, the method is easily adaptable either to measuring hearing loss, or, as is pointed out later, to comparing the performance of different hearing aids.

Naturally, careful speech tests demand sound-proofed and acoustically treated chambers. The talker is in a booth containing the microphone, the meter, and the attenuator controls. The patient is in a second room and places his head (or the hearing aid) in a standard position, provided the test is administered through a loudspeaker. A talk-back system allows the patient to communicate with the tester.

The measurement of acuity for speech ". . . gives information in regard to the patient's ability to use his residual hearing. . . ."[7] Furthermore, in evaluating the patient's need for a hearing aid, the results of the speech test and of pure tone audiometry can be profitably compared. The binaural loss for speech ordinarily equals the average for the 512-2048 c.p.s. band when this average is a composite which is based upon the better threshold at each test frequency. When sharp deviations from the foregoing rule appear, they ". . . help one assess (1) the reliability of the test results, (2) the credibility of the patient's loss, (3) the variability of his loss, (4) the referrals which may be required, and (5) the rehabilitation procedures which may be indicated."[1]

B. *Preliminary Decisions.* When the medical and audiologic examinations reveal that the patient should procure a hearing aid, two supplementary decisions are necessary.

The ear on which the instrument is to be worn must be chosen. The details of the individual case affect the decision, but a few general rules may be stated. When moderate loss exists in both ears and the patient can still use the telephone satisfactorily, the hearing aid is fitted to the ear *not* used in telephoning. When the disparity between the two ears is 20 decibels or more over the 512-2048 c.p.s. range, the ear with the lesser loss is chosen. The most com-

mon exception to this rule occurs when the average ear loss in the poorer ear does not exceed 60 decibels. Finally, when the average loss is approximately equal in the two ears, the ear with the more uniform loss or with the more regular audiometric contour is to be preferred.

Secondly, choice must be made between the air conduction and the bone conduction type of fitting. Experience in military programs demonstrates that air conduction is the fitting of choice for most patients. The primary exception occurs when there is medical contra-indication to the use of an earpiece in the external auditory canal. Here bone conduction instruments are the only possible type. Exceptions are also justified for cases with pure conductive impairment. Since these cases are helped by both types of fitting, decision can be made on secondary factors such as comfort, visibility, and aesthetic considerations.

C. *The Individual Earpiece.* If the patient is to use an air conduction fitting, he must have his own earpiece before the final procedures of selection are undertaken. This earpiece is individually constructed to conform to the contours of his ear.

Modern practice favors the use of acrylic or colloidal materials (rather than plaster-of-paris) for obtaining the impression from which the individual earpiece is manufactured. When the earpiece is completed, it must be carefully examined to insure its adequacy. The earpiece must be free from rough spots. It must fit the ear comfortably and snugly. It must attach to the hearing aid receiver firmly and without leakage. The physician can render his patients an important service if he insists upon an individual earpiece, if he makes the impression himself, and if he checks the finished earpiece for adequacy. These precautions are important in the interests of the patient's safety and because the condition of the earpiece modifies the behavior of an air conduction instrument.

D. *Pre-Selection of Instruments.* Even disregarding carbon instruments, hundreds of hearing aid combinations are available to the prospective user. Most manufacturers have several models of the vacuum-tube type, as well as different receivers and tone adjustments. It is therefore necessary to choose a few combinations which the patient is to subject to detailed tests. The major factors governing the choice may be summarized under the following headings.

1. As a protection to all concerned, only instruments approved by the Council on Physical Medicine should ordinarily be considered.

2. As a further protection, choice should be limited to instruments having local representation which is reputable and efficient. Maintenance and service problems can thus be kept at a minimum.

3. A person with knowledge of hearing aids should then select from the many possibilities which will remain, those instruments whose characteristics best conform to the needs of the patient as revealed by the tests mentioned earlier.

E. *The Hearing Aid Evaluation.* All of the military programs in aural rehabilitation recognized the need for careful tests to evaluate the patient's performance with a final group of instruments. Details of procedure differed from one rehabilitation center to another. Time does not allow discussion of these differences. Suffice it to say that clinical experience has proved the desirability of exploring several facets of efficiency in hearing aid use. The more important of these facets are:

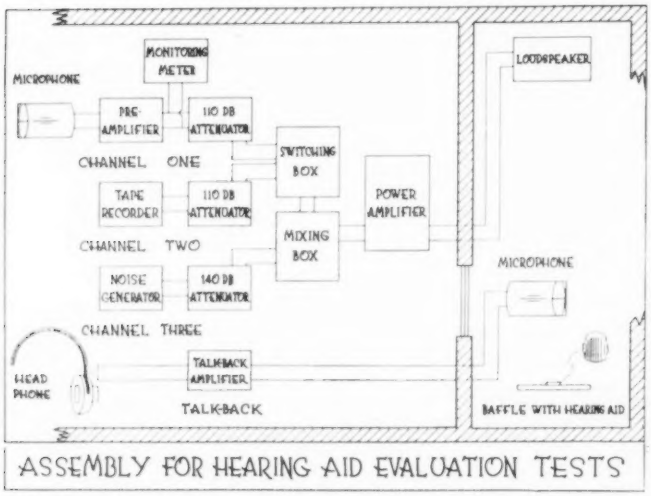



FIG. 2.—Schematic diagram of an assembly for administering tests to evaluate hearing aids. (From *Archives of Otolaryngology*).

sensitivity (gain), tolerance limit, hearing in noise, and precision in aural discrimination.[2]

1. Sensitivity, or gain, is measured by finding the patient's residual loss for speech while he is wearing the hearing aid. The procedures and equipment are basically similar to those used in determining the unaided threshold for speech. (Fig. 2.) Ordinarily, a well fitted hearing aid gives a gain of at least 30 decibels. However, patients with mild losses as well as atypical cases such as those with psychogenic involvement—frequently fail to show this much gain. The test of sensitivity both indicates the degree of help which the patient receives from a hearing aid, and—when the scores differ appreciably from one fitting to another—gives a basis for choosing between instruments.

2. The patient should be able to tolerate loud sounds when they are reproduced by the instrument chosen for him. We need merely determine which, if any, of the instruments under test have upper limits of power which are excessive for the patient. The technic is simple and employs the equipment already mentioned. Progressively increased levels of sound are brought to the hearing aid. The patient reports the first appearance of a definite pain, tickle, excess vibration, or dizziness. Threshold of intolerability is made a basis for a choice whenever differences between instruments appear. Furthermore, the patient who has difficulty tolerating standard instruments requires both training to raise his tolerance limit, and a hearing aid with a decreased output of power.

3. The level at which background noise interferes with his understanding of speech is an important consideration to the user of a hearing aid. This level is easily measured by presenting samples of speech at a fixed intensity while progressively raising the strength of a controlled background noise. When several instruments are tested in this way the results give a basis for choosing between instruments and also indicate the degree to which listening in noise will be difficult for the wearer.

4. Recognition of fine differences in sound is essential to socially effective hearing. Particularly important is the

ability to discriminate the phonetic elements which comprise human speech. The ability to do so can be measured with appropriate word lists or with standardized sentences. These materials are presented, through the equipment already described, at sufficient intensity so that all phonetic elements will be intelligible if the patient's discrimination is satisfactory. Such a test measures the degree to which the patient's perception falls short of perfection. Not only are comparisons between hearing aids possible, but the test also helps estimate the patient's need for training designed to improve his discrimination of speech elements.

F. *Recommendation of Hearing Aid.* The particular hearing aid to be recommended for the patient is chosen after weighing all the information gathered in the preceding steps. This information is evaluated in terms of the usefulness which the wearer may expect from different hearing aids in everyday situations. The instrument must yield adequate measures of sensitivity, of tolerance limit, of performance in noise, and of discrimination. Where a special problem appears in the use of a hearing aid, its nature and severity not only determine whether an instrument is feasible, but also indicate the special training required by the patient. Furthermore, when several fittings yield equivalent performance, the recommendation must consider the practical features of initial cost, maintenance expense, service facilities, wearability, weight, and aesthetic preference.

G. *Auditory Training.* Experience has shown that the patient will ordinarily require instruction in how to use his hearing aid before he can achieve maximum efficiency with it. Such instruction is known as auditory training. Almost every patient needs an orientation course which will foster psychologic acceptance of his instrument and will teach him how to manipulate it effectively. Many patients also need more extended courses in auditory training. For example: some children must build their habits of audition for the first time. Such children must be guided step-by-step from success in simple experiences of listening to success in complex ones. By contrast, many adults require re-education because their auditory habits have been disturbed by the loss in hearing. Such a person must learn to take full advantage of the auditory clues which remain at his disposal. Individualized programs in auditory training foster more successful use of residual hearing and, hence, must be the culminating step in careful programs for the selection of hearing aids.

## SUMMARY AND CONCLUSION

With proper medical and audiologic guidance, most patients who suffer permanent losses in hearing can counteract the handicap imposed by their impairments. Modern methods for the fitting of hearing aids are a phase of the total process. These methods emphasize: (1) detailed study of the patient as foundation for decision regarding his needs, (2) procurement—when necessary—of an individual earpiece, (3) preliminary screening to eliminate instruments which are undesirable for the patient, (4) careful tests to evaluate promising instruments, (5) selection of the final instrument, and (6) auditory training to teach the patient effective use of his hearing aid.

The present duty is to encourage growth of hearing clinics and other programs which will make available to civilians modern procedures in aural rehabilitation — including the fitting of hearing aids. Military experience has proved that such programs must be cooperative ventures. The otolaryngologist furnishes the diagnostic skill, the

medical guidance, and the supervisory control necessary to fully protect the patient. The clinical audiologist supplies the non-medical knowledge, the testing skill, and the specialized instruction required to meet the patient's problems. Stimulating experiences lie ahead for the workers who will build our civilian programs for aural rehabilitation.

## REFERENCES


1. Carhart, R.: Individual differences in hearing for speech, Ann. Otol., Rhin. & Laryng., 55:233-266, (June) 1946.
2. ————: Tests for selection of hearing aids, Laryngoscope, 56:780-794 (Dec.) 1946.
3. Davis, H. et al.: The selection of hearing aids, Laryngoscope, 56:85-115 (March) 135-163 (April) 1946.
4. Egan, J.: Articulation testing methods II, OSRD Report No. 3802 (Nov.) 1944. (Procurable through Office of Technical Services, Dept. of Commerce).
5. Fletcher, H. and Steinberg, J.: Articulation testing methods, Bell System Technical Journal, 8:806-854, 1929.
6. Hughson, W. and Thompson, E.: Correlation of hearing acuity for speech with discrete frequency audiograms, Arch. Otolaryng., 36:526-540 (Oct.) 1942.
7. Thompson, E. A.: The fitting of hearing aids, J. M. Soc. New Jersey, 43:317-320 (Aug.) 1946.

# METHODS FOR RECORDING AUDIOMETRIC FINDINGS RECOMMENDED BY THE COMMITTEE ON THE CONSERVATION OF HEARING

EVA A. THOMPSON, LT. (jg) USNR
PHILADELPHIA, PA.

THE charting of the results of hearing tests is a very important phase of audiometry. Incorrect charting may result in faulty diagnosis and statistical inaccuracies. Up to the present time each otologist or audiologist has chosen the form of chart and has used the charting method he preferred. This of course accounts for the differences in the format of charts and the symbols used, and for the variations in published audiometric data. The consensus of opinion of individuals interested in the testing of hearing is that greater uniformity in charting methods would clarify the interpretation of audiometric records.

The standardization of audiometers has been accomplished by the efforts of the Committee of Consultants on Audiometers and Hearing Aids of the American Medical Association's Council on Physical Medicine. They have set up "Minimum Requirements for Acceptable Audiometers" and all approved instruments are calibrated according to the National Bureau of Standards' specifications. It is necessary that audiometers be maintained in calibration and in proper working condition. Uniformity of performance may therefore be expected from audiometers which carry the approval of the American Medical Association.

The "Manual for Program Outline for Rehabilitation of Aural Casualties Both Military and Civilian" prepared by Hughson and Westlake and sponsored by the Committee on the Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology describes briefly the most widely accepted technic of testing hearing by means of an audiometer.

This committee, recognizing the divergence of opinion in regard to methods of charting audiometric test data, solicited opinions from a large number of audiologists and the following recommendations and suggestions for recording test findings are based on the results of this questionnaire. The Committee urges that whenever possible these recommendations be adopted. This will insure greater uniformity in the records of various clinics and in published articles.

Audiometry may rightly include tests other than those done on the audiometer. Modern usage, however, has limited this term to hearing tests carried out by means of electro-acoustic equipment. Speech reception tests as conducted in the Service Aural Rehabilitation Centers and in some of the modern hearing clinics certainly should be classified under Audiometry and the results of these tests may be indicated on the audiogram chart either by sketching a horizontal hatched bar between the speech frequencies 512-2048 at the decibel level for speech reception or by writing this decibel figure in the space allowed for comments. (Example: S. R. ———db.)

This speech reception figure must not be confused with the percentage loss figure obtained by averaging the decibel loss for the individual audiometric frequencies, 512, 1024 and 2048 c.p.s. This

average is purely a statistical figure, while the speech reception level in decibels is obtained from an entirely separate test. This test consists of the presentation of speech through a speech reception evaluator whereby all test conditions, including the intensity of the speech used, are accurately controlled and measured. The audiogram depicts residual hearing; the speech reception figure represents the individual's ability to use his residual hearing.

The audiometric chart* prepared by Edmund P. Fowler, M.D., and P. E. Sabine, Ph.D., for the Committee of Consultants on Audiometers and Hearing Aids of the American Medical Association conforms to all the recommendations contained in this report and well illustrates how audiometric charts may be varied, provided the fundamental points are in agreement. It should be noted that the method of computing hearing loss from this chart gives a figure which represents the *capacity* to hear speech. It may or may not be in agreement with the speech reception figure as obtained by means of a speech reception evaluator. This chart has been designed for use in medicolegal work and is useful in situations where speech reception apparatus is not available.

Audiometric charts should have the following uniform points:

1. The chart should be clear, simple and not cluttered with the results of other types of hearing tests or history.
2. All test frequencies of accepted commercial audiometers should be on the chart. These are 128, 256, 512, 1024, 2048, 4096, and 8192 c.p.s.† These should be charted on the horizontal axis. Half octaves, 1448, 2896, 5792, 9747, and 11,584 c.p.s. may be included.
3. The generally accepted intensity scale in 10 decibel steps should be on the vertical axis. This intensity scale should cover the total range of accepted audiometers.
4. The distance between adjacent octave intervals shall be twice as great as that between adjacent 10 decibel step intervals, for example, the space between 1024 and 2048 c.p.s. is twice the distance between 10 and 20 decibels.
5. The chart should allow space to indicate the make and model of audiometer used.
6. The symbols to be used for the right and left ear shall be printed on the form. For air conduction tests a circle (O) should be used for the right ear and a cross (X) for the left ear.
7. All frequencies tested should be indicated on the chart. If no response is obtained at maximum intensity the appropriate symbol should be used with an arrow pointing downward.
8. When masking for air conduction is employed, as is necessary in the unilateral case, a different symbol must be used. The triangle (△) for the right ear and the square (□) for the left ear is recommended. Both the symbol used and the intensity of the masking noise must be clearly indicated on the chart.
9. If bone conduction is charted on the audiogram the following points should be considered: (See figures 1-A and 1-B.)
   a. The same normal, zero on the chart, should be used for both air conduction and bone conduction.
   b. The symbols used must be indicated on the chart. It is recom-

*Information about this chart may be obtained by writing the Council on Physical Medicine of the American Medical Association.

†Round figures 120, 250, 500, 1000, 2000, 4000 and 8000 may be substituted if this change is made on accepted commercial audiometers.

mended that the half triangle (>) to the right of the vertical line represent the right ear and the half triangle (<) to the left of the vertical line represent the left ear.

c. Masking should be used when testing bone conduction, and the intensity of the masking noise indicated.

10. For repeated audiometric tests the numerical form of recording is recommended for the following reasons: (See figures 2-A and 2-B.)
    a. Ease of comparison of audiograms.
    b. Conservation of recording space.
11. For publication of clinical audiometric records the following forms are recommended:
    a. The audiometric chart for a single test or for one pre- and one postoperative test.
    b. The numerical (Fig. 2) or the composite (Fig. 3) audiogram form for a series of tests.
12. For publication of experimental research work the committee agrees that methods of recording data should be left to the discretion of the author.

AUDIOGRAM OF _____

DATE _____
AUDIOMETER _____
TEST BY _____

AIR CONDUCTION
O - RIGHT EAR
X - LEFT EAR
WITH MASKING
△ - RIGHT EAR AT ___ DB
□ - LEFT EAR AT ___ DB

BONE CONDUCTION
⟩ - RIGHT EAR
WITH MASKING AT ___ DB
⟨ - LEFT EAR
WITH MASKING AT ___ DB

COMMENTS

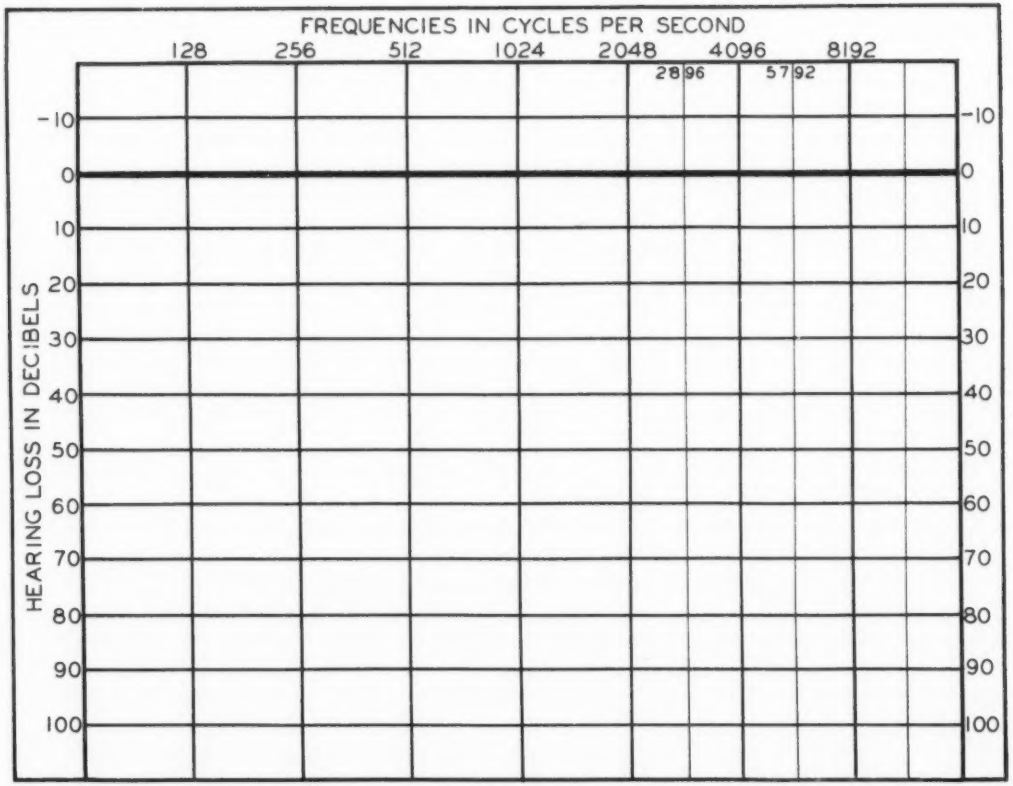



FIG. 1-A—Audiometric chart for the recording of one audiogram. Space has been left at the top of the form for the insertion of the name of the organization or clinic using the chart if so desired. This chart may be reproduced from the TRANSACTIONS by any local printer.

AUDIOGRAM OF_____

DATE_____
AUDIOMETER_____
TEST BY_____

AIR CONDUCTION
O - RIGHT EAR
X - LEFT EAR
WITH MASKING
△ - RIGHT EAR AT___DB
□ - LEFT EAR AT___DB

BONE CONDUCTION
|> - RIGHT EAR
WITH MASKING AT___DB
<| - LEFT EAR
WITH MASKING AT___DB

COMMENTS

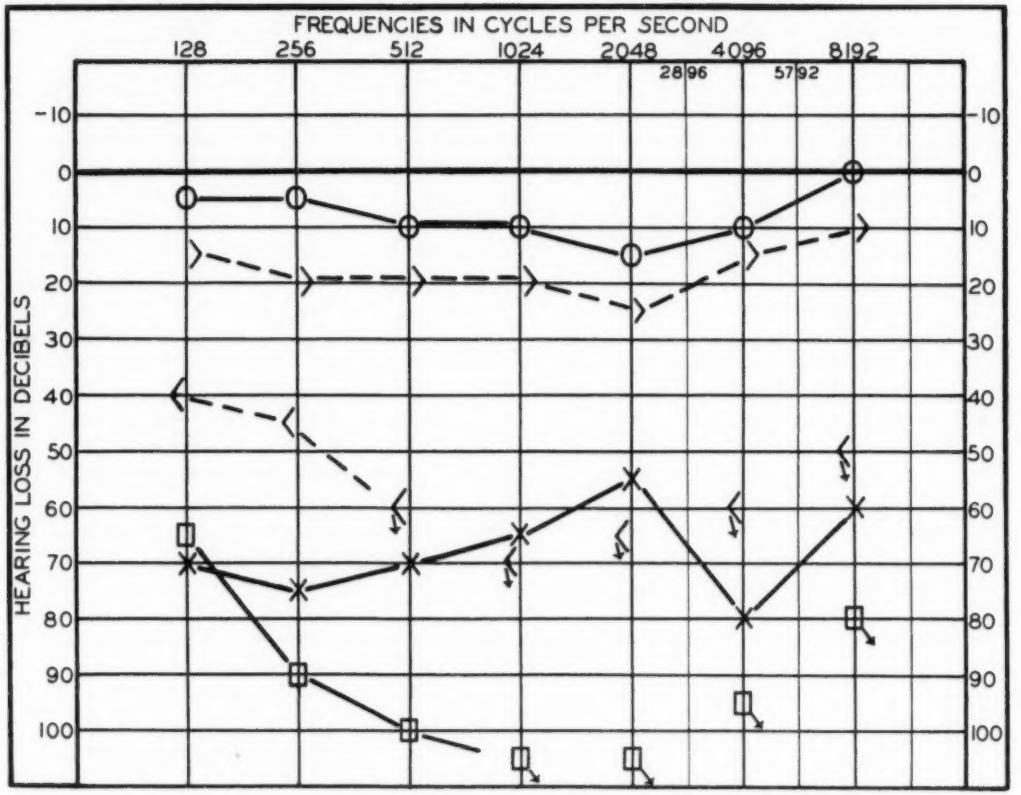



FIG. 1-B—Audiometric chart illustrating the approved symbols to be used and the manner in which the audiogram should be charted. The spaces for insertion of patient's name, etc. have not been filled in in this sample chart.

## AUDIOMETRIC RECORD

NAME _____ _____ AUDIOMETER

| DATE | TEST BY | RIGHT EAR AIR CONDUCTION | | | | | | | | | | | M* | LEFT EAR AIR CONDUCTION | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 128 | 256 | 512 | 1024 | 2048 | 2896 | 4096 | 5792 | 8192 | 9747 | 11584 | | 128 | 256 | 512 | 1024 | 2048 | 2896 | 4096 | 5792 | 8192 | 9747 | 11584 |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | RIGHT EAR BONE CONDUCTION | | | | | | | | | | | | LEFT EAR BONE CONDUCTION | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |

* INDICATE UNDER M THE EAR AND INTENSITY OF MASKING USED

FIG. 2-A—Numerical audiometric record form for the recording of repeated audiograms. This method is time saving and more ready comparison of audiograms can be made than by the use of the single audiometric chart. This chart may be reproduced from the TRANSACTIONS by any local printer.

## AUDIOMETRIC RECORD

NAME H.F.C. W.E. 6B AUDIOMETER

| DATE 1946 | TEST BY | RIGHT EAR AIR CONDUCTION | | | | | | | | | | | | LEFT EAR AIR CONDUCTION | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 128 | 256 | 512 | 1024 | 2048 | 2896 | 4096 | 5792 | 8192 | 11584 9747 | MM | | 128 | 256 | 512 | 1024 | 2048 | 2896 | 4096 | 5792 | 8192 | 11584 9747 |
| 14 SEP. | T | 40 | 55 | 50 | 55 | 50 | 50 | 50 | 55 | 60 | 60 | | | 40 | 50 | 45 | 45 | 50 | 45 | 45 | 50 | 50 | 55 | |
| 18 SEP. | K | 30 | 45 | 50 | 55 | 50 | | 45 | | 60 | | | | 30 | 40 | 45 | 45 | 45 | | 40 | | 45 | | |
| 30 SEP. | T | 30 | 50 | 50 | 50 | 50 | | 50 | | 65 | | | | 35 | 40 | 40 | 45 | 45 | | 40 | | 40 | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | RIGHT EAR BONE CONDUCTION | | | | | | | | | | | | LEFT EAR BONE CONDUCTION | | | | | | | | | | |
| 30 SEP. | K | 15 | 5 | 10 | 15 | 15 | | 10 | | 5 | | 40 | | 15 | 10 | 5 | 15 | 20 | | 20 | | 10 | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | |

* INDICATE UNDER M THE EAR AND INTENSITY OF MASKING USED

FIG. 2-B—Audiometric record illustrating the method of recording audiograms.

NAME _____ DATE _____ No. _____

EAR _____

AIR CONDUCTION

BONE CONDUCTION

## AUDIOGRAMS

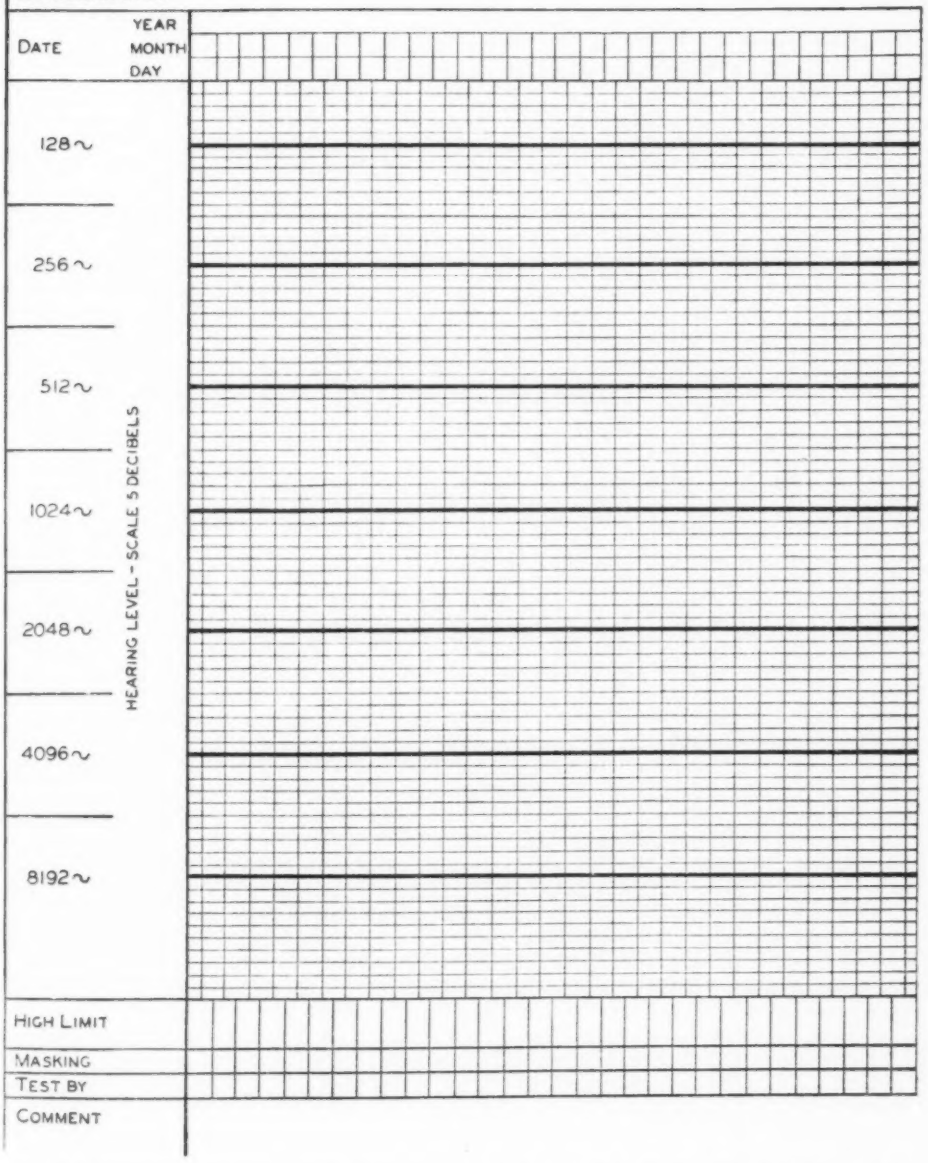



FIG. 3-A—Composite audiogram form recommended for the charting of repeated audiograms. This form is excellent for the publication of case histories where a number of audiograms on the same patient are to be presented. This composite audiogram form may be obtained from Kaufman Press, 30 Commerce Street, New Haven, Conn.

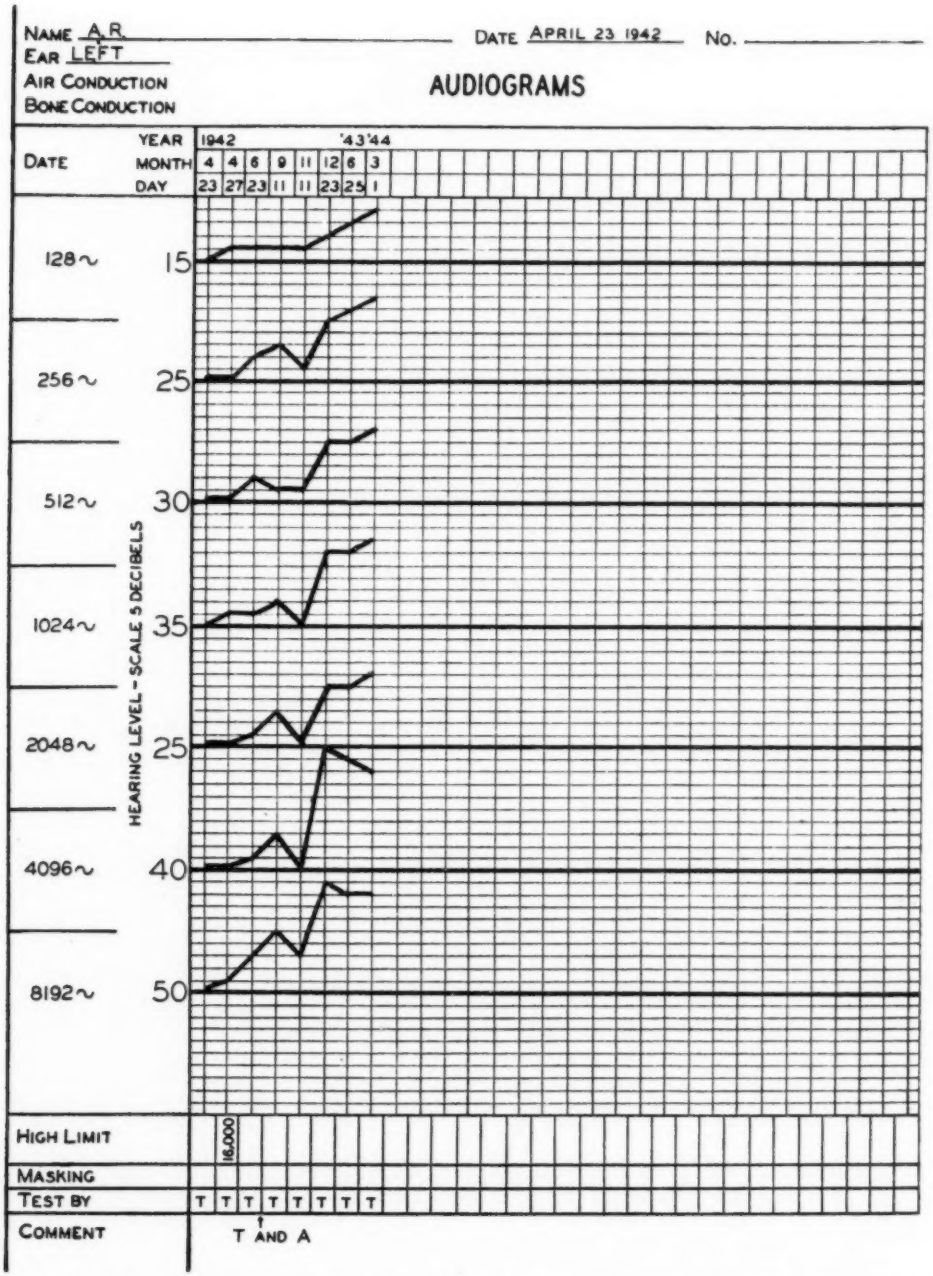



FIG. 3-B—Composite audiogram illustrating the method of recording audiograms. The decibel loss for each frequency is written beside the base line for that frequency and the first test is charted on the base line, the date of the test being written at the top of the first vertical column. Each frequency is charted on a separate base line and subsequent tests are charted in the same manner, the reference point being the level of the original audiogram.

# LIPREADING DEMONSTRATION

HOWARD CARTER, B.S.
CHICAGO, ILL.
*and*
HAROLD WESTLAKE, PH.D.
EVANSTON, ILL.

THE purpose of this demonstration is twofold; first, to show the usefulness of lipreading skill; and second, to summarize other information which may be useful to those who advise persons in need of lipreading instruction.

The adjustment of the person having reduced hearing will center about improving his skill in communication. This communication problem involves both the receptive and the expressive phases of oral language, since understanding what is said and replying in intelligible speech are the basic requirements in a communicative situation. Ordinarily speech deteriorates as a result of reduced acuity. The amount of change in the speech pattern depends upon the degree of loss, the type of loss, the age at onset of loss, the duration of loss, and finally an individual factor which has not been completely analyzed: certain individuals have retained practically normal speech over periods of many years in spite of profound hearing losses, while others have shown marked deterioration of the speech pattern within a few months of the incidence of the loss. Since the expectancy is that the speech will be affected as a result of the hearing problem, speech training should be an essential part of an adjustment program. Consequently, it is wise to introduce speech work as soon as a significant loss of hearing for speech is noted.

Secretary, Council on Physical Medicine, American Medical Association.
School of Speech, Northwestern University.

On the receptive side, the ability to read lips is a most important skill for a person with reduced hearing. The recommendations of the Committee on Conservation of Hearing of the Academy is that lip reading instruction be provided for "those whose better ears have an impairment of 25 decibels or more at 512, 1024, and 2048 cycles, which does not improve under medical care within six months." This statement is taken from the supplement to the TRANSACTIONS, edited by Dr. Horace Newhart and Dr. Scott Reger, published in April, 1945.

Without detracting from the importance of lipreading, it is essential that its usefulness not be over-stated, with consequent discouragement to the patient. Lipreading is far from being as satisfactory as hearing. Through hearing and print one has fairly complete representations of language, in that the sounds or letters are all represented, and are distinguishable. On the other hand, many of the speech movements cannot be seen. Many of the movements which are visible, as those for the *p*, *b*, and *m*, are identical to the observer, although the different sounds are readily identified by the listener. Consequently, the lipreader is relying upon many fewer clues than are available to the normal hearer or the reader, and he cannot be expected to get all of the exact information from conversation that the hearing person gets. While it is important to face honestly the limitations of lipreading, the demonstration which follows

will show its usefulness to be great enough to warrant the strong recommendation that this skill be learned by all persons whose hearing deficiencies make hearing of speech difficult.

Recognition of the fact that lipreading does not offer perfect reception, but that it is an important help leads naturally into the combined use of lipreading with other avenues or partial avenues the patient might have. The adjustment problem does not involve a choice between a hearing aid and lipreading, if a hearing aid can be used, but the use of both lipreading and the hearing aid — one re-enforcing the other to accomplish better communication. Dr. Walter Hughson was always insistent that every wearer of a hearing aid should be a lipreader, his reasoning being that the instrument rarely, if ever, gives a perfect correction, and that there are times and situations in which the instrument cannot give optimum usefulness. Likewise, you read in the 1946 supplement to the *Naval Medical Bulletin* that "Every patient (referring to those in aural rehabilitation) is required to take a concentrated speech reading course that is adapted to his needs." Experience and research in the education of the hard of hearing have established that the combined use of lipreading and amplification results in increased accuracy in understanding and language, and that the use of lipreading does not discourage the use of hearing nor does the use of a hearing aid discourage the use of lipreading as many individuals have thought.

In the demonstration to follow, Dr. Francis L. Lederer will interview two lipreaders. Dr. Lederer is not well known to the lipreaders, introductions having been made only this afternoon. Neither subject knows the material for his interview. The subjects cannot hear speech and consequently are relying entirely upon lipreading ability.

One will demonstrate the adequacy of lipreading in an ordinary social conversation. The other will demonstrate how lipreading can function in a business situation. (Demonstration.)

We hope that this demonstration has shown you that lipreading is a useful skill. In summary, these points have been pertinent:

1. Lipreading skill should be developed by all persons whose average hearing in the better ear in the speech range (512 through 4096 cycles) is 25 decibels below normal.

2. Lipreading and the hearing aid reenforce rather than interfere with each other, both contributing to more successful reception of oral language.

3. Successful communication is a two-way proposition, in which understandable speech is important. Speech training should be initiated early to prevent the deterioration of speech.

4. Local teachers of speech and lipreading may be contacted through the local Societies for the Hard of Hearing, or through their national organization, the National Hearing Society. Other sources are the National Association for Speech Correction and State Departments of Public Instruction.

# OCCUPATIONAL OPHTHALMOLOGY

## INTRODUCTORY STATEMENT

*It has long been the intention of the Joint Committee to present to the professions thru the vehicle of this section of the TRANSACTIONS, the range and types of questions that are channeled to the Committee. In the hope that such a report very simply gotten up into a "Question and Answer" recital will stimulate ophthalmologists throughout the country to send in reports of their experiences, we are confining the contributions of this issue of the TRANSACTIONS to such a question and answer series.*

*The industries of this country, of course, vary according to its geography. In the Northwest, the lumber and mining industries, the fruit raising and ship building and many small plants of all descriptions have experiences entirely different from any in other parts of the country. In the South, the textile industries, ship-building, fabricating, fishing, citrus-fruit are again different with experiences of value to all of us. In the Midwest are the heavy industries of the Lake region — steel, automobiles, chemicals, oil, boxcars, candy, cement, and many finished articles providing a still different series of experiences and problems. The type of eye injuries existing, the problems the professional eye consultants meet with in relation to management attitudes, unions, and the varying opportunities for educational contacts are different in each region. They are of importance to all of us.*

*The Joint Committee earnestly solicits your correspondence and your contribution. The latter need not be worked up into a polished paper, but gotten up as "plant experiences," or individual experiences parallel to "Case Histories" of the ordinary medical journal.*

*The following questions have been taken from the personal files of Committee members and the files of over the last few years. They speak for themselves.*

HEDWIG S. KUHN, M.D.
*Secretary, Joint Committee*
*Occupational Ophthalmology*

---

## QUESTIONS AND ANSWERS

*Question 1*

"We are confronted with the development of cataracts in the eyes of a man who was burning carbon from the cylinders of a Buick automobile. As you probably know, this operation is performed by removing the spark plugs from the cylinders, injecting a small amount of oil to saturate the carbon and then introducing pure oxygen which burns with explosive force, repeated explosions combining the oxygen and carbon to clean the cylinder. Each explosion results in a cone of bright light, fire, and burning carbon particles in a verticle direction thru the spark plug opening. It is claimed that constant exposure to these repeated flashes has caused eye cataracts and we are asking if you can give us any information as to development of similar cases within your knowledge and also if you could give us any statement as to the probability of such an effect from these operations. Analysis of this work by an industrial testing laboratory indicates negligible quantities of ultraviolet rays and negligible quantities of infrared rays. The exposure was spasmodic over a period of some months and with this information to go on we would like to have your reaction to the possibility of such an injury."

*Answer*: "True radiation cataract has been reported in individuals working with high intensities of heat (infra-red), over many years' time—as high as 25. The glass blower's cataract of the old days on the continent is an example. Ultraviolet radiation does not penetrate the eye beyond the cornea and does not produce lens changes. Radiation cataract is said to have been seen in this country. It presents a typical slit lamp picture with exfoliation of the anterior capsule of the lens, and posterior polar and capsular lens opacities. Long search of the records has revealed no record of a cataract due to burning carbons from the cylinders of cars."

*Comment*: The problem of intensity of infra-red and ultraviolet as recorded *on the job rather* than only in the laboratory, is one that is high in the agenda of ophthalmologists working with industrial eye problems—the biological and scientific work done in the laboratories does not seem to adequately impress the Industrial Commissioners. They are responsible for decisions on awarding compensation to individuals exposed to ultraviolet and infra-red. It is the opinion of most ophthalmologists who appear before Commissioners in these cases that work done under the actual conditions in which men find themselves in industry is needed to fortify their testimony. (HSK)

*Question II*

"We would like to have information relative to what states have passed laws prohibiting, for sanitary reasons, the use of community eye goggles by men who work on grinding machines, etc."

*Answer*: "As far as we can find out, there are no state laws involved in such prohibitions."

*Question III*

"An individual had 60,000 volts of milliamperage of about 5-7 pass thru him from his forehead to his right hand, coming out at the thenar eminence. The current entered his forehead with a jump. What might one expect with regards to cataract formation?"

*Answer*: "While both lens and macular changes might develop in time as a result of this electrical contact, it is held to be very unlikely as the usual damage is at the point of contact."

*Question IV*

"Which of the several depth perception tests, gives one an accurate appraisal of stereopsis whereby the standard for crane operators can be set up? The union being interested in the evaluation, we would like to be sure of our ground."

*Answer*: "A new employee who is being considered as a crane operator, or a payroll employee whose transferral to a crane operator is being considered, should have at least 80 per cent depth perception as tested on any of the standard stereopsis recording instruments. In addition to this, normal acuity for distance and normal muscle balance are of great importance and equally necessary. An individual may have normal depth perception and be unstable with marked exophoria which breaks down after fatigue sets in. The crane operator who has had an accident-free record of many years, even though he has no depth perception at all, having substituted other values for it, may be safer than a green operator."

*Question V*

"One of my patients is suffering from keratitis, cyclitis with secondary glaucoma, and rapidly deteriorating vision. There are dense corneal scars and vitreous opacities. The case is a compensation case and the insurance carriers seem to question the connection between the injury and the eye condition. The injury resulted from rubbing the eye with his hand immediately after

having cleaned some tools with picrochlorethylene."

*Answer*: "Dr. Alice Hamilton in her book, "Industrial Toxicology" page 221, speaks of corneal ulcers following anesthetization by this chemical in German factory inspectors, and mentions its selective action on the optic nerve. The intraocular complication would be secondary but would be considered as part of an evaluation. More information on the pathology of the eye would be indicated before a final answer could be given."

*Question VI*

"What information is there available on occupational glasses—particularly where the use of prisms and magnification is needed for loopers?"

*Answer*: "Professor Joseph Tiffin's book on "Industrial Psychology" goes into considerable length in describing the use of occupational lenses with prisms, special lens decentrations, and magnifications."

*Question VII*

"There is a question as to whether the jar of a fall causing a person to strike his head may result in a retinal detachment providing there is a pre-existing condition making detachment a possibility, also whether the jar of a fall which does *not* result in the striking of the head may be sufficient to cause detachment, and what, and when symptoms would be expected to become apparent."

*Answer*: The jar of a fall causing a person to strike his head may result in a detachment with or without a pathologic condition pre-existing, and the jar of a fall which does not result in the striking of the head also may be sufficient to cause a detachment. Such a detachment would be manifest very soon if one looked at the eye, but is not always reported and noticed soon by the individual sustaining the injury.

*Question VIII*

"Do you have available for release any statistical information covering injurious effects, either physical or mental, resulting from continuous work in offices or factories under artificial light? I particularly refer to workers on a 40 to 48-hour week basis over a period of 2 or more years, where fluorescent or artificial lighting may have been used. This information is being solicited in connection with our plans for satisfactory working conditions for members of the staff of the Secretariat."

*Answer*: There are no injurious effects, either physical or mental, resulting from continuous work in offices or factories under artificial light. Properly installed artificial lighting is satisfactory both from the standpoint of comfort and efficiency. The difficulties that one hears reported about glare and fluorescent lighting and other related problems can all be blamed on improper installation. These installations can either be hung improperly so that reflections and glare becomes quite bothersome, or the wrong type of fluorescent lamp can be used, from the standpoint of color. There are a number of different fluorescent lamps—the daylight lamp, the white light, a rosy lamp, and so forth. For instance, people working with blueprints had a good deal of trouble when their first fluorescent lights were hung that contained a good deal of blue light. The contrast was missing and they had complaints. Complaints also came in the early stages of fluorescent lighting indicating they thought it was excessive. What actually happened was that it was improperly installed."

*Question IX*

"One of our larger executives in town has taken into his head that everyone in his plant will wear glasses. This element of compulsion seems to me unnecessarily severe for many of the plant employees. In rebuttal, he informs me

that both International Harvester and Pullman Company have such a program in effect and that even the casual visitor may not enter the shop without putting on some type of protective goggle. The hazards in the particular plant I allude to seem to occur only at a few isolated spots; hence, the order which is contemplated is, in my opinion, unnecessary for the majority. Do you have any statistics on either International Harvester or Pullman Company showing actual improvement in morbidity eye rates following the enforcement of such a program or a similar program in any other factory?"

*Answer*: "The question that you bring up seems a most startling one. One of the problems in a good eye protection program is that "executives" make decisions about matters of which they know nothing. It is difficult to answer your letter properly without knowing what the plant manufactures in general. Although it is perfectly true that the Pullman Company, Chicago branch, has complete eye coverage including any and all visitors and that there are a few other companies that do this, they are all industries in which the hazards are great and are just as great if not greater for people walking through the aisles as for the operators.

"It has been our own policy in setting up such an eye program in any industry, to go through the plant with the safety director and on the basis of the layout and operations to decide:

1) The type of protection, namely what kind of goggles at what places.
2) Those sections of the plant in which it would be mandatory and those in which it would depend on the individual operations.
3) How much coming and going people do from one section to another.
4) Many other factors that are indigenous to the plant.

"As you have probably discovered, one company may have welding done, here, there and everywhere in the plant and that flash hazards exist in many spots where people working around welders have to be taken care of. Then again, all of the welders may be in one building as they are in one of the big Detroit plants. In this plant they do a great deal of welding and have about 500 welders in one building which makes an entirely different eye protection problem than will be found in any other part of the plant. Here, everyone wears not only goggles, but welder's goggles and this includes the casual visitor.

"An eye protection program will succeed only if:

1) It is practical and makes sense with the type of operation and lay-out of the plant, because the workers themselves know exactly what the score is and will lose confidence in a program handed down from management which does not have a thorough understanding of their problems.
2) The type of protection is chosen and decided on and no exceptions are made at the request of individual employees who would 'prefer' this or that.
3) An adjusting, fitting, and servicing maintenance plan is carefully outlined.

"If these items are carried out, a program will be successful. If they are not part of the picture, you can anticipate trouble in advance.

"From your description of the hazards in this plant which occur in a few isolated spots, I would certainly feel that the over-all compulsion would destroy the program and the objective, namely, the cutting down of eye accidents. An analysis of eye accidents as they have occurred in this plant over the last year or two would go a long way to assist in determining the type of eye protection program required."

*Question X*

"Would you be kind enough to let me have your appraisal of the Telebinocular and any other information that you think may be of value."

*Answer*: "The Telebinocular is a sturdy, well-made instrument built up from a hand-held stereoscope originally designed for viewing ordinary stereoscopic pictures. In 1933, this instrument was modified by attaching a special shaft and slide holder and mounting it on a stand. The slides are modifications of the original Betts Ready to Read Tests which were designed for E. A. Betts, Ph.D. to measure the visual performance of school children.

"Many capable applicants have probably been denied jobs, shifted or placed poorly because of unwise use of this instrument by well-meaning individuals, indicating its harmfulness *if* used *incorrectly*. On the other hand, a great deal of proper placement has been accomplished because of it. Many employees have had professional attention who would not have had it otherwise, thus indicating its usefulness *if* handled *correctly*.

"The Joint Committee of Industrial Ophthalmology has cooperated in a very careful analysis of thousands of records made from Telebinocular tests inside the gates of industry. These have been correlated statistically with such factors as job performance, accidents, conventional professional tests. From this factual evidence gathered, we can say:

1. The Keystone Record Forms which are supposed to classify employees for various jobs according to their visual capabilities evidently are based upon arbitrary judgments that were not justified by actual experimentation on thousands of industrial employees. A substitute and greatly simplified record form has been worked out by a member of the Joint Committee and has been proved to be more practical. The Keystone View Company is also working on a simplified record form.
2. The Telebinocular Visual Acuity Tests at distance, have, through the work of the Committee been correlated with Snellen equivalents.
3. The Depth Perception Test, while not an accurately graduated one, can be used to advantage in screening out those in the lowest quarter of performance in considering dangerous jobs.
4. The phoria tests will identify most employees with muscle imbalance lying outside of the general range of normality. These measurements cannot be named in prism diopter equivalents.
5. Several of the tests show little or no relationship with employee's success on the job or with standard clinical practice and are, therefore, likely to create the impression of an actual visual deficiency. In reality they detect what might better be called "substandard" visual function.
6. The Telebinocular has no adequate *near point test* of acuity included in their series. Therefore, as near point acuity is of major importance in many jobs, individual tests will need to be added where *near* vision skills are required.
7. The Color Vision test while inadequate as a whole is useful in identifying certain types of color deficiency.

"The Telebinocular when used wisely in the hands of a good tester who is properly trained can be a valuable aid in identifying individuals with subnormal visual performance as the tests include muscle balance, stereopsis, etc. in addition to central visual acuity. The

standards set for industry both as to pass and fail, or as to the classification of employees are impractical and not sufficiently accurate to use. Instead standards should be set up for each industry by its own medical and ophthalmological staff using the data accumulated.

*Question XI*

"Can you tell me the sources of the best information on eye strain in relation to general fatigue and its relation to accident causes. This question or problem has arisen in our plant which manufactures pulp and paper. I do not know if you are familiar with Seybold trimmers, a machine for trimming full reams of paper. There is a blade some 36" to 40" in length that moves down to cut the full length of paper. To high light this moving part that holds the blade, it has been painted orange with black striping (the DuPont Safety Color Scheme). My question is "Will this constant moving of orange and black in front of the two trimmer men tend to increase eye fatigue and thereby defeat the purpose intended?

"When the blade or trimmer is in operation, the operators are perhaps 12" to 16" from this moving part. The potential hazard is, of course, that some one may get a finger or hand under the blade even with control switches and guards wherever possible. In other words, the human element?"

*Answer*: "Actually, the word eye strain is one of the words like rheumatism which covers a multitude of sin. I presume when you speak of 'eye strain', you mean eye fatigue in relation to general fatigue and in relation to accidents. Any constantly moving object within the line of vision or within the field of vision is a source of visual irritation—a sort of bombardment against which efficient visual function rebels. The best example of this is bottle inspection. Here a conveyor belt carrying bottles moves from left to right—the inspector working through a large magnifying glass directly in front of her. The constant motion of objects in the periphery of her vision *is* a source of a visual fatigue. The proper screening of these lines of moving bottles with which she isn't concerned has increased the time which these girls can work. There are other factors in this particular operation which have also played a part in fatigue such as direct illumination into their eyes instead of up through the bottles (which is being corrected) adds to the length of time a girl can work. I doubt if the color except as it accentuates the part which holds the blades itself, adds to the fatigue, although the contrast does produce a greater retinal stimulation than if it were an obscure color.

"In this instance you will need to weigh the hazard of a potential loss of finger or hand versus the slightly increased visual fatigue due to the bombardment of moving color and action stimuli. I think, however, if I were the operator, I would prefer to have my hands protected.

"Of great assistance in cases like this is interruption of work at more frequent intervals than had been previously thought of. To change the distance in which one's eyes are working and to do other things with them with other backgrounds and colors, etc. is of tremendous value. You might, therefore, substitute for the increased irritability that the moving colored object makes, five minute intervals or these operators could do something else (visually speaking). Am looking forward to visiting several of the pulp and paper plants in Wisconsin *if* I can only get the time. Perhaps then I'll be a little bit more intelligent about answering questions relative to their problems. Meanwhile, I hope the above will be of some assistance."

## ITEMS OF INTEREST SENT THROUGH CORRESPONDENCE

1. Dr. Eugene Chan writing from Hong-Kong, China, sends the following:

"Studies conducted on the industrial health of employees in rug factories in China reveals an appalling number with trachoma—it would be fitting to start a campaign against this disease in each industrial center, and at the same time use this opportunity for the investigation of other ocular disabilities."

2. Joseph Minton, ophthalmologist to the Dept. of Industrial Ophthalmology at the Royal Eye Hospital in London reports cases of workers in an artificial silk factory suffering from punctate keratitis in which he felt that the hydrogen sulfide present in the air in the acid house was responsible. He states: "The presence of 10 parts in a million of hydrogen sulfide in the air, is sufficient to cause a keratitis." He also speaks of the inhalation of alcohol, used as a solvent for shellac, paint removers, anolin dyes, perfume and toilet preparations, as causing optic neuritis and ocular palsies. Other items he has mentioned are carbon bisulphide, the inhalation of which produces an amblyopia and optic neuritis. The loss of the peripheral field results from the use of carbon tetrachloride while a trichlorethylene used as a degreasing agent in dry cleaning operations, may cause an optic neuritis and retinitis. Dr. Minton described with great interest the new department of Industrial Ophthalmology set up at the new Royal Eye Hospital in London where he stressed the importance of a good history and the subsequent study of safety appliances used. Dr. Minton further stressed the opportunity for research in industrial medical departments and eye clinics of cities with industrial sections.

## CONTINUING BIBLIOGRAPHY ON OCCUPATIONAL EYE PROBLEMS


ALGER, A. L. *The Eye Test Fallacy.* Flying, March, 1946.

ANON. *Non-Magnetic Intraocular Foreign Bodies.* Editorial, Brit. M.J 1:228, Feb., 1945.

ANON. *Specifications and Methods for Testing Plastic Eye and Face Protective Devices used for the Prevention of Accidents to Workers in Industry.* Industrial Safety Equipment Association, 366 Madison Avenue, New York 17.

BEEBE-CENTER, J. G.; MEAD, L. C.; WAGONER, K. S. AND HOFFMAN, A. C. *Visual Acuity and Distance of Observation.* J. Exper. Psychol. 35:473-484, Dec., 1945.

BERRY, G. L. *Visual Discomfort in Eye Workers Due to Glasses; Causes and Cures.* J. Oklahoma M. A. 38:361-367, Sept., 1945.

BLACK, E. C. *Eye Wounds in Industry.* Tr. Ophthal. Soc. Australia (1944) 4:16-27, 1946.

COLEMAN, J. H. *Visual Classification Technique for Industry.* (Symposium on Screening Techniques for Eye Defects at the New York Society for Clinical Ophthalmology, April 2, 1945) Published by Sperry Gyroscope Co.

CROCKETT, J. A. *Causes of Economic Blindness in Texas: Statistical Survey of 6,106 Cases, Aid to Blind Program.* Texas State Dept. of Public Welfare. Texas State J. Med. 51:519-524, Feb., 1946.

DARLEY, W. G. AND GAETJENS, A. K. *What Price Industrial Eye Comfort.* Illuminating Engineering, Dec., 1944.

DIMMICK, FORREST LEE. *A Color Aptitude Test,* 1940 Experimental Edition. Jr. Appl. Psychol. 30:10-22, Feb., 1946.

FISHENDEN, R. B. *Types, Paper and Printing in Relation to Eyestrain.* Brit. J. Ophth. 30:20, Jan., 1946.

HOFFMAN, A. C. *Eye Movements During Prolonged Reading.* J. Exper. Psychol. 36:95-118, April, 1946.

JACOBSON, M. AND LEVIN N. *Epidemic Keratoconjunctivitis Cured Through the Combination of Penicillin and Sulfasuxidene.* New York State J. Med. 45:1990-, Sept. 15, 1945.

JOHNSTONE, I. L. *Heat Ray Cataract.* Tr. Ophth. Soc. U. Kingdom (1944) 64:252-256, 1945.

KUHN, HEDWIG S. *Emergency Nursing Care of the Eyes in Industry.* Am. Jr. of Nursing, Vol. 47, Jan., 1947.

LUKE, W. R. F. *Eye Conditions in Industrial Medicine.* W. R. F. Luke. Bull. Acad. M. Toronto, 19:121-24, March, 1946.

McLAUGHLIN, RALPH S. *Chemical Burns of the Human Cornea.* Am. J. Ophth. 29:11, 1355-1362, Nov., 1946.

MASTACHE AND SCHNOOR, THOS. *Visual Aid for Presbyopic Welders.* Permanante Foundation Medical Bulletin, 3:2, April, 1945.

MINTON, JOSEPH. *Ophthalmic Problems in Industry.* Nursing Times 41:588-89, Sept. 8, 1945.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS. *Eye Health — A Teaching Handbook for Nurses.* Nat. Soc. for Prev- of Blindness, New York, 1946, 108 pages. Publication No. 447.

NELSON, FLORENCE. *Industrial Aspects of Eye Care.* Trained Nurse and Hospital Review, June, 1946.

PINSON, E. A. AND CHAPANIS A. *Visual Factors in Design of Military Aircraft.* Jr. Aviation Medicine. 17:115-122, March-April, 1946.

RENSHAW, SAMUEL. *Seeing is More than Vision.* Visual Digest 9:1, Summer, 1945. Pamphlet Public Health Bureau, Amer. Opt. Assoc., 707 Jenkins Bldg., Pittsburgh, Pa.

SCHAEFER, A. J. *Effect of Certain Amino Acids on Healing of Experimental Wounds of Cornea.* Soc. Exper. Biol. and Med. 61:165-166, Feb., 1946.

SILVER, EDWIN H. *Illumination of Eye Charts for Traffic Bureaus.* Dr. Edwin H. Silver, American Optom. Assoc. Washington, D. C., 1945.

STRAYER, G. *Intra-ocular Steel Particles; 5 Successive Cases.* J. M. A. Alabama, 15:41-43, Aug., 1945.

WRIGHT, L. D. *Eyesight Conservation and Lighting; Need for Medical Interest and Leadership.* Tr. Ophthal. Soc. Australia (1944) 4:28-39, 1946.

# SECTION ON INSTRUCTION

## FIFTY-SECOND ANNUAL CONVENTION

## American Academy of Ophthalmology and Otolaryngology

## OCTOBER 12 TO 17, 1947

## PALMER HOUSE

*Chicago, Illinois*

DEAN M. LIERLE, M.D.
*Secretary for Instruction — Otolaryngology*

A. D. RUEDEMANN, M.D.
*Secretary for Instruction — Ophthalmology*

This Section Presents for 1947:

1. One hundred ten individual and forty-two continuous courses in ophthalmology, with a faculty of one hundred fifty-five instructors, covering three hundred fifty-nine hours.
2. Eighty-three individual and thirty-nine continuous courses in otolaryngology, with a faculty of ninety-eight instructors, covering three hundred forty-one hours.

### General Instructions

*Members only who have paid their* 1947 *dues may order instruction tickets in advance of the meeting.* 1947 Academy candidates are not privileged to reserve tickets. After the session opens no preference will be given to members that is not extended to candidates and guests.

*Orders unaccompanied by properly executed checks will not be filled.* Checks should be made payable to Dr. W. L. Benedict.

Refunds will be granted only if request for reimbursement is received prior to October 10.

All orders for tickets will be filled according to postmark on envelope. Previous to October 1, send orders to 100 First Avenue Building, Rochester, Minnesota; after October 1, mail orders direct to Dr. William L. Benedict, c/o Palmer House, Chicago, Illinois.

### Ordering Instruction Tickets

At the end of this section is printed a colored sheet to be used when ordering tickets in advance. Specify preferences on this sheet and mail to the executive office. Careful following of instructions will insure the correct filling of your requests. *Since number of tickets for each course is limited it is necessary that you select more courses than it is possible for you to attend; in other words, a list of three preferences for each period selected should be given.* Please note carefully that where continuous courses are selected the additional periods open in your schedule are varied.

If you are requesting registration in the continuous courses, please specify preferences for individual courses as substitutes in order to avoid disappointment should the continuous courses which you select be sold out when your order is received.

### Claiming Instruction Tickets

There will be an *Advance Order Section* at the Registration Desk during the week of the Convention where members who have sent advance orders will register and secure tickets.

## Selection of Courses

($1.50 for each period of instruction)

SEVENTH FLOOR — PALMER HOUSE

The periods will be designated with the following key: Select tickets for— Otolaryngo'ogy forenoons, periods 1-2-3 — Ophthalmology, afternoons, periods 4-5-6

| Day | (M)onday | (T)uesday | (W)ednesday | (Th)ursday | (F)riday |
|---|---|---|---|---|---|
| Period a.m. | | 1 2 3 | 1 2 3 | 1 2 3 | 1 2 3 |
| Period p.m. | 4 5 6 | 4 5 6 | 4 5 6 | 4 5 6 | |

M-4 ..... 2:00 p.m. to 3:00 p.m.
M-5 ..... 3:15 p.m. to 4:15 p.m.
M-6 ..... 4:30 p.m. to 5:30 p.m.
T-1 ..... 9:00 a.m. to 10:00 a.m.
T-2 .....10:15 a.m. to 11:15 a.m.
T-3 .....11:30 a.m. to 12:30 p.m.
T-4 ..... 2:00 p.m. to 3:00 p.m.
T-5 ..... 3:15 p.m. to 4:15 p.m.
T-6 ..... 4:30 p.m. to 5:30 p.m.
W-1 ..... 9:00 a.m. to 10:00 a.m.
W-2 .....10:15 a.m. to 11:15 a.m.
W-3 .....11:30 a.m. to 12:30 p.m.
W-4 ..... 2:00 p.m. to 3:00 p.m.
W-5 ..... 3:15 p.m. to 4:15 p.m.
W-6 ..... 4:30 p.m. to 5:30 p.m.
Th.-1 ..... 9:00 a.m. to 10:00 a.m.
Th.-2 .....10:15 a.m. to 11:15 a.m.
Th.-3 .....11:30 a.m. to 12:30 p.m.
Th.-4 ..... 2:00 p.m. to 3:00 p.m.
Th.-5 ..... 3:15 p.m. to 4:15 p.m.
Th.-6 ..... 4:30 p.m. to 5:30 p.m.
F-1 ..... 9:00 a.m. to 10:00 a.m.
F-2 .....10:15 a.m. to 11:15 a.m.
F-3 .....11:30 a.m. to 12:30 p.m.

(Familiarity with this key will be helpful and will avoid confusion)

# PROGRAM OF COURSES

## OPHTHALMOLOGY

### Continuous Courses

### 1947

**COURSE 1**

Room 706 Periods M-4, M-5

**COURSE 2**

Room 706 Periods T-4, T-5

**JAMES H. ALLEN, M.D.**
Iowa City, Iowa

**Bacteriology**

*A two-period course—$3.00*

Dr. Allen's two-hour course "Bacteriology" is being presented twice as Course 1 and Course 2. **Please select course number according to day on which you wish to attend.**

Methods of bacteriologic examination and diagnosis which may be used in routine office practise will be discussed as well as indications for further laboratory examinations. Therapy will be discussed in relation to the etiology of infectious diseases of the eye.

**COURSE 3**

Room 786 Periods M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5

**HAROLD W. BROWN, M.D., et al**
New York, N. Y.

**Eye Muscles**

*An eight-period course—$12.00*

**M-4 & 5 — Physiology and Pathology of the Ocular Muscles**
Harold W. Brown, M.D.

The physiological action of the individual muscles, conjugate and disjunctive movements, (convergence, divergence) will be discussed and illustrated. From these normal physiological acts, the pathologic variations are developed and classified. Methods of different diagnosis, measurements of deviation and the surgical and nonsurgical correction of these deviations will be presented.

**T-4 & 5 — Tests**
Maynard C. Wheeler, M.D.

This will include a demonstration and discussion of the most important methods

used in the diagnosis and measurement of muscle defects. Particular emphasis will be placed on the screen test and its actual application in routine practice.

**W-4 & 5 — Congenital Anomalies**

Rudolf Aebli, M.D.

The subject matter will cover the more usual congenital anomalies such as retraction syndrome and its variations, strabismus fixus, Marcus Gunn jaw winking syndrome, paralyses of both elevators of one eye with true and pseudoptosis, and also congenital individual muscle anomalies. Differential diagnosis and treatment, if any, will be discussed.

**Th-4 & 5 — Surgery of the Extraocular Muscles**

Harold W. Brown, M.D.
and
Rudolf Aebli, M.D.

The indications and contraindications of surgical correction of heterophoria and heterotropia will be discussed. Special emphasis will be placed on the methods used in selecting the proper muscle or muscles to be operated on and the amount of correction that can be expected from the different types of surgical procedures.

---

**COURSE 4**

Room 716 Periods W-4, W-5

**COURSE 5**

Room 716 Periods Th-4, Th-5

**J. C. COPELAND**
Chicago, Ill.
BY INVITATION

**Streak Retinoscopy**

*A two-period course—$3.00*

Mr. Copeland's two-hour course "Streak Retinoscopy" is being presented twice as Course 4 and Course 5. **Please select course number according to day on which you wish to attend.**

The following outline describes the steps of the demonstration:

1. Determination of the neutral point.
2. Revealing low astigmatic errors.
3. Locating the zone of the astigmatic band.
4. Determining the power of the cylinder.
5. Estimating the type and degree of error without lenses.
6. "Guide Angle" technic, in refining axis position.
7. Plus cylinder procedure.

---

**COURSE 6**

Room 722 Periods M-5, M-6

**COURSE 7**

Room 722 Periods T-5, T-6

**FREDERICK C. CORDES, M.D.**
San Francisco, Calif.

**Acquired and Congenital Cataracts**

*A two-period course—$3.00*

Dr. Cordes' two-hour course "Acquired and Congenital Cataracts" is being presented twice as Course 6 and Course 7. **Please select course number according to day on which you wish to attend.**

The course deals with various types of cataracts. The first hour will be devoted to a brief review of the embryology of the lens followed by discussion of congenital cataracts. In the second hour, the various forms of acquired lens opacities will be described. Kodachrome slides will be used.

---

**COURSE 8**

Room 752 Periods M-5, T-5, W-5, Th-5

**JOHN H. DUNNINGTON, M.D.**
New York, N. Y.

**Eye Muscles — Surgical Treatment of Heterophoria and Heterotropia**

*A four-period course—$6.00*

Included in this course will be a discussion of the preoperative analysis, surgical technic and postoperative care of various motor anomolies. Emphasis will be placed upon factors influencing the surgeon in the selection of the type of operative procedure to be employed.

---

**COURSE 9**

Room 710 Periods Th-5, Th-6

**HAROLD F. FALLS, M.D.**
Ann Arbor, Mich.

**Heredity**

*A two-period course—$3.00*

An introduction to the genetics of common hereditary ocular anomalies. Primary principles of heredity will be briefly discussed and demonstrated. Illustrations and slides of the ocular dyscrasias will accompany the didactic subject material. An outline of information requisite to accurate prognostication of the inheritance pattern of numerous ocular defects will be presented.

---

**COURSE 10**

Room 788 Periods M-4, M-5, T-4, T-5

**BRUCE FRALICK, M.D.**
and
**JOHN HENDERSON, M.D.**
BY INVITATION
Ann Arbor, Mich.

**Anatomy of the Orbit**

*A four-period course—$6.00*

Understanding the anatomy of the eye is a great help to any ophthalmic surgeon in performing the various surgical procedures on the globe and adnexa. It familiarizes one with normal relationships, thereby giving confidence when conducting corrective operations in complicated cases. A knowledge of anatomy also includes an understanding of the variations from the normal and the effect these variations play on the functioning of the part. This aids the surgeon in encountering anomalous relationships.

A brief description will be given of the anatomical characteristics of the bony orbit and its contents, the eyelids, the lacrimal apparatus and nervous pathways to illustrate the various surgical problems which each tissue presents to the ophthalmic surgeon. Practical illustrations will be used throughout in an attempt to demonstrate the value of a thorough knowledge of applied ophthalmic anatomy, if one is to get the highest percentage of good surgical results.

---

**COURSE 11**

Room 782 Periods M-4, M-5, M-6

**JONAS FRIEDENWALD, M.D.**

and

**WILLIAM F. HUGHES, JR., M.D.**

Baltimore, Md.

**Studies on the Physiology and Pathology of the Cornea in Relation to Chemical Burns**

*A three-period course—$4.50*

War time studies on the mechanism of injury of the cornea by toxic agents have added greatly to our knowledge of the physiology, metabolism, pathology, and toxicology of the cornea. The new data will be reviewed and their implications in respect to civilian clinical problems discussed.

---

**COURSE 12**

Room 726 Periods W-4, W-5

**COURSE 13**

Room 726 Periods Th-4, Th-5

**ISADORE GIVNER, M.D.**

New York, N. Y.

**Practical Application of Ophthalmological Bacteriology**

*A two-period course—$3.00*

Dr. Givner's two-hour course "Practical Application of Ophthalmological Bacteriology" is being presented twice as Course 12 and Course 13. **Please select course number according to day on which you wish to attend.**

During the first period the following will be discussed:

A. Mode of action of antibiotics
B. Normal conjunctiva
C. Conjunctivitis of the new-born
D. Handling of lid infections (bacterial and viral)
E. Bacterial synergisms in lid necrosis
F. Acute conjunctivitis (bacterial and viral)
G. Fungus disease of the eye
H. Bacteriophage in ophthalmology
I. Chronic conjunctivitis and its handling

During the second period the following will be discussed:

A. Ulcers of the cornea (bacterial and viral)
B. Sarcoid of the eye
C. Brucellosis
D. Schwartzman phenomena in the eye
E. Harada's Disease and Vogt-Koyanagi Disease
F. Uveitis

---

**COURSE 14**

Room 728 Periods M-5, M-6

**COURSE 15**

Room 728 Periods T-5, T-6

**HENDRIE W. GRANT, M.D.**

St. Paul, Minn.

**Practical Orthoptics**

*A two-period course—$3.00*

Dr. Grant's two-hour course "Practical Orthoptics" is being presented twice as Course 14 and Course 15. **Please select course number according to day on which you wish to attend.**

A consideration of the practical principles involved in paralytic strabismus, convergent strabismus, divergent strabismus, and heterophoria.

---

**COURSE 16**

Room 704 Periods M-4, M-5

**COURSE 17**

Room 704 Periods T-4, T-5

**LeGRAND HARDY, M.D.**

and

**GERTRUDE RAND, Ph.D.**

BY INVITATION

New York, N. Y.

**Recent Developments in Color Vision Testing**

*A two-period course—$3.00*

Dr. Hardy's and Dr. Rand's two-hour course "Recent Developments in Color Vision Testing" is being presented twice

as Course 16 and Course 17. **Please select course number according to day on which you wish to attend.**

Color vision testing requires standardization of two out of three variables involved in the process. The most important factors in color vision testing will be outlined. The important triads in seeing color, in illumination, in the color vision process and in the characteristics of the object will be described and related to the ICI-Coordinate System and Standard Observer. Color tests will be described, evaluated and demonstrated. Recent progress in research will be discussed.

**COURSE 18**

Room 782 Periods T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6

**LeGRAND HARDY, M.D., et al**

**Orthoptics**

*A nine-period course—$13.50*

**T-4 — Evaluation of Orthoptics**
LeGrand Hardy, M.D.
New York, N. Y.

**T-5 — Clinical Orthoptics**
John B. Hitz, M.D.
Milwaukee, Wis.

**T-6 — Fusion**
Earnest A. W. Sheppard, M.D.
Washington, D.C.

**W-4 — Orthoptics in Children**
Frank D. Costenbader, M.D.
Washington, D.C.

**W-5 — Treatment of ARC**
Julia Lancaster
San Francisco, Calif.

**W-6 — Orthoptics Without Technician**
Elizabeth Stark
New York, N. Y.

**Th-4 — Adult Orthoptics**
Beulah Cushman, M.D.
Chicago, Ill.

**Th-5 — Orthoptics With Neuro-ophthalmology**
George P. Guibor, M.D.
Chicago, Ill.

**Th-6 — Discussion**

**COURSE 19**

Room 727 Periods M-4, M-5

**COURSE 20**

Room 727 Periods T-4, T-5

**FRITZ JARDON**
BY INVITATION
and
**CONRAD NOELLE**
BY INVITATION
Southbridge, Mass.

**Plastic Prosthesis**

*A two-period course—$3.00*

Mr. Jardon's and Mr. Noelle's two-hour course "Plastic Prosthesis" is being presented twice as Course 19 and Course 20. **Please select course number according to day on which you wish to attend.**

Methods used in estimating the size of the prosthesis and the center of the cornea, color, etc.

**COURSE 21**

Room 786 Periods M-6, T-6, W-6

**FERDINAND L. P. KOCH, M.D.**
New York, N.Y.

**Integration of Classifications of Systemic Hypertensive Disease**

*A three-period course—$4.50*

The useful few of the several classifications of essential hypertension employed by the specialty and/or general practitioner should be readily applicable to daily office and hospital practice and to research purposes. Synonomous correlation of these classifications and their salient features will be illustrated by slides in this course.

**COURSE 22**

Room 738 Periods W-4, W-5

**COURSE 23**

Room 738 Periods Th-4, Th-5

**PETER KRONFELD, M.D.**
Chicago, Ill.

**Gonioscopy**

*A two-period course—$3.00*

Dr. Kronfeld's two-hour course "Gonioscopy" is being presented twice as Course 22 and Course 23. **Please select course number according to day on which you wish to attend.**

There are many situations in ophthalmology in which gonioscopy yields information of great practical value, diagnostic, as well as therapeutic. Aside from these situations, gonioscopy in nearly every case in which it is used, gives information that serves to integrate our knowledge and understanding of ophthalmology. On the other hand, gonioscopy has failed to give answers to a number of questions which the ophthalmologists had, as it were, saved up for the advent of this new method.

**COURSE 24**

Room 784 Periods M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6

**HEDWIG S. KUHN, M.D. et al**

**Industrial Ophthalmology**

*A twelve-period course—$18.00*

The following subjects will be discussed:

**M-4 — Opportunities and Responsibilities.**
**M-5 — What is an Industrial Eye Program?**
**M-6 — Testing Technic.**
**T-4 — Fact Finding Methods and Statistical Methods.**
**T-5 — Eye Protection.**
**T-6 — Toxic Hazards.**
**W-4 & 5 — Round Table — "Plant Experience."**
**W-6 — Compensation and Insurance.**
**Th-4 — Radiation — Ultraviolet, Infrared, etc.**
**Th-5 — Visual Skills and Correcting for the Position.**
**Th-6 — Questions and Answers for Participants in the Course.**

---

**COURSE 25**

Room 736 Periods W-5, W-6, Th-5, Th-6

**WALTER B. LANCASTER, M.D.**
and
**ARTHUR LINKSZ, M.D.**
BY INVITATION
Boston, Mass.

**Aniseikonia**

*A four-period course—$6.00*

We shall present the subject from the point of view of theory and practice, explaining fully its influence on binocular vision and also its bearing on eyestrain from a clinical point of view. We shall try to make it clear that the number of cases needing aniseikonic lens correction in their spectacles is very small.

---

**COURSE 26**

Room 731 Periods M-5, M-6

**COURSE 27**

Room 731 Periods T-5, T-6

**RALPH I. LLOYD, M.D.**
Brooklyn, N. Y.

**Ophthalmoscopy — Central Macular Degeneration — Senile and Hereditary**

*A two-period course—$3.00*

Dr. Lloyd's two-hour course "Ophthamoscopy—Central Macular Degeneration —Senile and Hereditary" is being presented twice as Course 26 and Course 27. **Please select course number according to day on which you wish to attend.**

A two-hour session devoted to diagnosis of macular lesions. Red-free light; binocular ophthalmoscopy; and the laboratory supplement, the hand scope, and observations over an extended period are especially valuable. Hereditary lesions of the macula and pseudo-tumors receive particular attention.

**COURSE 28**

Room 796 Periods T-4, W-4, Th-4

**DONALD LYLE, M.D.**
Cincinnati, Ohio

**Neuro-Ophthalmology — Clinical Studies of the Patient With Ocular Manifestations of Neurological Diseases**

*A three-period course—$4.50*

Lesions produced by inflammations, primary degenerations, tumors, and vascular conditions affecting: 1. Visual system. 2. Eye motor system.

Ophthalmologic consideration of increased intracranial pressure from space consuming lesions: 1. Above the tentorium. 2. Below the tentorium.

A grouping into syndromes is made, in so far as is possible, for the purpose of simplicity.

---

**COURSE 29**

Room 793 Periods M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6

**ROBERT J. MASTERS, M.D.**
Indianapolis, Ind.
**HARVEY E. THORPE, M.D.**
Pittsburgh, Pa.
**PETER KRONFELD, M.D.**
Chicago, Ill.
**EVERETT L. GOAR, M.D.**
Houston, Texas

**Slit Lamp Biomicroscopy**

*A twelve-period course—$18.00*

The lectures and demonstrations of this course are planned for beginners in slit lamp microscopy and for those registrants who desire a review of the elementary features of biomicroscopy.

The early part of each session is devoted to an illustrated lecture, which is followed by a period of actual work with the slit lamps by the members of the class.

The illustrated lectures cover, in regular order from day to day, studies of the normal and pathologic cornea, anterior chamber, iris, crystalline lens, and vitreous humor. Special attention is given to the findings which can be made with no other instrument, and which make the slit lamp indispensable in the routine practice of ophthalmology.

The demonstration period is utilized to make each class member familiar with the manipulation of the instruments. During each demonstration period a round table, question-and-answer discussion is carried on for the class members who are already familiar with the instruments.

**COURSE 30**

Room 754 Periods M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5

**JOHN M. McLEAN, M.D.**
New York, N.Y.
and
**JACK S. GUYTON, M.D.**
Baltimore, Md.

**Cataract Surgery**

*An eight-period course—$12.00*

A course on the technic of intracapsular cataract extraction. It includes detailed discussion of preoperative and postoperative management, anesthesia and akinesia, various types of incisions and sutures, management of iris, methods of intracapsular lens delivery, and indications. Operative and postoperative complications. Motion picture and lantern slide demonstrations.

---

**COURSE 31**

Room 796 Periods M-5, M-6, T-5, T-6, W-5, W-6, Th-5, Th-6

**SAMUEL J. MEYER, M.D. et al**

**Glaucoma**

*An eight-period course—$12.00*

**M-5 — The Glaucomas — Definition, Mechanisms, Classification**
Peter C. Kronfeld, M.D.
Chicago, Ill.

This paper deals with the definition, mechanisms, and classification of the ocular diseases comprised under the term "the glaucomas," associated with or due to elevated intraocular pressure. The three important factors of inflow of aqueous, outflow of aqueous, and intraocular blood volume are discussed in relation to hypertension, as well as the mechanisms influencing each. A tentative classification of the glaucomas, based upon the present knowledge, is suggested and elaborated upon.

**M-6 — The Histopathology of Acute Glaucoma** (half period)
Brittain F. Payne, M.D.
New York, N.Y.

Discussion of the microscopic findings of the different ocular tissues under the influence of sudden and acute increase of intraocular pressure. The variations in size and content of the various parts of the clinical picture of the disease and these are correlated by the author. The lack of specimens of fresh acute hypertension leaves many gaps in the pathological picture.

**M-6 — The Histopathology of Chronic Glaucoma** (half period)
Georgiana Dvorak-Theobald, M.D.
Oak Park, Ill.

The microscopic anatomy of the all-important chamber angle is described in detail and is correlated with the clinical picture of that type of glaucoma that is not associated with congestion. Of especial importance is the relationship between the Canal of Schlemm and the trabeculum. Due to the lack of specimens of early cases, some of the gaps in the histological picture have to be filled by deductions and speculations.

**T-5 — Narrow Angle Glaucoma.**
Thomas D. Allen, M.D.
Chicago, Ill.

In certain cases of elevated intraocular pressure, the angle of the anterior chamber is very shallow and this is associated with definite clinical pictures. In the majority of cases, acute congestion of the eyeball accompanies and complicates the condition, although in some cases that phase is lacking. The clinical appearance of both conditions is described and discussed at length as well as the therapeutic measures indicated.

**T-6 — Wide Angle or Compensated Glaucoma.**
Samuel J. Meyer, M.D.
Chicago, Ill.

This is the most common form of glaucoma and the one that in its earliest stages requires the most careful diagnostic acumen. The condition is described in detail, structure by structure, and the diagnostic phases are evaluated for the various stages of the disease. Provocative methods are elaborated upon for both suspected as well as existing hypertension. The over-all picture is then rounded out.

**W-5 — Diagnostic and Differential Diagnostic Problems.**
Frederick C. Cordes, M.D.
San Francisco, Calif.

Before the management of a case of glaucoma may be undertaken, it is essential that it be placed in one of the accepted classifications where the etiological aspect is considered. For that reason, the author deals at length with the differential diagnostic problems of the various forms of elevated intraocular pressure and points out in detail the various aspects that allow of an accurate diagnosis which in turn indicates the proper course of treatment necessary to control the condition.

**W-6 — Glaucoma Secondary to Uveitis.**
F. Bruce Fralick, M.D.
Ann Arbor, Mich.

This is an elaborated discussion of hypertension due to inflammations of the uvea. There are considered the sudden pressure increases due to acute and chronic uveitis and the gradual pressure increases due to these causes. Methods of treatment are discussed, both medical and surgical.

**Th-5 — The Principles of Nonsurgical Treatment of Hypertension.**
Glen W. Nethercut, M.D.
Chicago, Ill.

In the first part of the lecture, the effects of elevated intraocular pressure upon the retina and optic nerve are discussed, from an anatomical as well as from a functional standpoint. The two aspects are then correlated. In the second part, the principles and methods of non-surgical treatment of elevated intraocular pressure are described at length. The lecture closes with a detailed description and discussion of methods of use of the various medications, both local and systemic, that are employed.

**Th-6 — The Principles of Surgical Treatment of Hypertension.**
Samuel J. Meyer, M.D.
Chicago, Ill.

---

**COURSE 32**

Room 754 Periods M-6, T-6, W-6, Th-6

**BRITTAIN F. PAYNE, M.D.**
New York, N.Y.

**Advanced Eye Pathology**
*A four-period course—$6.00*

**M-6 — Pathologic Changes in Unsuccessful Cataract Operations.**
**T-6 — Histologic Causes of Failure in Glaucoma Surgery.**
**W-6 — The Pathology of Sympathetic Ophthalmitis.**
**Th-6—Intraocular Tumors.**

---

**COURSE 33**

Room 736 Periods M-4, M-5

**COURSE 34**

Room 736 Periods T-4, T-5

**DOHRMAN K. PISCHEL, M.D.**
San Francisco, Calif.

**Retinal Detachment — Treatment**
*A two-period course—$3.00*

Dr. Pischel's two-hour course "Retinal Detachment—Treatment" is being presented twice as Course 33 and Course 34. **Please select course number according to day on which you wish to attend.**

After a brief review of the anatomical factors involved in detachments of the retina, the present theory as to its development will be briefly presented. The history of a case of retinal detachment will be discussed with especial reference to liability cases. The diagnosis and operative treatment, together with preoperative and postoperative care will be taken up.

---

**COURSE 35**

Room 781 Periods M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6

**BENJAMIN RONES, M.D.**
and
**M. NOEL STOW, M.D.**
Washington, D.C.

**Clinical Pathology**
*A twelve-period course—$18.00*

This course is designed to carry on from the point of termination of the basic course on histopathology of the eye. The clinical entities of disease affecting the cornea, lens, uveal tract, retina, optic nerve, and orbit will be related to the underlying pathologic disturbances. There will be copious illustrations of both the clinical and pathologic material.

---

**COURSE 36**

Room 738 Periods M-4, M-5

**COURSE 37**

Room 738 Periods T-4, T-5

**C. WILBUR RUCKER, M.D.**
and
**J. GRAFTON LOVE, M.D.**
BY INVITATION
Rochester, Minn.

**Differential Diagnosis and Treatment of Lesions in Region of Chiasm**
*A two-period course—$3.00*

Dr. Rucker's and Dr. Love's two-hour course "Differential Diagnosis and Treatment of Lesions in Region of the Chiasm" is being presented twice as Course 36 and Course 37. **Please select course number according to day on which you wish to attend.**

The various types of field defects produced by lesions at the chiasm will be demonstrated. Aids to differential diagnosis of pituitary adenomas, craniopharyngiomas, meningiomas, epidermoids, aneurisms, and chiasmal arachnoiditis will be suggested. Surgical procedures for diagnosis and treatment will be discussed. The effects of surgery on vision will be demonstrated.

**COURSE 38**

Room 776 Periods W-5, W-6

**COURSE 39**

Room 776 Periods Th-5, Th-6

**RICHARD G. SCOBEE, M.D.**
St. Louis, Mo.

**The Value of the Routine Check of Ocular Muscles**

*A two-period course—$3.00*

Dr. Scobee's two-hour course "The Value of the Routine Check of Ocular Muscles" is being presented twice as Course 38 and Course 39. **Please select course number according to day on which you wish to attend.**

Muscle balance is often checked as an after-thought in doing a refraction, if it is checked at all. Between 20-30 per cent of patients have some upset in their muscle balance. Such an upset should modify the final lens prescription in many instances. Significant findings will be considered in detail.

**COURSE 40**

Room 788 Periods M-6, T-6, W-6, Th-6

**CHARLES SHEARD, Ph.D.**
Rochester, Minn.
BY INVITATION

**Applied Physiological Optics**

*A four-period course—$6.00*

**M-6 — Fundamental Physical and Mathematical Principles Underlying Physiological and Ophthalmic Optics.**

**T-6 — The Eye: Physical and Optical Data and Considerations, Including Applications to Combinations of Eye and Spectacle Lens.**

**W-6 — Catoptrics and Dioptrics: Fundamental Principles of and Applications to Ophthalmoscopy, Ophthalmometry, Phorometry, Retinoscopy and Subjective Testing.**

**Th-6 — Applications to Problems of Accommodation, Convergence and Subnormal Vision.**

**COURSE 41**

Room 795 Periods M-4, M-5, M-6, T-4 T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6

**GEORGIANA D. THEOBALD, M.D. et al**

**Histopathology — Elementary**

*A twelve-period course—$18.00*

The staff of the elementary pathology course will be comprised of the following:

Theodore Sanders, M.D.
St. Louis, Mo.
Alson Braley, M.D.
New York, N. Y.
John McGavic, M.D.
Bryn Mawr, Pa.
Charles Perera, M.D.
New York, N.Y.
Michael Hogan, M.D.
San Francisco, Calif.
Merrill Reeh, M.D.
St. Louis, Mo.
Edith Parkhill, M.D.
Rochester, Minn.
BY INVITATION
Helenore Campbell Wilder
Washington, D.C.
BY INVITATION
Brittain Payne, M.D.
New York, N. Y.
Georgiana D. Theobold, M.D.
Oak Park, Ill.

**COURSE 42**

Room 752 Periods M-6, T-6, W-6, Th-6

**ALAN C. WOODS, M.D.**
and
**WILLIAM C. OWENS, M.D.**
Baltimore, Md.

**Medical Ophthalmology**

*A four-period course—$6.00*

**M-6 — Clinical Symptomatology of Ocular Tuberculosis.**

**T-6 — Diagnosis, Pathogenesis, and Treatment of Ocular Tuberculosis.**

**W-6 — Ocular Complications of Congenital Syphilis.**

**Th-6 — Ocular Complications of Acquired Syphilis.**

# OPHTHALMOLOGY

## Individual Courses

## 1947

**COURSE 101**

Room 712 Periods M-4, T-4

**MOACYR E. ALVARO, M.D.**
Sao Paulo, Brazil

**Uveitis**

Chronic uveitis, prevention of sympathetic ophthalmia, etiology, pathogeny, clinical symptoms, treatment, and prognosis of chronic uveitis will be discussed along with the different views on sympathetic ophthalmia. The importance of eliminating all possible causes of uveitis in every case of perforating ocular wounds will be stressed as an important measure in prevention of sympathetic ophthalmia.

**COURSE 102**

Room 705 Periods M-6, T-6

**BENNETT Y. ALVIS, M.D.**

St. Louis, Mo.

**Evaluation of Operations for Glaucoma**

Operations will be classified as those designed for:

a. Opening natural drainage.
b. New intraocular drainage.
c. Extraocular drainage; in order of effectiveness:
   1. Ragged iridectomy.
   2. Iris inclusion.
   3. Iridosclerectomy.
   4. Trephining.
d. Reducing intraocular fluid, cyclodiathermy.

Technic of operations will be discussed to elucidate their relative effectiveness and their suitability for different types of glaucoma.

**COURSE 103**

Room 710 Periods M-6, T-6

**WALTER S. ATKINSON, M.D.**

Watertown, N. Y.

**Recent Trends in Cataract Surgery**

A new era in cataract surgery began following the development of complete anesthesia accompanied by hypotony with the eye practically immobile. Improved technic assures an accurately placed section. Firm corneoscleral suturing is no longer hazardous. All has led to the long sought for ideal—a safe intracapsular extraction with round pupil.

**COURSE 104**

Room 705 Periods M-4, T-4

**CHARLES A. BAHN, M.D.**

New Orleans, La.

**Vitamins, Hormones and Enzymes in Ophthalmology**

The etiology and treatment of known ocular diseases caused or aggravated by vitamin, hormone, and enzyme abnormalities will be discussed and illustrated.

**COURSE 105**

Room 705 Periods M-5, T-5

**JAMES MASON BAIRD, M.D.**

Atlanta, Ga.

**Vitamins and Other Adjuncts to Diet**

1. Discussion of the clinical manifestations of vitamin A deficiency: i.e., xerosis, keratomalacia, xerophthalmia and nyctalopia.
2. Ariboflavinosis as manifest by glossitis, chelitis and keratitis.
3. Thiamin chloride in toxic amblyopia and vitamin D as an adjunct in calcium deficiencies.
4. Discussion of cholesteremia as manifest in xanthoma and hyperprotemenia in nephritis and arteriosclerosis.

This lecture will be illustrated with lantern slides and diagrams.

**COURSE 106**

Room 711 Periods M-4, T-4

**GEORGE S. BAKER, M.D.**

Rochester, Minn.

BY INVITATION

**The Differential Diagnosis and Treatment of Neuralgias of the Face**

This course will include a discussion on pains of ophthalmic origin.

**COURSE 107**

Room 710 Periods M-4, T-4

**OTTO BARKAN, M.D.**

San Francisco, Calif.

**Goniotomy for Congenital Glaucoma: Importance of Early Diagnosis and Early Adequate Surgical Intervention**

Stripping the chamber angle of embryonic tissue results in permanent normalization of the intraocular pressure. Goniotomy is performed by direct vision under a surgical contact glass. If cloudiness of the cornea prevents use of the glass, the operation is performed without it. The principal and alternative technics are given. Urgent need of early diagnosis and operation is stressed.

**COURSE 108**

Room 710 Periods M-5, T-5

**S. JUDD BEACH, M.D.**

Portland, Maine

**The Therapeutic Value of Refraction**

Patients seek relief from local complaints such as impaired vision, red eyes, discharge, scratching, pain, asthenopia, diplopia, and the like; as well as general disturbances as anorexia, vertigo, drowsiness, fatigue, headache, and inability to concentrate. Differential points and choice of lenses for suitable cases will be discussed.

**COURSE 109**

Room 705 Periods W-6, Th-6

**JOHN G. BELLOWS, M.D.**

Chicago, Ill.

**Etiology and Type of Lens Changes**

Senile cataract comprises 60-90 per cent of all types of lens opacities. Cataract is

particularly common after the sixtieth year. It is difficult to point to any single fundamental etiologic factor. Theories may be divided into two main groups: those based upon physical or chemical derangements, and those based upon senile involutional and hereditary factors.

**COURSE 110**

Room 705 Periods W-4, Th-4

**RAYNOLD N. BERKE, M.D.**
Hackensack, N. J.

**Exophthalmos**

Exophthalmos is the most common symptom of disease of the orbit. It may be an expression of disease of the globe, the extraocular muscles, the retrobulbar tissues, the orbital walls or some systemic malady. The clinical differential diagnosis of exophthalmos is one of the most difficult problems in ophthalmology.

**COURSE 111**

Room 706 Periods M-6, T-6

**MILTON BERLINER, M.D.**
New York, N. Y.

**Slit Lamp Microscopy**

This course is designed to consider the basic technics of biomicroscopy as they are applied to ordinary daily diagnostic procedures. The importance of this method of examination in the study of cases preoperatively is especially stressed. Many clinical entities will be considered.

**COURSE 112**

Room 711 Periods M-5, T-5

**JOHN A. BILLINGSLEY, M.D.**
Kansas City, Kan.

**Headaches and Head Pains of Ocular Origin**

Anatomy, physiology, and pathology of the neurovascular system involving the eye, orbit, external (soft tissue) structures of the face and skull, and internal structures of the skull having pain sensation.

The correlation of psychology with this type of pain, especially "headaches."

Refractive errors, muscle imbalance, binocular single vision, monocular vision with only one seeing eye, or alternating.

Inflammations of the uveal tract, acute and chronic glaucoma, etc.

Diagnosis; importance of history.

**COURSE 113**

Room 710 Periods W-4, Th-4

**PAUL BOEDER, Ph.D.**
Southbridge, Mass.

BY INVITATION

**Aniseikonia**

Aniseikonia is defined and its causes, symptoms, and incidence are discussed. The Eikonometer is described, and methods of measurement outlined and demonstrated. Iseikonic lenses are explained, and the results of surveys given. The effect of aniseikonia on binocular space perception is discussed and demonstrated with the new space Eikonometer target.

**COURSE 114**

Room 706 Periods W-4, Th-4

**DONALD BOGART, M.D.**
New York, N. Y.

**Eye Shortening Procedures**

**COURSE 115**

Room 705 Periods W-5, Th-5

**TRUMAN L. BOYES, M.D.**
New York, N. Y.

**Questions and Answers Arising in Surgical Cases**

Everyone has his own practical clinical problems. This course is designed to give helpful information and advice in the solution of these problems. Every member of the group may submit one prepared question in writing on entrance to the course and an answer will be given with celerity and definiteness. In case of duplication an alternate question may be submitted. The whole program is extemporaneous.

**COURSE 116**

Room 711 Periods M-6, T-6

**ALBERT L. BROWN, M.D.**
Cincinnati, Ohio

**Focal Infection — Investigation and Treatment**

The mechanism of focal infection is not clear. It would seem that a focus can cause a remote inflammation by releasing organisms in the blood stream for transmission to the eye, by the liberation of dead products or endotoxins, or by the transmission of protein products to the eye, creating a local sensitization which can be activated by the same circulating products later.

**COURSE 117**

Room 716 Periods M-4, T-4

**A. B. BRUNER, M.D.**
Cleveland, Ohio

**Visual Aids**

Various types of visual aids that are being used and the possibilities of helping the patient with other means than glasses will be discussed.

**COURSE 118**

Room 716 Periods W-6, Th-6

**EDWARD P. BURCH, M.D.**

St. Paul, Minn.

**Anesthesia in Ophthalmology**

This course consists of a review of the various local anesthetic agents, indications for their use, technic of administration, and a discussion of their employment in a number of commonly performed opthalmic operations.

**COURSE 119**

Room 724 Periods M-4, T-4

**FRANK E. BURCH, M.D.**

St. Paul, Minn.

**Glaucoma — Choice of Surgery**

Procedures favored by the author in the various manifestations of glaucoma will be described. The objectives desired in sclerotomy, sclerectomy, iris inclusions, cyclodialysis, cyclodiathermy, opticociliary neurectomy, and trabeculotomy will be considered. Operations for relief of tension in glaucoma caused by intumescent lens, venous thrombosis, iridocyclitis, and when cataract and other conditions complicate glaucoma will be discussed.

**COURSE 120**

Room 711 Periods W-4, Th-4

**FRANK D. CARROLL, M.D.**

New York, N. Y.

**Optic Neuritis**

This lecture will consider all the common and most of the rare diseases of the optic nerve. These include bacterial and virus inflammations, vascular disease, multiple sclerosis, degenerative conditions, nutritional deficiency states, hereditary optic atrophy, and the toxic amblyopias. Differential diagnosis and recent advances in treatment will be discussed.

**COURSE 121**

Room 716 Periods M-5, T-5

**VIRGIL G. CASTEN, M.D.**

Boston, Mass.

**Practical Perimetry**

Visual field taking is a subjective examination and much depends on the cooperation of the patient, the training of the perimetrist, knowledge of the anatomy of the visual pathways and of the lesions which injure it. This discussion will be limited to: 1. Essentials of technic, 2. Instrumental methods of study, 3. Brief review of the visual pathways, and 4. Color field and test objects. Specific types of pathologic fields will be shown.

**COURSE 122**

Room 722 Period M-4

**RAMON CASTROVIEJO, M.D.**

New York, N. Y.

*Surgical Treatment of Lesions of the Cornea*

**Treatment of Corneal Injuries**

a. Nonpenetrating wounds.
b. Penetrating wounds.
c. Foreign bodies.
d. Burns.
e. Corneal ulcers.

**COURSE 123**

Room 722 Period T-4

**RAMON CASTROVIEJO, M.D.**

New York, N. Y.

*Surgical Treatment of Lesions of the Cornea*

**Conjunctival Keratoplasty Including Operations for Extensive Pterygium, Recurrent Pterygium, Pseudopterygium and Symblepharon.**

a. Review of all surgical procedures used up-to-date for the treatment of conical cornea with the exception of keratoplasty.
b. Review of surgical treatment for the treatment of staphyloma of the cornea.

**COURSE 124**

Room 722 Period W-4

**RAMON CASTROVIEJO, M.D.**

New York, N. Y.

*Surgical Treatment of Lesions of the Cornea*

**Corneal Tattooing and Keratectomies**

a. Tattooing of the cornea with India ink, gold, and platinum chloride.
b. Indications, surgical procedures and postoperative treatment of keratectomies, including irradiation both by x-ray and radium to prevent recurrence of corneal vascularization.

**COURSE 125**

Room 722 Period Th-4

**RAMON CASTROVIEJO, M.D.**

New York, N. Y.

*Surgical Treatment of Lesions of the Cornea*

**Keratoplasty**

a. Short review of literature on keratoplasty.
b. Indications.
c. Description of technics.
d. Postoperative care.
e. Treatment of complications.

**COURSE 126**

Room 711 Periods W-5, Th-5

**C. P. CLARK, M.D.**

Indianapolis, Ind.

**Preparation of the Patient for Cataract Surgery**

Discussion will be made of preoperative steps to lessen or control anticipated surgical and postsurgical complications.

**COURSE 127**

Room 722 Periods W-5, Th-5

**WILLIAM H. CRISP, M.D.**

Denver, Colo.

**Testing Astigmatism With Dials and Cross Cylinders**

Subjective measurement of refraction, when practicable, is more precise and more reliable than objective testing; but subjective testing should be given, as nearly as possible, an objective quality. As to spherical correction, the possibility of error in testing is satisfactorily excluded by painstaking use of the fogging method. The fogging method is often misunderstood and wrongly applied. Bilateral fogging is unknown to many workers but is of great value.

As regards astigmatism, while other tests have their value, the two most reliable subjective lines of procedure are critical use of the best forms of astigmatic dial, on the one hand, and, on the other hand, the cross cylinder tests for amount and axis of astigmatism. The astigmatic dials recommended are those designed by Verhoeff and by Lancaster and Regan.

**COURSE 128**

Room 706 Periods W-6, Th-6

**BEULAH CUSHMAN, M.D.**

Chicago, Ill.

**Endocrine Disorders in Relation to the Eyes**

The extremely sensitive eyes often present symptoms in any early endocrine disorder. The eye findings in thyrotoxicosis as differentiated from the thyrotropic disturbances will be taken up as well as hypothyroidism, the asthenopic symptoms related to deficiency in ovarian and anterior pituitary products, and the parathyroid and its relation to the metabolism of the eye.

**COURSE 129**

Room 712 Periods M-6, T-6

**NORMAN L. CUTLER, M.D.**

Wilmington, Del.

**Reconstruction of the Orbit**

The various materials used in reconstruction around the orbit and their value will be discussed. The selection of operative procedures to be used, planning the surgical steps, anesthesia, complications, and treatment will also be discussed.

**COURSE 130**

Room 712 Periods W-4, Th-4

**RALPH W. DANIELSON, M.D.**

Denver, Colo.

**Differential Diagnosis of Retinoblastoma from Metastic Ophthalmia, Retrolental Fibroplasia, and Other Conditions Simulating Retinoblastoma**

This conference will aim to aid in clinical differentiation of retinoblastoma (glioma) and many other conditions simulating a tumor. The most common pseudogliomas are metastatic ophthalmia, proliferative retinitis, retrolental fibroplasia (Terry), and other congenital anomalies. Armamentarium and methods of examination will be discussed.

**COURSE 131**

Room 706 Periods W-5, Th-5

**WINDSOR S. DAVIES, M.D.**

Detroit, Mich.

**Optic Discs — Comparison of Clinical and Pathologic Changes**

This course shall consist of the study of various lesions involving the optic nerve head. The pathologic changes occurring in these lesions will be considered and a correlation made between the clinical and pathologic findings. Appropriate treatment will be discussed.

**COURSE 132**

Room 712 Periods M-5, T-5

**CLIFFORD A. DICKEY, M.D.**

San Francisco, Calif.

**Levator Paralysis — Causes and Treatment**

This course will be a discussion of:

1. Anatomy of levator and relationship to Muller's muscle; also central nervous system pathways.
2. Etiology which will include peripheral and central lesions.
3. Various types of surgical procedures along with their indications.

**COURSE 133**

Room 710 Period W-5

**HAROLD FALLS, M.D.**

Ann Arbor, Mich.

**Embryology**

A didactic but concise outline of the embryological development of the human eye. The material will be presented so as

to make possible a background for adequate clinical interpretations of ocular aberrations of development. The differential diagnosis of developmental and diseased conditions of the eye will be approached. Special consideration will be given to the embryology of the anterior ocular segment and its aberrations.

**COURSE 134**

Room 710 Period W-6
Room 724 Period Th-6

**WALTER H. FINK, M.D.**
Minneapolis, Minn.

**Anatomical Study of the Oblique Muscles**

The various anatomical features of the oblique muscles are presented with special emphasis on the clinical application. The presentation consists of a series of anatomical specimens, slides, and charts which serve to demonstrate the normal. Various anomalies are also presented.

**COURSE 135**

Room 724 Periods M-5, T-5

**WATSON GAILEY, M.D.**
Bloomington, Ill.

**Complications and Treatment in Cataract Surgery**

The lecturer will discuss the following:

1. Means which may be employed and which will aid materially in the prevention of complications occurring both during and following cataract extraction.
2. Complications which arise during cataract surgery and his methods of handling them.
3. Postoperative complications and their management.

**COURSE 136**

Room 724 Periods M-6, T-6

**J. CONRAD GEMEROY, M.D.**
Detroit, Mich.

**External Diseases of the Eye**

This lecture on external eye diseases is illustrated in color and shows usual and some unusual diseases of the lids, conjunctiva, and cornea with discussion of their diagnosis and treatment. The lecture will be illustrated throughout with Kodachrome slides.

**COURSE 137**

Room 724 Period W-6

**J. CONRAD GEMEROY, M.D.**
Detroit, Mich.

**Anterior Segment Photography of the Eye**

This is a discussion of the method the author uses to take colored photographs of the anterior segment of the eye. The camera used, how almost any camera can be adapted to do the same; the lighting, exposure time, etc. The easy slip from single pictures to stereoscopic pictures in color. Many pictures in color will be used to show reasons for failures versus good pictures.

**COURSE 138**

Room 716 Periods M-6, T-6

**GLEN G. GIBSON, M.D.**
Philadelphia, Pa.

**End Results in Retinal Detachment**

The various ways of evaluating end results in retinal detachment will be discussed. The importance of the general health of the eyeball with particular reference to the status of the choroid and its circulation will be considered. It is felt that present criteria for surgical success are too idealistic, and an effort will be made to point out the value of partially successful cases.

**COURSE 139**

Room 712 Periods W-5, Th-5

**HAROLD GIFFORD, M.D.**
Omaha, Nebr.

**Dacryocystorhinostomy**

This course will present a review of the common surgical technics used in tear sac surgery. The surgical anatomy of the region will be covered. The technic of transplantation of the tear sac will be described in detail with slides and moving pictures.

**COURSE 140**

Room 712 Periods W-6, Th-6

**D. F. GILLETTE, M.D.**
Syracuse, N. Y.

**Uveitis — Chronic**

The course will consider the etiology, pathology, clinical course and prognosis of the various types of chronic uveitis. Special consideration will be given to the differential diagnosis and treatment of disease and its complications.

**COURSE 141**

Room 728 Periods M-4, T-4

**J. F. GIPNER, M.D.**
Rochester, N. Y.

**Recent Advances in Ophthalmology**

Scleral resection for retinal detachment. Ruedemann and Cutler basket-type implants after enucleation. Sulfhydryl in treatment of burns of the eye. D.F.P. in

the treatment of glaucoma. Retrolental fibroplasia, persistent primary vitreous, persistent tunica vasculosa lentis. Congenital cataract in children born of mothers who contracted measles during the first three months of pregnancy. Hemorrhage into the vitreous masking retinal detachment. New uses for x-ray in ophthalmology.

**COURSE 142**

Room 726 Periods M-6, T-6

**JAMES N. GREEAR, JR., M.D.**
Washington, D.C.

**Rehabilitation of the Blind**

1. Application of knowledge gained in rehabilitation of the war blinded applied to the newly blinded civilian.
2. Methods and technics employed in the early phases of blindness.
3. Social adjustment, prevocational and vocational training of the blind.
4. The responsibility of the ophthalmologist toward instituting a workable plan of rehabilitation for newly blinded patients who come under his care.

**COURSE 143**

Room 728 Periods W-5, Th-5

**MILLARD E. GUMP, M.D.**
Oakland, Calif.

**Iridocyclitis — Diagnosis and Treatment**

A resume of findings, gross and slit lamp. A routine for study of possible conditions of which iridocyclitis is an associated condition. Discussion of local and systemic therapy and treatment of complications and evaluation of foci studies.

**COURSE 144**

Room 728 Periods W-4, Th-4

**F. HERBERT HAESSLER, M.D.**
Milwaukee, Wis.

**Diagnosis and Treatment of Divergence Excess or Convergence Insufficiency**

Convergence and divergence are units of activity which consist of integrated complexes of simpler neuromuscular actions. Our knowledge and its limitations of such supranuclear functions will be described as a basis for judgment of disturbances of binocular coordination. Treatment, such as it is, will be mentioned.

**COURSE 145**

Room 726 Periods M-5, T-5

**HENRY L. HILGARTNER, M.D.**
Austin, Texas

**Keratoplasty and Other Procedures for the Treatment of Corneal Opacities**

Keratoplasty and the treatment of corneal opacities will be discussed from many angles. Stress will be given to importance of selection of proper cases for surgery. Different surgical procedures now used will be discussed, as well as treatment of cases unfavorable to keratoplasty by other means; i.e., partial resection of the cornea, x-ray, and radium.

**COURSE 146**

Room 726 Periods M-4, T-4

**JOHN B. HITZ, M.D.**
Milwaukee, Wis.

**Problem of Visual Failures in School Children — Diagnostic Methods and Treatment**

1. Conditions which can be attributed to prolonged close work in children.
2. Causes of myopia. Review.
3. Diagnosis of early difficulties by surveys in schools. Methods.
4. Present accepted methods of treatment. A critique.
5. Suggested further studies.

**COURSE 147**

Room 711 Periods W-6, Th-6

**L. G. HOFFMAN, M.D.**
Chicago, Ill.

**Acute Infectious Lesions of the Lid — Differential Diagnosis and Treatment**

1. Anatomy and physiology — external anatomy (muscles, tarsus, glands, vessels, and nerves) and movements.
2. Skin inflammations—chemical, herpes, allergy, eczema, edema.
3. Hordeolum—surgery, manganese.
4. Chalazion—diathermy, surgery (external or internal excision).
5. Blepharitis marginalis—sicca, ulcerosa.
6. Diseases of palpebral muscles—ptosis, blepharospasm.
7. Anomalies of position—ectropion, entropion, symblepharon.
8. Injuries.
9. Congenital anomalies—coloboma, epicanthus.
10. Tumors — xanthelasma, carcinoma, etc.

**COURSE 148**

Room 724 Period W-4

**WENDELL L. HUGHES, M.D.**
Hempstead, N. Y.

**Photography — Motion Pictures**

A brief outline of some of the apparatus

needed to take satisfactory pictures of conditions and procedures in ophthalmology.

A discussion of methods of titling will also be conducted.

Properly taken motion pictures are very valuable:

a. In teaching:

1. Undergraduates and graduates new technics and classical technics. The details of operative technic can be shown in full to as large a group as desired in just as complete a manner as might be given to one person standing directly at the side of the operator.

The film can be shown repeatedly to review all the details. The clinical appearance or unusual lesions can also be shown in such a way as to impress the details upon those viewing the picture.

---

**COURSE 149**

Room 731 Periods M-4, T-4

**WENDELL L. HUGHES, M.D.**

Hempstead, N. Y.

**Surgical Repair of the Lids**

Lacerations of the lids of various types will be discussed as to their frequency and particularly their surgical repair. The lecture will be illustrated by slides and motion pictures.

---

**COURSE 150**

Room 727 Periods M-6, T-6

**S. RODMAN IRVINE, M.D.**

Beverly Hills, Calif.

**Manifest Refraction**

Routine method of doing manifest refraction. Advantage and disadvantage of fogging; quick check for astigmatism with cross cylinder; diagram of conoid of Sturm showing position of retina in hyperopic, mixed, and myopic astigmatism; use of astigmatic dials; special procedures in cases with low visual acuity; correction of high errors, aphakic eye, and high myopes. Reproducibility of refraction.

---

**COURSE 151**

Room 732 Periods M-4, T-4

**FRED JOBE, B.S.**

Rochester, N. Y.

BY INVITATION

**Practical Physiological Optics**

The function of an optical system, whether it is a spectacle lens or a rangefinder, consists of optically moving a physical object into a position where the object can be used. In refraction, this position is the far point of the eye. The general laws governing the performance of a lens or prism are readily understandable. A knowledge of these principles is extremely valuable in the practice of refraction.

These general laws will be discussed in relation to refraction, effective power, transposition of formulae, spherical equivalents, cross cylinders and Sturm's interval, as well as some ophthalmic instruments such as refractors, duochrome tests and keratometers.

---

**COURSE 152**

Room 726 Periods W-6, Th-6

**LORAND V. JOHNSON, M.D.**

Cleveland, Ohio

**Pigmented Lesions of the Choroid**

The influence of the pigment epithelium and other fundamental considerations concerning pigmented lesions of the choroid. Especial reference will be given to toxoplasmosis and its differential diagnosis and laboratory identification, the influence of additional pregnancies to the mother and to later children.

Kodachrome slides of toxoplasma choroiditis.

---

**COURSE 153**

Room 727 Periods W-4, Th-4

**J. H. JUDD, M.D.**

Omaha, Nebr.

**Differential Diagnosis and Treatment of Optic Neuritis**

All types of neuritis will be discussed with special emphasis on the correlation of the signs and symptoms with the histopathology. A practical method of differentiation of the clinical entities will be outlined. The most recent methods of treatment for each will be presented.

---

**COURSE 154**

Room 728 Periods W-6, Th-6

**DEWEY KATZ, M.D.**

Hartford, Conn.

**Interpretation of Field Defects as an Aid in the Localization of Cerebral Pathology**

Field defects when properly analyzed will only indicate, and that with a remarkable degree of accuracy, the area in the visual pathway which has become involved by the pathologic process present. In and of themselves field defects result-

ing from intracranial pathology usually give no indication as to the origin or nature of the lesion present.

When the involved area in the visual pathway is known and to this is added other ophthalmologic factors such as the rate of development of the field defects, the appearance of the nerve heads and the reaction of the pupils, the nature of the pathologic process can be definitely diagnosed in some instances.

With an accurate knowledge of the components of the visual pathway and their course from the retina to the occipital cortex it becomes relatively simple to analyze the various types of field defects which are typical of the segment of the visual pathway involved.

The field defects resulting from intracranial pathology fall into one of five main categories depending on the site of involvement of the visual pathway. The delimiting zones are fairly clear cut anatomically.

---

**COURSE 155**

Room 724 Periods W-5, Th-5

**JOHN E. L. KEYES, M.D.**
Shaker Heights, Ohio

**Interpretation of Experimental and Clinical Retinal Arteriovascular Disease**

The object of this study is to correlate the clinical ophthalmoscopic picture of retinal vascular disease with the associated pathologic process. An ophthalmologist who can visualize in anatomical terms the extent of local vascular disease in an eye is better qualified to make a prognosis.

Most of the visible arterial vascular tree of the retina is arteriolar. Arteriolar disease is segmental. An arteriole does not develop disease of its wall in a uniform manner. Serial sections of a diseased vessel often show extensive damage in areas of close propinquity to a zone of minor pathologic changes.

Such a vessel viewed through an ophthalmoscope will present considerable variation in the intensity and width of its central light reflex in the presence of early disease. At a later stage, definite segmental narrowing of the arteriole can be noted. The lumen of such vessels is narrowed locally and the volume of the blood carried is reduced accordingly. Arteriolar disease of this type does not resolve, but is progressive.

The appearance of a retinal artery in a state of spasm is well known. The narrowing of the affected vessel may be general or localized. One vessel may be involved or the whole arterial tree show varying degrees of involvement. The spasm may be transitory or permanent and pass over into sclerosis. Many vessels with transitory spasm appear normal after the attack has passed. Repeated observation of such a spastic vessel will ultimately show evidence of segmented disease. The localized spasm and edema of an artery seen in toxemia of pregnancy may resolve completely if the patient is not a hypertensive person. Vessels with general permanent spasm become sclerosed.

Both arteries and veins bordered by parallel white streaks are seen in retinal vascular disease. Such a vessel frequently develops a white sheath or covering. These are not silver wire vessels but examples of perivasculitis. A silver wire vessel has extreme sclerosis of its wall, is smaller than it was originally, and the light reflex is keen. A vessel with a white sheath appears wider than it was originally and the usual central light reflex is soft, partly obscured or replaced by a soft white patchy coating. Microscopically the changes are mostly in the wall of the white wire vessel. The vessel lumen is reduced to a minimum or closed; adventitial proliferation and edema predominate in sheathed vessels; medial and intimal disease is usually present also, but most of these vessels are patent and carry blood.

The obscuring of an underlying vein at an arteriovenous crossing is secondary to loss of transparency of the overlying artery and chiefly due to proliferation of the adventitial tissue binding the two vessels together. Further progress of this process pulls the artery with greater blood pressure against the vein with lower blood pressure. This mechanism alone will cause indentation of the vein. Thickening of the media and intima of an artery is usually at the expense of the vessel's lumen, but will add to the extent of disturbance at an arteriovenous crossing. Obscuring of the vein at crossings is often physiological and due to excessive connective tissue. The sheathing seen on some vessels, on and slightly beyond the disc in healthy eyes, is congenital.

It is not within the scope of this communication to discuss retinopathies associated with disease of the retinal blood vessels.

**COURSE 156**

Room 796 Period M-4

**DANIEL B. KIRBY, M.D.**
New York, N. Y.

**Types of Surgical Procedures for Cataract Surgery**

Discussion of whole lens in congenital cataract and subluxation. Linear extraction for soft cataract. Capsulotomy extraction. Intracapsular extraction with loop, suction cup, eresiphake, forceps. Various maneuvers used in each. Pressure, traction, rotation and combinations of each. Direct rupture of zonule. Delivery of cataract.

**COURSE 157**

Room 727 Periods W-5, Th-5

**WILLIS S. KNIGHTON, M.D.**
New York, N. Y.

**Diagnosis and Analysis of Glaucoma Cases**

Classification and pathology. Secondary glaucoma a result of other conditions. Acute primary glaucoma differs from chronic primary glaucoma in pathology and treatment. Glaucoma simplex requires painstaking analysis and checking. Early diagnosis important but difficult.

**COURSE 158**

Room 724 Period Th-4

**FERDINAND L. P. KOCH, M.D.**
New York, N. Y.

**Fundus Photography**

Photographic color delineation of the ocular fundi in health and in disease remains a valuable adjunct to the ophthalmologist whether such data is for clinical records, medicolegal teaching, or research purposes. Technic of color film transparencies and ophthalmoscopic interpretations will be stressed and illustrated with Kodachrome projection slides.

**COURSE 159**

Room 713 Periods M-4, T-4

**IVAN J. KOENIG, M.D.**
Buffalo, N. Y.

**Ocular Tuberculosis**

Ocular tuberculosis and conditions simulating tuberculosis. Underlying pathology will be discussed and illustrated in a few lesions. Differential diagnosis will be emphasized and difficulties encountered in making conclusive diagnosis in such conditions. Consideration of primary and secondary tubercular lesions. Local and general treatment to be outlined and discussed.

**COURSE 160**

Room 713 Periods W-4, Th-4

**ARLINGTON C. KRAUSE, M.D.**
**Chicago, Ill.**

**Biochemistry of the Eye**

The biochemistry of the eye is concerned with the normal and pathologic chemical structures and chemical processes of the various ocular tissues. The most important subjects in ocular chemistry are the conjunctiva, nutrition of the cornea, nature of the aqueous and vitreous humor, chemical processes in the formation of cataract, and the chemistry of vision.

**COURSE 161**

Room 713 Periods W-6, Th-6

**JAMES E. LEBENSOHN, M.D.**
Chicago, Ill.

**Physiology of Vision**

Recent advances in the study of visual sensation, form discrimination, color perception, and psychic interpretation will be discussed and their practical implications emphasized. Personal problems are especially invited, and if submitted by mail in advance will be given detailed attention.

**COURSE 162**

Room 713 Periods M-5, T-5

**LOUIS LEHRFELD, M.D.**
Philadelphia, Pa.

**Ocular Manifestations of Allergy**

One or all tissues of the eye may be involved in an allergic process. The sulfonamides, penicillin, and other antibiotics have been added to the many contactants which are responsible for ocular allergies. There will be included: the role of inhalants, ingestants, contactants, injectants, bacteria (tuberculosis), fungi, physical agents (light), nonspecific agents, and ophthalmic migraine.

**COURSE 163**

Room 713 Periods M-6, T-6

**ALBERT LEMOINE, M.D.**
Kansas City, Mo.

**Vitreous Opacities**

The discussion will be on the nature of vitreous opacities and sources of origin. Etiology: tuberculosis, lues, foci of infection, toxemias, hemorrhages, and others. Treatment: Removal of foci of infection, treat lues with vaccines, hygienic measures.

**COURSE 164**

Room 713 Periods W-5, Th-5

**CECIL LEPARD, M.D.**

Detroit, Mich.

**Juvenile Cataract — Treatment: Soft Cataract — Treatment**

Classification of types of juvenile cataract. Hereditary factors and the relation to other anomalies. Details of surgical procedures. Complications following surgery. Postoperative management. Procedures in the treatment of soft cataract.

**COURSE 165**

Room 734 Periods W-6, Th-6

**AUREL E. MANGOLD**

New York, N. Y.

BY INVITATION

**The Optician's Part in the Fitting of Glasses**

1. Prescription analysis to give the patient the maximum of comfort and efficiency.
2. Methods of measuring interpupillary distance for near and far.
3. Vertical imbalance and its correction.
4. Influence of vertex distance on lens power.
5. Lens form and its effect on retinal image.
6. Types of bifocals and proper selection in cases of hyperopia and myopia.
7. Colored lenses and their uses.
8. Suggested office procedure for the examination of a finished pair of glasses.
9. Errors commonly made in the dispensing and processing of a pair of glasses.

**COURSE 166**

Room 732 Periods W-6, Th-6

**WILLIAM A. MANN, M.D.**

Chicago, Ill.

**Refraction of the Cataract Patient**

Problems of refraction of the patient with early cataract. Correction of aphakia. Methods of objective and subjective testing. Precautions in ordering lenses. Analysis of unsatisfactory visual results with finished glasses. Types of cataract lenses. Methods of securing single binocular vision in bilateral and monocular aphakia. Catmin lenses, contact lenses.

**COURSE 167**

Room 733 Periods W-4, Th-4

**DON MARSHALL, M.D.**

Kalamazoo, Mich.

**Differential Diagnosis and Treatment of Chronic Infections of the Lid**

This lecture reviews the diagnosis and therapy of eyelid infections which generally are or become chronic. A few acute or questionably infectious conditions are considered briefly, but conjunctivitis is omitted except for special types. Principal topics will be blepharitis, chalazion, and meibomian infection, fungus infections, molluscum, wart, and parasites.

**COURSE 168**

Room 733 Periods M-4, T-4

**A. W. McALESTER, III, M.D.**

Kansas City, Mo.

**Pigmentary Retinal Changes — Etiology and Treatment**

Pigmentary retinal changes are divided into primary pigmentary degeneration, typical of which is retinitis pigmentosa. Other forms and atypical types will be discussed; the secondary pigmentary changes associated with acquired retinitis such as luetic and traumatic will be dealt with. Etiology of both types and experimental work as to etiologic factors will be presented. New forms of therapy, such as tissue therapy, also chemical and physical therapy, will be dealt with in detail.

**COURSE 169**

Room 732 Periods M-5, T-5

**MURRAY F. McCASLIN, M.D.**

Pittsburgh, Pa.

**Refraction of Children's Eyes**

Indications for refractions. Muscle imbalances. Symptoms of errors of refraction. Marginal lid infections. Technic of preliminary examination. Choice of cycloplegia. Technic of retinoscopy. Cycloplegic examinations. The interval of rechecking various classifications of patients.

**COURSE 170**

Room 734 Periods W-5, Th-5

**P. ROBB McDONALD, M.D.**

Philadelphia, Pa.

**Tinted Lenses — Their Value in Ophthalmology**

The physiologic and pathologic effects of radiant energy on the normal and aphakic eye will be considered in detail. Samples of the various types of tinted lenses now on the market will be exhibited. The optical properties of the various types of lenses will be discussed with particular reference to their clinical application.

**COURSE 171**

Room 734 Periods M-4, T-4

**RAYMOND E. MEEK, M.D.**

New York, N. Y.

**Surgical Anatomy of the Orbit**

The essence of good surgery of the eye

is a thorough knowledge of the anatomy of the orbit and its contents. A skilled eye surgeon does not fumble. He knows what he wants to accomplish and how. At all times he is familiar with the tissues and is never lost. Operative procedures are thus expedited, and infection and trauma are held to a minimum if the anatomy is familiar.

**COURSE 172**

Room 733 Periods W-5, Th-5

**W. HOWARD MORRISON, M.D.**

Omaha, Nebr.

**Disturbances of the Lacrimal Glands — Etiology and Treatment**

Embryology, physiology, and pathology of the lacrimal gland will be discussed. Differential diagnosis of various nonmalignant changes will be outlined. Particular emphasis will be placed on neoplastic changes of the gland; their diagnosis, treatment, and surgical approach.

**COURSE 173**

Room 733 Periods M-6, T-6

**M. PAUL MOTTO, M.D.**

Cleveland, Ohio

**Treatment of the Muscle Problem in the Free Clinic**

The routine followed in an eye muscle clinic includes a complete history; associated muscle movements; the deviation for distance and near; convergence near point; diplopia test, if indicated; refraction under cycloplegic; orthoptic training if necessary; review of use; prisms; indications; and favorable age to operate and results to be expected.

**COURSE 174**

Room 733 Periods W-6, Th-6

**GLEN W. NETHERCUT, M.D.**

Chicago, Ill.

**The Etiology and Treatment of Chorioretinitis**

The course will cover the etiology and treatment of the commoner forms of chorioretinitis, with some reference to the less frequently seen forms.

**COURSE 175**

Room 732 Periods M-6, T-6

**D. H. O'ROURKE, M.D.**

Denver, Colo.

**Refraction: Cycloplegic and Manifest**

The indications for cycloplegic and manifest refractions will be considered. The various age groups, as well as the character of the ametropia, influence the decision as to the use of a cycloplegic. Skiascopy (streak refractascope) and the technics of the Jackson crossed cylinder and Lancaster astigmatic dials will be reviewed.

**COURSE 176**

Room 734 Periods M-5, T-5

**R. TOWNLEY PATON, M.D.**

New York, N. Y.

**Corneal Surgery**

Classification of cases for operation, operative technic, postoperative complications, discussion of donor material, preservation and handling of donor material, results.

**COURSE 177**

Room 733 Periods M-5, T-5

**OLIVE PECK**

Cleveland, Ohio

BY INVITATION

**The Value of Sight-Saving Classes in the Education of Poor-Sighted**

A discussion of the education of the school child with visual difficulties. A review of the types of cases referred; methods of referral; cooperative plan of education; financial support; transportation; tuition and educational, social, and vocational results. Need for the cooperation of ophthalmologists and educators in understanding the needs of the child. Illustrated.

**COURSE 178**

Room 734 Periods W-4, Th-4

**CHARLES PERERA, M.D.**

New York, N. Y.

**End Results in Retinal Detachment**

Untreated retinal detachment results in degeneration of the retina, often followed by uveitis and secondary glaucoma. Successful treatment depends upon a knowledge of the pathologic factors involved. These factors are presented so that the ophthalmologist may recognize contraindications to operation, perfect his operative technic, and increase his percentage of cures.

**COURSE 179**

Room 777 Periods W-5, Th-5

**RAYMOND L. PFEIFFER, M.D.**

New York, N. Y.

**The Value of X-Ray Studies in Differential Diagnosis of Lesions of the Orbit**

The roentgen findings characteristic of many different conditions of the orbit will be discussed from lantern slides. In addi-

tion, the roentgen diagnosis of retinoblastoma, toxoplasmosis, enophthalmos, and other conditions affecting the eye and the position of the eye will be illustrated.

**COURSE 180**

Room 727 Periods W-6, Th-6

**BERNARD D. RAVDIN, M.D.**

Evansville, Ind.

**Psychiatric Problems in Ophthalmologic and Otolaryngologic Patients**

During my thirty years of practice of ophthalmology and otolaryngology, I have recognized many, many times in the care of patients who present themselves, the objective findings do not satisfactorily explain the severity of the symptoms in a given case or lack of improvement in the course of treatment. In the management of such cases, we must realize that we are dealing with a person as well as a disease. Emotional problems may and often do account for many of such situations. Between the small number of obviously psychotic persons whom a physician sees and the larger number of patients who are sick solely because of physical disease, are a vast number of sick people who do not have any definite bodily disease to account for their illness.

It is this type of patient I've been interested in for many years and whom I would like to call to your attention. In my experience with cases of psychosomatic problems, most of them present the following characteristics:

1. Initially, they all present a picture of more or less severe illness with a host of bizarre complaints.
2. Despite the most careful ophthalmologic or otolaryngic and medical study, no adequate physical basis can be found for their illness. Their symptoms are predominantly subjective.
3. They all show a favorable response once an accurate diagnosis has been made and proper psychotherapy has been instituted.

**COURSE 181**

Room 732 Periods W-5, Th-5

**JAMES J. REGAN, M.D.**

Boston, Mass.

**The Use of Astigmatic Dials in Subjective Refraction**

A lecture on the underlying principles involved in the use of astigmatic dials for measuring the amount and axis of astigmatism, followed by a clinical demonstration. Inasmuch as this subject has not gained sufficient prominence to be found in medical textbooks, it should be of interest to all.

**COURSE 182**

Room 722 Periods W-6, Th-6

**SHALER RICHARDSON, M.D.**

Jacksonville, Fla.

**Office Management**

A study has been made of ophthalmologists' offices in various sections of the country. There will be a discussion made of record keeping, both clinical and financial. Office arrangement will be discussed with emphasis on innovations that may make offices more comfortable and expedite the handling of patients.

**COURSE 183**

Room 776 Periods W-4, Th-4

**ROY O. RISER, M.D.**

Park Ridge, Ill.

**Fundus Lesions — Differential Diagnosis**

A presentation of Kodachrome fundus photographs exhibiting a variety of congenital and acquired conditions. Differential diagnosis will be stressed and the progress of certain interesting cases noted in a series of photographs.

**COURSE 184**

Room 732 Periods W-4, Th-4

**KENNETH L. ROPER, M.D.**

Chicago, Ill.

**Chemotherapy**

In the field of chemotherapy, during the past few years, the advances have been in the nature of consolidation rather than of spectacular new discoveries. New preparations that have some advantages over the well-tried favorites will be discussed. The value of penicillin and the scope, limitations, and dangers associated with the drugs of the sulfonamide group will be reviewed.

**COURSE 185**

Room 788 Periods W-4, Th-4

**RALPH O. RYCHENER, M.D.**

Memphis, Tenn.

**External Diseases of the Eye — Differential Diagnosis and Treatment**

A Kodachrome exposition of usual and unusual conditions of the lids, conjunctiva, and cornea, including the allergies, oculoglandular syndromes, granulomata, lacrimal affections, malposition of lid borders and ptosis.

One hundred fifty illustrations of various pathologic states with concise and

pertinent remarks regarding their mode of treatment.

---

**COURSE 186**

Room 734 Periods M-6, T-6

**HAROLD G. SCHEIE, M.D.**
Philadelphia, Pa.

**Injury to Iris and Ciliary Body**

The anatomy and physiology of the iris and ciliary body in relation to ocular injuries are briefly reviewed. A classification of injuries to the iris and ciliary body is attempted. Treatment, especially relating to the major complications; namely, lens injuries, sympathetic ophthalmia, traumatic iridocyclitis, secondary glaucoma, and hyphemia, is outlined.

---

**COURSE 187**

Room 736 Periods M-6, T-6

**FREDERICK O. SCHWARTZ, M.D.**
St. Louis, Mo.

**The Mechanics of Lens Extraction**

Mechanics in general. The incision: kind, size, position. Iris. Zonule: its state. Lens: firm or flabby, mature and immature changes. The nucleus: intra- and extracapsular extraction. Pressure: kinds and where and how to apply it. Factors involving successful extraction.

---

**COURSE 188**

Room 744 Periods M-4, T-4

**G. VICTOR SIMPSON, M.D.**
Washington, D.C.

**The Treatment of Myopia — Glasses and Medical Adjuncts**

The correction of myopia with glasses will be fully discussed in this course. The newer types of lenses available and the most satisfactory bifocals will be shown. The present status of the medical treatment by adrenalin, vitamins, and exercises will be reviewed.

---

**COURSE 189**

Room 744 Periods M-5, T-5

**MASON SMITH, M.D.**
Richmond, Va.

**Presbyopia**

The ocular changes with advancing age producing a diminished amplitude of accommodation will be considered together with the appropriate treatment of presbyopia. The individual's ocular demands as well as practical points including fogging, dynamic retinoscopy, prisms, and special lens types will be evaluated.

**COURSE 190**

Room 731 Period W-6

**ALBERT C. SNELL, M.D.**
Rochester, N. Y.

**Medicolegal Ophthalmology**

1. Expert witness — qualifications of, choice of status.
2. The hypothetical question.
3. A physician's responsibility.
4. Malpractice.
5. Evaluation of subnormal vision.
6. Examination of eyes.
7. Malingering.

---

**COURSE 191**

Room 744 Periods W-6, Th-6

**DANIEL SNYDACKER, M.D.**
Chicago, Ill.

**Treatment of the Vertical Phoria**

The discussion will include a brief outline of the etiology of hyperphorias. Treatment will be divided into three phases: the use of prisms, surgery, and orthoptics, and the avoidance of artificially induced hyperphoria.

---

**COURSE 192**

Room 752 Period M-4

**EDMUND B. SPAETH, M.D.**
Philadelphia, Pa.

**Plastic Surgery — Congenital Ptosis**

---

**COURSE 193**

Room 752 Period T-4

**EDMUND B. SPAETH, M.D.**
Philadelphia, Pa.

**Plastic Surgery — Acquired Ptosis**

---

**COURSE 194**

Room 752 Period W-4

**EDMUND B. SPAETH, M.D.**
Philadelphia, Pa.

**Plastic Surgery — Ectropion and Entropion**

---

**COURSE 195**

Room 752 Period Th-4

**EDMUND B. SPAETH, M.D.**
Philadelphia, Pa.

**Plastic Surgery — Scar Tissue Resections**

---

**COURSE 196**

Room 744 Periods W-5, Th-5

**SCOTT STERLING, A.B.**
Rochester, N. Y.

BY INVITATION

**Lens — Practical Optics**

In this course a review will be given of the significant optical characteristics of

the various types of single vision and bifocal lenses, with a brief discussion of the advantages and disadvantages of each. Emphasis will be placed upon the practical problem of establishing criteria by which the ophthalmologist may select suitable lens types for specific cases.

**COURSE 197**

Room 777 Periods W-4, Th-4

**GEORGE H. STINE, M.D.**

Colorado Springs, Colo.

**Retinal Detachment — Diagnosis and Location of Tears**

The theoretical aspects of retinal localization will be considered and practical application will be described and demonstrated. The various methods, such as the method of disc diameters, dioptric elevation, transillumination, and perimetric methods, especially that devised by the lecturer, will be described and illustrated. Recent advances in correcting for meridian as well as limbus distance shift will be discussed, all of which add to precision in practical application.

**COURSE 198**

Room 777 Periods M-4, T-4

**GILBERT C. STRUBLE, M.D.**

West Los Angeles, Calif.

**Intraocular Foreign Bodies**

Methods of localization and the surgical indications and procedures for the removal of magnetic foreign bodies from the eyeball will be presented. The posterior approach using the nonpenetrating technic with pin point localization will be shown in detail and illustrated with slides and 16 mm. movies.

**COURSE 199**

Room 776 Periods M-4, T-4

**KENNETH C. SWAN, M.D.**

Portland, Ore.

**The Action of Drugs on the Iris and Ciliary Body**

Action of autonomic drugs upon the iris and ciliary body will be discussed with particular reference to factors which determine their clinical application. Discoveries in the field of corneal permeability will be reviewed and new drugs evaluated in relation to anterior segment therapeutics.

**COURSE 200**

Room 744 Periods W-4, Th-4

**EDWIN F. TAIT, M.D.**

Norristown, Pa.

**Accommodation and Convergence Reflex Mechanisms**

Accommodation and convergence are considered in the terms of the stimuli which produce them as responses.

Most accommodation and binocular vision abnormalities are the result of deficiencies in these reflexes, and are thus subject to a clinically useful classification which can be utilized as a rational basis for corrective procedures.

**COURSE 201**

Room 776 Periods M-5, T-5

**ISAAC S. TASSMAN, M.D.**

Philadelphia, Pa.

**Chemotherapy**

A consideration of chemotherapy and the use of modern drugs employed with special reference to the sulfonamides and penicillin. The present knowledge of methods of administration and reactions, dosage, and concentrations. Relation of general condition of patient. Results obtained in various ocular diseases and infections. Comparison with use of other drugs and methods of treatment.

**COURSE 202**

Room 776 Periods M-6, T-6

**EDWARD R. THOMAS, M.D.**

Dayton, Ohio

**The Diagnosis, Complications and Treatment of Diabetes and Blood Dyscrasias in the Lesions of the Eye**

The diagnosis and treatment of ocular complications of diabetes mellitus. Consideration of general systemic pathologic changes of diabetes and the correlation of changes found in the ocular structures. Discussion of carbohydrate metabolism, glycosuria, hyperglycemia, lipoid metabolism, and association of hypertension and arteriosclerosis. Ocular complications in the lids, cornea, iris, ciliary body, lens, vitreous, and retina, and their treatment.

**COURSE 203**

Room 738 Periods M-6, T-6

**ARNO E. TOWN, M.D.**

New York, N. Y.

**Difficulties With Contact Lens Fittings**

The requisites for a good contact glass are given, and a description of the various technics used. The difficulties which arise in contact glass fitting are enumerated and methods for avoiding improperly fitted contact glasses are given.

**COURSE 204**

Room 744 Periods M-6, T-6

**C. DWIGHT TOWNES, M.D.**

Louisville, Ky.

**Hyperphoria**

The various types of tests for hyperphoria, methods of treating with prisms, and surgical intervention, when necessary, will be discussed.

---

**COURSE 205**

Room 731 Periods W-4, Th-4

**C. A. VEASEY, JR., M.D.**

Spokane, Wash.

**Refraction — Practical Points**

Classification of causes of dissatisfied refraction cases. Routine procedure to detect sources of discomfort and dissatisfaction. Corrective procedures to relieve same. Practical points to avoid same. Unique problems.

---

**COURSE 206**

Room 777 Periods M-5, T-5

**C. W. WEISSER, M.D.**

Pittsburgh, Pa.

**Differential Diagnosis and Treatment of Iritis**

Etiologic factors. Gross characteristics of iritis and comparison with similar diseases. Important office procedures and differential examinations with emphasis on slit lamp studies. Laboratory studies indicated. Consultations suggested. Time honored therapy. Newer treatment endeavors, including chemotherapy and antibiotics. Results of therapy. Complications and sequelae.

---

**COURSE 207**

Room 738 Periods W-6, Th-6

**HAROLD F. WHALMAN, M.D.**

Los Angeles, Calif.

**Traumatic Lesions of the Cornea**

Lesions caused by solid foreign bodies will be considered. These will be grouped as superficial, penetrating and perforating wounds, simple and complicated. Since complications present the most serious problems, emphasis will be placed there.

---

**COURSE 208**

Room 736 Periods W-4, Th-4

**ELMER L. WHITNEY, M.D.**

Detroit, Mich.

**Lacrimal Apparatus**

Management of traumatic lacerations of canaliculus. Reconstruction of a canaliculus. Surgery of lacrimal sac. The cystorhinostomies.

**COURSE 209**

Room 731 Periods W-5, Th-5

**MEYER WIENER, M.D.**

Coronado, Calif.

**Practical Points in Surgery**

The course is planned to emphasize some of the vital points of technic of the various surgical procedures which will make the operations easier to perform. Application of the knowledge of anatomical structure of the tissues, their relation to other parts, as well as the physics involved will be discussed.

---

**COURSE 210**

Room 777 Periods M-6, T-6

**BENJAMIN J. WOLPAW, M.D.**

Cleveland, Ohio

**Hyperopia**

The basic optics of hyperopia as related to the correction of refractive errors in simple and compound hyperopia. The necessity for associating the hyperopic error with accommodation, muscle balance, type of individual, and occupation. Cycloplegics indicated in the refraction of hyperopic eyes of all age periods.

---

# OTOLARYNGOLOGY

## Continuous Courses

## 1947

---

**COURSE 51**

Room 731 Periods T-2, T-3

**COURSE 52**

Room 731 Periods W-2, W-3

**JOHN E. BORDLEY, M.D.**

Baltimore, Md.

BY INVITATION

**The Nasopharynx, Eustachian Tube and Middle Ear**

*A two-period course—$3.00*

Dr. Bordley's two-hour course "The Nasopharynx, Eustachian Tube and Middle Ear" is being presented twice as Course 51 and Course 52. **Please select course number according to day on which you wish to attend.**

This course will take up the anatomy physiology and pathology of the nasopharynx, eustachian tube and middle ear Special attention will be paid to the methods of examination, diagnosis and indications for treatment of the hyperplastic lymphoid tissue in the nasopharynx with radium.

**COURSE 53**

Room 782 Periods T-1, T-2

**COURSE 54**

Room 782 Periods W-1, W-2

**J. BARRETT BROWN, M.D.**

St. Louis, Mo.

BY INVITATION

**Facio-Maxillary Surgery**

*A two-period course—$3.00*

Dr. Brown's two-hour course "Facio-Maxillary Surgery" is being presented twice as Course 53 and Course 54. **Please select course number according to day on which you wish to attend.**

With the use of colored lantern slides, an outline will be presented of the lesions that have been studied in the Army and reports will be made of methods of repair.

**COURSE 55**

Room 752 Periods T-1, W-1, Th-1

**RAYMOND CARHART, Ph.D.**

Evanston, Ill.

BY INVITATION

**Audiometric Technic and the Fitting of Hearing Aids**

*A three-period course—$4.50*

This course will begin with a consideration of the methods of testing hearing by modern electronic equipment. The use of the audiometer as a diagnostic tool will be discussed, with special emphasis on its application in administering functional tests of hearing.

The theory and practice of audiometry will be covered in detail. Special tests such as "loudness balance" will be described. Monitored voice tests (speech reception) and their relation to pure tone results will be emphasized. The specific application of auditory masking will be explained. Group audiometry, methods of exposing malingering, and the interpretation of audiograms will be considered.

The tone field method of testing hearing in little children will be discussed.

The first half of the course will be devoted to the pure tone audiometry and other methods of testing hearing with particular emphasis upon the clinical application.

A survey of the construction and function of the modern audiometer will be followed by a consideration of currently acceptable instruments. The standards for audiometers set up by the Council on Physical Medicine of the American Medical Association will be reviewed. Procedures for the calibration and maintenance of audiometers will be outlined.

Technics for determining auditory malingering will be described and evaluated.

The last half of the course will be devoted to hearing aids.

The construction of the typical hearing aid and the main feature of hearing aid function will be discussed. Contemporary hearing aids can be grouped according to special characteristics. Examples will be given. Practical features of convenience, breakdown, maintenance, and repair will be outlined. The standards for hearing aids of the Council on Physical Medicine will be reviewed and the role of the Council discussed.

The course will conclude with an analysis of the otologist's responsibilities and opportunities. Lay abuses which threaten the best interests of those with impaired hearing must be eliminated. The practicing otologist must assert aggressive leadership in mobilizing community attitudes and facilities to meet hearing conservation needs and dealing with the problems of hearing aids.

**COURSE 56**

Room 786 Periods T-3, W-3, Th-3

**LOUIS H. CLERF, M.D.**

Philadelphia, Pa.

**Causes of Hoarseness With Particular Reference to Treatment**

*A three-period course—$4.50*

This course is devoted to a brief consideration of the symptom "hoarseness" and the diagnosis of conditions producing it.

Diseases of the larynx are discussed especially from the standpoint of treatment, and many of the more common typical and atypical laryngeal lesions are shown in kodachrome with movie film.

**COURSE 57**

Room 776 Periods T-1, T-2, W-1, W-2, Th-1, Th-2

**JOHN M. CONVERSE, M.D.**

New York, N. Y.

BY INVITATION

**Plastic Surgery of the Nose**

*A six-period course—$9.00*

This course is intended to give a review of recent developments in the technic of corrective and reconstructive surgery of the nose. This review will be divided into three parts:

1. Surgical anatomy of the nose. The anatomy of the nasal contour.
2. Traumatic deformities:
   A. Fractures and dislocations of the bones and cartilages of the nose.
   B. Nasal deviations.

C. Nasal depressions: saddle deformities.
D. Scars and loss of the skin covering of the nose.
E. Scars and loss of the inner lining of the nose.
F. Reconstruction for total or subtotal loss of the nose.

3. Congenital deformities:
A. Abnormal noses. Embryological and developmental defects. Nasal deformities in harelip.
B. Subnormal noses. Individual and racial variations in nasal shape.

Sociological and psychological aspects of nasal deformities. Use and abuse of the nasal plastic operation. The surgical technic will be demonstrated with colored moving pictures.

---

**COURSE 58**

Room 731 Periods Th-2, Th-3

**COURSE 59**

Room 731 Periods F-2, F-3

**STUART C. CULLEN, M.D.**
Iowa City, Ia.
BY INVITATION

**Practical Aids to Safe and Satisfactory Anesthesia for Otolaryngological Surgery**

*A two-period course—$3.00*

Dr. Cullen's two-hour course "Practical Aids to Safe and Satisfactory Anesthesia for Otolaryngological Surgery" is being presented twice as Course 58 and Course 59. **Please select course number according to day on which you wish to attend.**

Experience with this course during the past years has demonstrated that the factors in which most otolaryngologists are interested are the proper use of preanesthetic medication with morphine, scopolamine, and the short-acting barbiturates, the use of the endotracheal technic, the rational use of pentothal for anesthesia by the intravenous route, regional blocks for head and neck work, the safe use of cocaine and similar drugs, recognition of and treatment of reactions to these drugs, and the use of curare for difficult endoscopies.

These are the elements that will be emphasized in this two-hour continuous course. The discussion will be detailed but limited to the practical aspects. Specific examples will be used where possible and illustration of the material by slides will be done where it is feasible.

The first hour will be utilized in the discussion of preanesthetic medication, the endotracheal technic and the use of pentothal. The second hour will be devoted to the consideration of the use of cocaine and similar drugs, the reactions to these drugs, the regional blocks adapted to otolaryngological surgery, and the use of curare for difficult endoscopies.

Practical application of the fundamentals to be discussed in the course should result in more cooperative, comfortable and satisfied patients, safer and more convenient use of pentothal, extension of surgery under block anesthesia, safer and better results with cocaine, and facilitation of endoscopy in the difficult patient.

---

**COURSE 60**

Room 736 Periods T-1, T-2

**COURSE 61**

Room 736 Periods W-1, W-2

**MORRIS FISHBEIN, M.D.**
Chicago, Ill.
BY INVITATION

**The Technic of Medical Writing**

*A two-period course—$3.00*

Dr. Fishbein's two-hour course "The Technic of Medical Writing" is being presented twice as Course 60 and Course 61. **Please select course number according to day on which you wish to attend.**

The course will include an analysis of medical writing and of style. It will consider types of articles, such as the case report, new instruments. Next will be bibliographic research. Then will come a discussion of the construction of a suitable manuscript—from the title to the summary and conclusion, and the lecturer will then take up such details as the technical methods of preparation, including charts, illustrations, tables and other materials.

---

**COURSE 62**

Room 786 Periods T-1, T-2, W-1, W-2, Th-1, Th-2

**EDMUND P. FOWLER, JR., M.D.**
New York, N. Y.

**Histology of the Ear**

*A six-period course—$9.00*

Low and high power study of horizontal and vertical sections through the temporal bone at various critical levels.

The aim of the course is to teach the participants enough cross section anatomy in the temporal bone so that when they see medical illustrations of histological material they will be oriented.

**COURSE 63**

Room 750 Periods T-2, T-3

**COURSE 64**

Room 750 Periods W-2, W-3

**W. M. FOWLER, M.D.**

Iowa City, Ia.

BY INVITATION

**Practical Discussion of Blood Dyscrasias in Relation to Otolaryngology**

*A two-period course—$3.00*

Dr. Fowler's two-hour course "Practical Discussion of Blood Dyscrasias in Relation to Otolaryngology" is being presented twice as Course 63 and Course 64. **Please select course number according to day on which you wish to attend.**

**The Leukemias and Lymphomas**

A brief discussion of their manifestations and diagnosis with particular reference to their significance in the field of otolaryngology.

**The Anemias and Hemorrhagic States**

Their recognition and an evaluation of the various therapeutic measures for their control.

---

**COURSE 65**

Room 744 Periods T-2, T-3

**COURSE 66**

Room 744 Periods W-2, W-3

**COURSE 67**

Room 744 Periods Th-2, Th-3

**COURSE 68**

Room 744 Periods F-2, F-3

**WILLIAM C. HUFFMAN, M.D.**

Iowa City, Ia.

**Plastic Reconstruction of the Nasal Septum**

*A two-period course—$3.00*

Dr. Huffman's two-hour course "Plastic Reconstruction of the Nasal Septum" is being presented four times as Course 65, Course 66, Course 67, and Course 68. **Please select course number according to day on which you wish to attend.**

Dr. Huffman's course includes a new concept and approach to surgery of the nasal septum, the new concept being that the septal cartilage acts as a support to the external nose only when there is excessive scarring of the septal mucosa. In plastic reconstruction of the septum, the entire septal cartilage is removed and is either replaced with the patient's own cartilage or preserved cartilage. This operation allows correction of defects in the anterior-most portion of the septum without fear of a subsequent saddle nose.

**COURSE 69**

Room 744 Periods T-1, W-1, Th-1

**CHEVALIER L. JACKSON, M.D.**

Philadelphia, Pa.

**Surgery of the Larynx and Hypopharynx**

*A three-period course—$4.50*

---

**COURSE 70**

Room 752 Periods T-2, T-3

**COURSE 71**

Room 752 Periods W-2, W-3

**COURSE 72**

Room 752 Periods Th-2, Th-3

**COURSE 73**

Room 752 Periods F-2, F-3

**JULIUS LEMPERT, M.D.**

New York, N. Y.

**Modern Temporal Bone Surgery**

*A two-period course—$3.00*

Dr. Lempert's two-hour course "Modern Temporal Bone Surgery" is being presented four times as Course 70, Course 71, Course 72 and Course 73. **Please select course number according to day on which you wish to attend.**

Raison d'etre, plus lantern slide demonstration and description of new surgical technics developed and employed advantageously for:

Exostosis of the external auditory canal; correction of postauricular fistula; radical mastoidectomy; simple mastoidectomy, sinus thrombosis; middle fossa abscess; posterior fossa abscess; suppurative labyrinthitis; suppurative apicitis; facial nerve repair; modified radical mastoidectomy; improvement of hearing in clinical otosclerosis; decompression of labyrinthine hydrops; relief of tinnitus aurium.

---

**COURSE 74**

Room 796 Periods T-1, T-2

**COURSE 75**

Room 796 Periods W-1, W-2

**COURSE 76**

Room 796 Periods Th-1, Th-2

**COURSE 77**

Room 796 Periods F-1, F-2

**WERNER MUELLER, M.D.**

Boston, Mass.

**Practical Audiometry**

*A two-period course—$3.00*

Dr. Mueller's two-hour course "Practical Audiometry" is being presented four times as Course 74, Course 75, Course 76, and Course 77. **Please select course number according to day you wish to attend.**

The most important phase of the prob-

lem of deafness is its recognition. In this course the diagnosis of deafness will be dealt with under the following headings:

1. History taking.
2. The physical examination.
3. Audiometry.
4. Differential diagnosis.

The subject will be presented from the standpoint of the practicing otologist. Special emphasis is laid on idiopathic deafness.

---

**COURSE 78**

Room 736 Periods Th-1, Th-2

**COURSE 79**

Room 736 Periods F-1, F-2

**PHILIP MYSEL, M.D.**

Boston, Mass.

BY INVITATION

**Diagnosis and Treatment of Labyrinthitis Secondary to Acute and Chronic Suppuration**

*A two-period course—$3.00*

Dr. Mysel's two-hour course "Diagnosis and Treatment of Labyrinthitis Secondary to Acute and Chronic Suppuration" is being presented twice as Course 78 and Course 79. **Please select course number according to day you wish to attend.**

This instructional course deals with the subject of labyrinthitis both academically and clinically—that is, it reviews the anatomy and physiology of the labyrinth; gives the present-day value of the tests of labyrinthine function.

It summarizes the pathologic changes which occur in the labyrinth and the sources of infection which cause these changes.

Under the clinical topics will be the description of the standard operations on the labyrinth, when the labyrinth should be operated upon, the results which may be expected from the operation.

Associated conditions such as epidural abscess, petrositis, sinus thrombosis, meningitis, brain abscess and facial paralysis will be discussed in so far as they influence the management of labyrinthitis.

The effect of chemotherapy in the treatment of labyrinthitis will be compared with the results antedating the sulfonamides and the antibiotics.

---

**COURSE 80**

Room 754 Periods T-1, T-2

**COURSE 81**

Room 754 Periods W-1, W-2

**COURSE 82**

Room 754 Periods Th-1, Th-2

**COURSE 83**

Room 754 Periods F-1, F-2

**HENRY B. ORTON, M.D.**

Newark, N. J.

**Applied Surgery of the Neck**

*A two-period course—$3.00*

Dr. Orton's two-hour course "Applied Surgery of the Neck" is being presented four times as Course 80, Course 81, Course 82, and Course 83. **Please select course number according to day you wish to attend.**

The course will include the surgical procedures of the neck. Reviewing very briefly the fascial planes, surgical landmarks, and the important anatomical levels in the neck, before taking up the following surgical procedures:

Surgical approaches to deep infections of the neck; resection submaxillary gland; block dissection of the neck; thyroglossal duct cysts; branchial cysts and fistulae; cystic hygroma colli; tracheotomy; thyrotomy; laryngectomy; laryngopharyngectomy and plastic repair; lateral transthyroid pharyngotomy; arytenoidectomy; cervical esophagotomy and cervical mediastinotomy; pharyngeal diverticulum; phrenectomy; approach to roots of brachial plexus; operations on the thyroids; traumatic wounds of the neck; laryngostomy; and ligations.

Due to the large number of surgical procedures to be covered within the allotted time, the history, symptoms, and diagnosis of the various conditions will be eliminated.

---

**COURSE 84**

Room 740 Periods T-2, T-3

**COURSE 85**

Room 740 Periods W-2, W-3

**COURSE 86**

Room 740 Periods Th-2, Th-3

**COURSE 87**

Room 740 Periods F-2, F-3

**E. W. SCHELDRUP, M.D.**

Iowa City, Ia.

BY INVITATION

**Anatomy of the Neck**

*A two-period course—$3.00*

Dr. Scheldrup's two-hour course "Anatomy of the Neck" is being presented four times as Course 84, Course 85, Course 86, and Course 87. **Please select course number according to day on which you wish to attend.**

The neck will be presented in a simplified form based chiefly upon fascial relations. The connective tissue spaces and approaches to these will be discussed. The

major nerves and vessels will be considered with guides in approaching or avoiding the same. Lantern slides will illustrate the discussion.

---

**COURSE 88**

Room 734 Periods W-2, W-3

**COURSE 89**

Room 734 Periods Th-2, Th-3

**FRANK TURCHIK, M.D.**

Bridgeport, Conn.

**Plastic Reconstruction of the Nasal Septum**

*A two-period course—$3.00*

Dr. Turchik's two-hour course "Plastic Reconstruction of the Nasal Septum" is being presented twice as Course 88 and Course 89. **Please select course number according to day you wish to attend.**

A new concept of septal surgery has been evolved which restores physiologic nasal ventilation and avoids secondary deformity; namely, saddling of the dorsum, retraction of the columella, and drooping of the nasal tip.

Four operations on the septum will be described, and the indications for each approach will be discussed.

Operations:

1. Galloway modification, a purely rhinologic procedure.
2. Fomon operation which involves a complete transfixion to facilitate a typical rhinoplasty.
3. Total reconstruction of the nasal septum, applicable to the twisted nose.
4. Cosmetic septal operation to facilitate osteotomy and prevent asymetry of the nasal dorsum.

Diagrams and animated motion pictures will be used to illustrate technic.

---

# OTOLARYNGOLOGY

## Individual Courses

## 1947

---

**COURSE 301**

Room 716 Periods T-1, W-1, Th-1, F-1

**DANIEL H. ANTHONY, M.D.**

Memphis, Tenn.

**Affection of the Salivary Glands and Their Ducts**

1. (a) Anatomy
   (b) Physiology
2. (a) Symptoms
   (b) Diagnosis
   (c) X-ray findings

The following conditions will be discussed:

1. Congenital abnormalities of the salivary glands and their ducts.
2. Acute infection of the salivary glands and their ducts.
3. Acute epidemic inflammation of the salivary glands (mumps).
4. Chronic inflammation of the salivary glands and their ducts.
5. Calculi and foreign bodies in the salivary glands and their ducts.
6. Fistula of the salivary glands and their ducts.
7. Tuberculosis of the salivary glands.
8. Syphilis of the salivary glands.
9. Actinomycosis of the salivary glands.
10. Mikulicz's disease (pseudohypertrophy).
11. Ranula.
12. Neoplasma of the salivary glands.
13. Xerostoma.
14. Ptyalism.

---

**COURSE 302**

Room 705 Periods T-2, Th-2, F-2

**LOUIS J. BIRSNER, M.D.**

St. Louis, Mo.

**The Applied Surgical Anatomy of the Skull as a Whole**

This will be a practical presentation. It will not be a dry anatomical discussion. With the aid of slides and other material, a very thorough study of the important bony and muscular landmarks will be presented. Special emphasis will be laid upon the routes and pathways of infection. This course will be of great value to the younger men. In this course I will attempt to bring up the outstanding clinical points of many years of teaching and thirty-two years of practical experience.

---

**COURSE 303**

Room 716 Periods T-2; W-2

**IRL H. BLAISDELL, M.D.**

Syracuse, N. Y.

**Bronchography**

I. Introduction.
   (a) A brief resume of the use of iodized oil and other opaque media in the tracheobronchial tree for the purpose of lung mapping.

II. Indications and contraindications for the use of iodized oil.
   (a) A discussion of the problems of bronchiectasis, lung abscesses, and tumors of the lung.

(b) Since the operability of a patient must be determined as far as pneumonectomy and lobectomy are concerned, a definite localization of the pathology is important as well as a determination of the integrity of one side of the chest.

(c) The relationship of the bronchoscopist to the thoracic surgeon.

(d) The use of iodized oil in asthmatics and other pulmonary conditions will be considered.

III. Technics of Lung Mapping:

(a) The introduction of iodized oil by nasal catheter, by intralaryngeal catheter, through the bronchoscope, and hypodermic injection into the trachea will be illustrated by means of colored moving pictures.

IV. Consideration of bronchograms showing various types of pulmonary pathology will be shown, with sequelae following the use of opaque media.

---

**COURSE 304**

Room 705 Periods T-1, W-1, Th-1, F-1

**LAWRENCE R. BOIES, M.D.**

Minneapolis, Minn.

**Current Problems in Otology**

1. Is drainage of an acute middle ear suppuration important, or should we rely solely on the use of a sulfonamide or penicillin?
2. Has the local use of the newer medications changed the indications for surgical therapy of chronic suppurative otitis media?
3. Will the endaural approach to the temporal bone supplant the postaural approach?
4. Is the use of the audiometer alone adequate to determine the degree and type of hearing loss?
5. What advice shall we give our patients at the present time regarding the newer surgical procedures in otology:
   a. Fenestration for clinical otosclerosis.
   b. Labyrinthotomy for incapacitating Meniere's disease.
   c. Tympano-sympathectomy for the relief of tinnitus.

---

**COURSE 305**

Room 716 Periods T-3, W-3, F-3

**BEN L. BRYANT, M.D.**

Los Angeles, Calif.

**The Sphenopalatine Ganglion**

The role of the sphenopalatine ganglion will be considered as a trigger zone in relation to certain cephalgias and myalgias, as well as its connection with and influence on conditions elsewhere in the body. As a basis for this, its anatomy, and its anatomical relationship will be studied. The differential diagnosis of sphenopalatine ganglion neuralgia will be discussed, and the therapy will be described with emphasis placed on the practical aspect of the subject.

---

**COURSE 306**

Room 726 Periods T-1, W-1, Th-1

**EDGAR B. BURCHELL, D.Sc.**

New York, N. Y.

**Temporal Bone — Its Variations and Surgical Anatomy**

A lantern-slide lecture illustrating 75 dissected temporal bones from birth to adult age will be followed by an exhibit of dissected temporal bones showing anatomical variations that are impossible to show with lantern slides.

Some anatomic features which will be demonstrated are:

1. Different stages of pneumatization of the mastoid process and of the petrous pyramid; also the squamous portion of the temporal bone.
2. The sigmoid sinus groove situated far forward, and also superficially.
3. The jugular fossa, its variations in depth, its relation to the bulbar cells, and dehiscenses occurring in it.
4. Variations in Dorello's canal.
5. Long styloid processes (some of which extend into the tonsillar fossa).
6. The course of the VIIth cranial nerve from the internal meatus to the stylomastoid foramen.
7. The chorda tympani nerve in its relation to the middle ear.
8. The internal ear showing the cochlea, the vestibule and the 3 semi-circular canals.
9. Surgical landmarks that are an aid in the odd types of temporal bones.
10. Variations in dehiscence found in the tegmen mastoideum and tegmen tympani.
11. The intercommunication of the cellular structure demonstrated by Wood's metal casts of the temporal bone.

---

**COURSE 307**

Room 726 Periods T-3, W-3, Th-3, F-3

**PAUL A. CAMPBELL, M.D.**

Chicago, Ill.

**Differential Diagnosis of Hearing Loss**

The problem of hearing loss should be approached in an orderly manner by first

categorizing in one's mind the possible causes, then systematically eliminating the groups, segment by segment, through the use of intelligently applied tests and study of the symptomologic patterns. It is the purpose of this presentation to discuss rational thinking relative to the problem of hearing loss and to demonstrate the application of critical tests and short cuts to diagnosis.

---

**COURSE 308**

Room 705 Periods T-3, W-3

**NORTON CANFIELD, M.D.**
New Haven, Conn.

**Organization and Management of an Auditory Rehabilitation Clinic**

In the preliminary remarks I will make short reference to the organization of the units which were used in the Army and Navy and the plans that the Army and Navy have for peace-time continuation of their centers. The elements in these centers which can be applied to civilians will be mentioned and then I will continue by giving the organization of the centers which are being constructed and are in operation by the Veterans Administration.

There is considerable interest in this entire subject in a number of universities throughout the country and the organizational plans which they have will be discussed briefly. There are also community rehabilitation centers which are being advocated and constructed in several cities and the plans for these will be discussed. There are also several rehabilitation clinics for the deaf which are already in operation and have been for some time. The problems here involved will be reviewed.

The important thing for the organization and management of an auditory rehabilitation clinic, however, is to have the proper facilities from the standpoint of personnel available to all of the beneficiaries of the clinic. By this is meant that the professional groups which are represented by both the medical and the nonmedical professions must be available to every patient in need of their services. These various specialists will include the otolaryngologist, psychiatrist, clinical psychologist, acoustic expert, acoustic physicist, auditory trainer, lip and speech reading instructor, speech correctionist and the vocational guidance counsellor. Unless all of these facilities are available to individuals as needed and unless their entire program can be envisioned with all these various types of personnel, the rehabilitation of many individuals will be entirely incomplete.

The method by which the money can be raised to provide these facilities in any particular community will be mentioned and some experience of groups already in operation will be presented.

Great emphasis will be put upon the fact that this phase of medical attention to civilians must be under the control of the medical doctor. Many other nonmedical groups are extremely active in this country and are contributing greatly to this work. Unless the medical approach is given its due consideration, it will not receive the public support which it should because in the final analysis what the patient really wishes is to have his hearing acuity improved and that can properly come only through medical channels.

---

**COURSE 309**

Room 713 Periods T-1, W-1, Th-1

**ALFRED J. CONE, M.D.**
St. Louis, Mo.

**Direct Laryngoscopy — Advantages and Difficulties of Various Methods**

Direct laryngoscopy is usually accomplished easily by the Jackson, Lynch or Haslinger methods. The importance of head position has been stressed by Jackson for years. Anatomical variations in position of the thyroid cartilage, anomalies of the tongue or mandible may be such that direct examination is impossible except for the most skilled examiner.

The most common difficulty encountered is the heavy short-necked patient with emphysema and cardiac complications. Mechanical head holders will be reviewed. A new hydraulic adjustable head holder will be presented.

---

**COURSE 310**

Room 713 Periods T-3, W-3, Th-3

**JAMES B. COSTEN, M.D.**
St. Louis, Mo.

**The Diagnosis of Mandibular Joint Neuralgia**

Reflex pain and irritative effects produced by abnormal action of the mandibular joint will be reviewed. Age factors as shown by X-ray study will be shown, analyzing underlying defects in younger years, which produce the trismus cycle. Destructive results in later age groups will be evaluated, showing as far as possible how neuroses complicate actual pain values. The relationship of this symptom complex as a secondary or atypical neu-

ralgia to the major neuralgias will be differentiated. Symptomatic treatment during the process of restorative dentistry will be outlined. In addition to slides during the lecture, films of typical cases will be displayed.

**COURSE 311**

Room 706 Periods W-1, Th-1, F-1

**DANIEL S. CUNNING, M.D.**

New York, N. Y.

**Diagnosis and Treatment of Laryngeal Tumors**

The diagnosis and treatment of practically all laryngeal lesions, as seen in the Laryngeal Clinic of the Manhattan Eye, Ear and Throat Hospital, both benign and malignant, will be discussed with lantern demonstrations, including papilloma, polyp, vocal nodules, contact ulcers, fibroma, cystoma, lipoma, adenoma, myoma, lymphoma, chondroma, osteoma, varices, mycosis, fungoides, thyroid tumors, tuberculosis, syphilis and cancer. Paralysis of one or both vocal cords with the Kelly operation for bilateral abductor paralysis will also be discussed if time permits.

In the malignant cases, x-ray therapy and surgical treatment will be discussed. The technic of direct laryngoscopy, laryngofissure and laryngectomy will be taken up in detail.

**COURSE 312**

Room 713 Periods W-2, Th-2, F-2

**ARTHUR C. CURTIS, M.D.**

Ann Arbor, Mich.

BY INVITATION

**Diagnosis of Common Oral Lesions**

Diagnosis of oral lesions can be a difficult or a simple problem, depending upon a knowledge of the characteristics of the lesions commonly seen in the mouth, and also upon the possibility of related lesions appearing elsewhere. Illustrations by colored lantern slides and differential diagnosis of such lesions will be discussed.

It is hoped that a seminar type of presentation can be used rather than a didactic lecture.

**COURSE 313**

Room 706 Periods T-2, W-2, Th-2, F-2

**WARREN B. DAVIS, M.D.**

Philadelphia, Pa.

**Some Problems in Plastic and Reconstructive Surgery of the Face**

The importance of adequate training for plastic and reconstructive surgery of the face. Technic preferred in correction of various nasal deformities. Conservatism in operations upon alar cartilages urged. The use of rib and septal cartilage transplants. Care of deformities of the lips and of prolapsed ears.

**COURSE 314**

Room 706 Periods W-3, Th-3, F-3

**KENNETH M. DAY, M.D.**

Pittsburgh, Pa.

**Diagnosis and Treatment of Hydrops of the Labyrinth**

The pathology and symptomatology of hydrops of the labyrinth, or labyrinthine dropsy will be described. Differential diagnosis between this condition and others showing the Meniere syndrome. A brief discussion of the efficacy of various forms of conservative medical therapy. More detailed discussion of surgical procedures with a description of the speaker's operation upon the labyrinth and a report of his results to date.

**COURSE 315**

Room 726 Periods T-2, W-2, Th-2, F-2

**ROBERT M. DEARMIN, M.D.**

Indianapolis, Ind.

**Treatment of Chronic Mastoiditis**

Present concepts of conservative treatment with urea, sulfa drugs, penicillin, etc.

Treatment and surgery in childhood infections. Should radical mastoid surgery be done?

Indications for and method of doing the modified radical mastoidotomy.

Radical mastoidotomy.

1. Indications.
2. Pre-operative care.
3. Surgical procedure. Endaural and postaural approach.
4. The importance of after care and the results expected.

**COURSE 316**

Room 712 Periods W-1, Th-1, F-1

**RUSSELL M. DECKER, M.D.**

Pasadena, Calif.

**Management of Early Deafness**

The course will include a review of the routine examination necessary for detection of early deafness and an evaluation of the clinical significance of existing symptoms, with regard to the determination of extent and type of deafness. Early diagnosis in very young children requires the testing of hearing, especially when otoscopic examination reveals no defect and in the absence of a history of previous

illness or injury. The management of early deafness will be outlined, with special emphasis on the treatment of the pathologic processes involved, the prevention of further hearing loss, and the problems of social and vocational adjustment.

**COURSE 317**

Room 712 Periods T-2, W-2, Th-2

**WATT W. EAGLE, M.D.**

Durham, N. C.

**Some Uncommon but Important Otolaryngological Syndromes**

Some of the obscure headaches or pains in the face, pharynx and ear may be explained by some of the more uncommon conditions encountered in otolaryngological practice. Some of the syndromes are: sphenopalatine ganglion neuralgia, temporomandibular joint syndrome, acetanilid poisoning and brominism, elongated styloid process, Thornwaldt's bursa, and secretory otitis media. These topics will be discussed with emphasis on the diagnosis and therapy.

**COURSE 318**

Room 712 Periods W-3, Th-3, F-3

**BERT E. ELLIS, M.D.**

Indianapolis, Ind.

**Congenital Anomalies of the Head and Neck**

This course is designed to discuss the origin and treatment of some of the common as well as some of the rare congenital lesions around the head and neck. Harelip and cleft palate will not be discussed in this hour. Attention will be given to such lesions as dermoid cysts, branchial cleft, and thyroglossal cysts. Atresia of the posterior nares and atresia of the external auditory canal will also be discussed.

**COURSE 319**

Room 711 Periods T-2, W-2

**JOHN B. ERICH, M.D.**

Rochester, Minn.

**Fractures of the Maxilla and Malar Bones**

This course deals with fractures involving the maxilla and malar bones; methods of treatment which have been found most effective at the Mayo Clinic; and a classification which facilitates the management of such traumatic injuries. Various complications, including those associated with the cribriform plate of the ethmoid bone, will be discussed in detail.

**COURSE 320**

Room 711 Periods T-1, W-1, Th-1, F-1

**J. BROWN FARRIOR, M.D.**

New Orleans, La.

**Ear Surgery: Indications and Technic**

The purpose of this course is to summarize the present-day indications for ear surgery. Current therapeutic methods have greatly altered the criteria for surgical interference and the armamentarium of the otological surgeon has been greatly enhanced. The course will present the effect of these factors upon mastoidectomies, the choice of incisions, facial nerve surgery, the tympanosympathectomy, coagulation at the labyrinth and fenestration operation for otosclerosis, clarifying the indications for each, the reasons for success and the pitfalls of technic. The course will be illustrated with kodachrome slides of anatomical dissections, anatomical drawings and diagnostic charts.

**COURSE 321**

Room 710 Periods T-1, W-1, Th-1, F-1

**A. C. FURSTENBERG, M.D.**

Ann Arbor, Mich.

**Organic Neurological Considerations of Interest in Rhinolaryngology**

An anatomical study is combined with a discussion of clinical manifestations produced by neurological lesions which affect the laryngeal nerves, the olfactory nerves and the nerve supply to the salivary glands. Central lesions producing a laryngeal paralysis and those causing a disturbance of the sense of smell and a dysfunction of the salivary glands are particularly emphasized. Traumatic lesions of the facial nerve are described and methods of restoring function are offered. In this connection end-to-end anastamosis and the nerve implant are described in detail.

**COURSE 322**

Room 711 Periods W-3, Th-3, F-3

**T. C. GALLOWAY, M.D.**

Evanston, Ill.

**Respiratory Obstruction, Bulbar Poliomyelitis and Anoxia**

More important than obstruction at high level are the secondary effects due largely to increased negative thoracic pressure. These are congestion, exudation, atelectasis, and with superimposed infection various types of lung damage including bronchiectasis. Anoxia is important from the outset and, along with other quickly irreversible effects, determines the necessity and time of intervention.

Causes of respiratory obstruction are outlined. Tracheobronchitis is discussed as an example. Its diagnosis, physiopathology and treatment are outlined, including the indications and technic of cannulation and irrigation and postural drainage.

Bulbar polio is often a laryngeal emergency in which not enough attention has been paid to flooding of the airway and lungs. Anoxia, reflex respiratory difficulty, and, if a respirator is used without successful aspiration of secretion, drowning may ensue. Anoxia frequently accounts for important symptoms ascribed to the virus. Oxygen, aspiration, postural drainage and the respirator can save most cases. Tracheotomy may, however, be life saving by relieving asphyxia, short circuiting secretions and permitting better aspiration.

Flame injuries, anesthetic crises and sudden death after successful tracheotomy will be touched upon. Blood chemistry and pH will be discussed in relation to the use of oxygen, carbon dioxide and surgical intervention.

---

**COURSE 323**

Room 711 Periods Th-2, F-2

**WILLIAM D. GILL, M.D.**
San Antonio, Tex.

**External Otitis**

A classification of the various clinical types of inflammation of the external ear will be presented with discussion of the differential diagnostic features of each type. Clinical and laboratory diagnostic aids will be considered. Otomycosis will be especially emphasized. Therapeutic measures employed in treating the various types of otitis externa will be reviewed and the antibiotics and newer fungicides will be considered. Prophylactic measures will be included.

---

**COURSE 324**

Room 734 Periods T-1, W-1, F-1

**OLIVER S. GILLILAND, M.D.**
Kansas City, Mo.

**Therapy and Surgery in Sinusitis in Children**

Development of ethmoids and antra in fetal life makes the infant liable to infections in these sinuses any time after birth.

Involvement of sphenoids and frontals occurs much later in childhood.

Sinus disease not only a complication but probably a component part of every nasal infection in childhood.

Roentgenograms often unreliable in diagnosing this condition.

Aspiration of antra sometimes required to make a definite diagnosis.

Systemic and local therapy including the use of antibiotics usually successful in clearing up infections.

Occasionally irrigation of antra or an intranasal antrotomy necessary.

Radical surgery not indicated.

---

**COURSE 325**

Room 728 Period T-1
Room 722 Period W-1
Period Th-1

**HENRY M. GOODYEAR, M.D.**
Cincinnati, Ohio

**Neck Infections. Practical Points in Diagnosis and Approach to Infections Involving the Pharynx and Spaces of Neck and Mouth**

General and local treatment plus nasal feeding where indicated.

Agranulocytic angina and the use of pyridoxine.

Vincent's angina and the use of copper sulphate solution.

Method of aspirating retropharyngeal abscesses.

Peritonsillar infection; anesthesia, indications for enucleation.

Infections of the jugular vein. Indications for ligating the carotid artery.

External and internal approach to pharyngomaxillary infections; infections of the submaxillary gland and the removal of calculi through the floor of the mouth.

Ludwig's angina, its approach and relation to the molar teeth.

Slides and anatomical specimens.

---

**COURSE 326**

Room 722 Periods T-2, W-2

**COLBY HALL, M.D.**
Los Angeles, Calif.

**The Management of Acute Neck Infections**

This course will deal with the *clinical aspects* of acute neck infections.

1. The immediate evaluation of the patient.
2. The problems of dyspnoea and sepsis.
3. Differential diagnosis of disease in the pharyngomaxillary space, the masticator space, Ludwig's, and other suprahyoid areas.
4. The importance of clinical pathology: suppurative, nonsuppurative, vascular (arterial and phlebitic), diphtheria.
5. Practical anatomy.

This will *not* be an hour of fascias and anatomy.

This will be an hour devoted to the discussion of the clinical management of patients with acute neck infections.

**COURSE 327**

Room 724 Periods T-1, W-1, Th-1, F-1

**FRENCH K. HANSEL, M.D.**

St. Louis, Mo.

**Allergy of the Nose and Paranasal Sinuses**

In the discussion of this subject will be considered the fundamental principles of diagnosis and treatment. A new rapid technic of staining ocular, nasal, and bronchial secretions will be demonstrated. Among the various methods of treatment, detailed consideration will be given to dust therapy.

**COURSE 328**

Room 724 Periods W-3, Th-3, F-3

**GORDON F. HARKNESS, M.D.**

Davenport, Ia.

**The Private Practice of Otolaryngology**

Otolaryngology as a surgical specialty.

A short resume of its development.

Its present-day position.

Its future.

Some of the art and economics of otolaryngologic practice.

A potpourri of little things in private practice not usually mentioned in text books that help make it successful.

**COURSE 329**

Room 728 Periods T-3, W-3, Th-3, F-3

**V. K. HART, M.D.**

Charlotte, N. C.

**Clinical Aspects of Bronchiectasis From the Bronchoscopic and Rhinologic Viewpoint**

This talk covers primarily the etiology of acquired bronchiectasis. Evidence is presented to show that much bronchiectasis results from bronchial obstruction with a secondary atelectasis. Such atelectasis if unresolved in six or eight weeks results almost inevitably in bronchial cavitation.

Consequently, great stress is laid on prevention. A high percentage of all atelectasis is amenable to bronchoscopic treatment in the early stages.

Evidence is also presented to show that frequent concomitant sinus suppurations are relatively unimportant etiologically. Their treatment is advisable prior to chest surgery.

The lecture is illustrated with lantern slides and a motion picture film.

**COURSE 330**

Room 705 Periods Th-3, F-3

**FRED Z. HAVENS, M.D.**

Rochester, Minn.

**Plastic Repair of Extensive Deformities of the Face**

Extensive deformities of the face may result from trauma or from the treatment of disease. Lesions which may lead to extensive deformity will be discussed briefly and methods of repair of such deformities and of those resulting from trauma will be illustrated and discussed in considerable detail. Owing to the fact that correction of major deformities of the face may present unusual problems, the discussion necessarily will touch upon a variety of methods—skin grafts, cartilage, bone, dermal and fat implants, use of adjacent tissue, of forehead and sickle flaps as well as of tubes and flaps from more distant parts.

**COURSE 331**

Room 710 Periods T-3, W-3, Th-3, F-3

**FREDERICK T. HILL, M.D.**

Waterville, Maine

**The Management of Antro-Oral Fistulae**

Antro-oral fistulae frequently present problems which tax the ingenuity of the surgeon. While the majority result from dental extractions, they may follow cysts or tumors of the upper jaw, infection, trauma or operation on the antrum. Successful management depends upon careful study of the individual case and the application of sound principles of plastic surgery. Control of antral infection is essential.

No single procedure is adaptable to all cases. Sufficient alveolar process to allow the use of dentures should be preserved in all alveolar cases. Canine fossa fistulae may be extremely difficult to manage.

Various methods of closing both alveolar and canine fossa fistulae, including the use of the hinged and the sliding flaps, will be presented.

**COURSE 332**

Room 722 Periods Th-3, F-3

**PAUL H. HOLINGER, M.D.**

Chicago, Ill.

**Photography in Otolaryngology and Broncho—Esophagology**

Principles of endoscopic photography will comprise the basic subject of this

course. These will consist of a general consideration of illumination, finding and focusing, types of endoscopes, criteria for judging cameras and other equipment, and an evaluation of results obtained. The specific problems encountered in photography of the various endoscopic fields — the ear drum, anterior nasal chamber and post nasal space, the pharynx, larynx, tracheobronchial tree, and esophagus — will be presented, with methods now in use for photography of each.

**COURSE 333**

Room 722 Periods T-1, Th-2, F-2

**PAUL H. HOLINGER, M.D.**
and
**WILLIAM H. CASSELS, M.D.**

BY INVITATION
Chicago, Ill.

**Technics of Anesthesia for Peroral Endoscopy**

The various types of local and general anesthetic agents suitable for peroral endoscopic procedures will be discussed. Mention will be made of the specific indications and contraindications for each, with emphasis on the technics of administration. Most endoscopic procedures are carried out under topical anesthesia. The diagnosis, prophylaxis, and treatment of toxic reactions which may occur with any of these agents will be presented. General anesthesia for endoscopic procedures presents some unique problems. The anesthetist, as well as the endoscopist, is concerned about the patient's respirations. Cooperation and mutual understanding are essential. Certain methods have been found most satisfactory, while others involve serious hazards. The administration of curare to the conscious patient is sometimes desirable and the use of this agent will be discussed.

**COURSE 334**

Room 17 Periods T-2, W-2, Th-2 F-2
Club Floor

**BAYARD T. HORTON, M.D.**
Rochester, Minn.

BY INVITATION

**Headaches and Head Pains**

The specific types of headaches and head and face pains, the recent advancements in their treatment, and the pain mechanisms involved will be considered. There will be a discussion of the differential diagnosis and treatment of the various syndromes, including the migraine and histaminic cephalgia. The clinical features such as location and distribution of the pain, associated ophthalmologic and rhinologic manifestations will be emphasized as an aid in classification. Each clinical entity will be clearly illustrated by technicolored movies.

**COURSE 335**

Room 728 Periods W-1, Th-1, F-1

**HOWARD P. HOUSE, M.D.**
Los Angeles, Calif.

**Endaural Approach to Temporal Bone Surgery**

This illustrated course is designed to acquaint the otologic surgeon with the advances and technic of the endaural approach to the temporal bone.

The endaural incision will be illustrated and discussed at length. The necessity of making the incision exactly right will be stressed.

The advantages of the dental burr in temporal bone surgery, together with the technic of its use, will be noted.

The following operations will be discussed:

1. Simple mastoidectomy.
2. Modified radical mastoidectomy.
3. Radical mastoidectomy (mastoidotympanectomy).
4. Surgery of the petrous apex (mastoidotympanoapicectomy).
5. Surgery of the labyrinth.
   a. Lempert fenestration operation for otosclerosis.
   b. Day operation of Meniere's disease.
   c. Other labyrinthine procedures.

The importance of proper postoperative care in endaural cases, together with the end results of the surgery, will be noted.

**COURSE 336**

Room 727 Periods T-1, W-1, Th-1, F-1

**LELAND G. HUNNICUTT, M.D.**
Pasadena, Calif.

**The Practical Application of Histopathology to Office Practice**

An understanding of histopathology is necessary to satisfactorily treat ear, nose, and throat conditions. Slides to illustrate the points brought out are from the speaker's material. Of special value is the effect of medication on the tissues. A copy of the course will be given each student.

**COURSE 337**

Room 727 Periods T-2, W-2, Th-2

**PERCY E. IRELAND, M.D.**
Toronto, Can.

**Surgical Emergencies in the Practice of Otolaryngology**

It has been said that hemorrhage and choking are the only two emergencies critical in our specialty. The causes and treatments of these are to be discussed. The course is designed to present a definite plan which may be used when we are suddenly confronted with a serious problem.

A short review of the physiology of hemorrhage will be given. The reasons for failure to control bleeding will be discussed and all the methods of control, including our present armament of the newer hemostatic agents. The uses of styptics, caustics and various methods of packing, ligature and pressure will be considered, followed by the expected results from the use of thromboplastics, oxidized cellulose, bovin thrombin, human thrombin and fibrin foam.

The problem of "choking" will be discussed from the viewpoint of causes of obstruction to breathing. This will include indications, methods and difficulties in the tracheotomy procedure. Methods of resuscitation will also be considered.

In addition to the two grave emergencies above, there will be added cocaine poisoning and a few other more worrisome problems. An attempt will be made throughout to make all of the material presented of a clinical, rather than a scientific, consideration of the subject.

---

**COURSE 338**

Room 727 Periods T-3, Th-3, F-3

**ARTHUR C. JONES, M.D.**
Boise, Idaho

**Office Procedures in Otolaryngology**

A few words regarding the general conduct of the office and some suggestions as to how to cut down on the foot mileage in a large office.

Suggestions in handling a group of assistants, both nurses and doctors in the office.

Instructions and suggestions as to the danger of using cocaine in the office and directions as to how to prevent toxic reactions of cocaine.

My own personal outline in treating acute head colds, acute ears and acute sinuses.

Remarks in the use of sulfa and penicillin in the prevention of mastoiditis and other infections.

Special technic for treating pain from irritation from the Meckels ganglion.

Special technic in injecting the occipital nerve.

A rapid way of relieving a closed allergic nose for a period of six weeks to three months.

The instructions for making a cocaine nontoxic oil spray to be used in the office for shrinking of the nose preliminary to Proetz and other nasal medications.

A few suggestions regarding the use of diathermy as a surgical instrument.

Special suggestions for the after care of submucous operations.

Highly satisfactory method of treating external otitis, either bacterial or fungus.

A brief form of advice to people with otosclerosis and nerve deafness.

---

**COURSE 339**

Room 728 Periods T-2, W-2, Th-2

**CHARLES E. KINNEY, M.D.**
Cleveland, Ohio

**Practical Office Audiometry**

I. General Consideration:
Accurate hearing testing and experimental work along these lines requires considerable technical training and extensive equipment. The average otologist can do 85 per cent as good work with a little practical common-sense understanding and no more equipment than an audiometer.

II. A quick practical way to survey all new patients is presented, thus eliminating the old watch tick and spoken voice tests.

III. Accurate audiometric diagnosis:
a. Practical considerations of extraneous noises. So-called "Sound Proof Room" NOT necessary.
b. Consideration of the audiometer with particular reference to frequent office calibrations.
How to get the audiometer out from under a stack of medical journals.
c. Practical points as to the recording and evaluating of findings.

---

**COURSE 340**

Room 784 Periods W-1, Th-1, F-1

**CLAIR M. KOS, M.D.**
Des Moines, Ia.

**Interpretation of the Vestibular Test in Office Practice**

This course will consist of a discussion of current theoretical and factual concepts of vestibular labyrinthology. The various methods and technics employed to excite the elements of the vestibular complex will be described. Vestibular responses reflected by the presence or absence of directional preponderance of nystagmus evoked by

induced excitation with warm and cold thermal alterations equivariant with body temperature will be demonstrated. Observations made on patients in whom the Fenestra-Nov-Ovalis was created as well as studies conducted on patients exhibiting a variety of neurologic disturbances will be presented. Vestibular test results will be interpreted according to the principle that induced excitation of the vestibular apparatus merely amplifies or magnifies normal, latent or spontaneous vestibular activity.

### COURSE 341

Room 788 Periods W-2, Th-2, F-2

**J. R. LINDSAY, M.D.**
Chicago, Ill.

**Vertigo from the Otological Standpoint**

The course has been designed to cover the routine examination necessary in cases of vertigo, the differential diagnosis and treatment. It includes:

1. Definition of the terms Meniere's symptom complex, Meniere's disease, and pseudo-Meniere's syndrome as commonly used.
2. Routine examination and method of recording findings.
3. Significance of the findings in localizing the site of disease. Significance of postural vertigo.
4. Differential diagnosis in so-called pseudo-Meniere's and Meniere's syndrome.
5. Discussion of Meniere's disease, including clinical stages, auditory characteristics, pathology and treatment.

### COURSE 342

Room 722 Periods T-3, W-3

**JOHN S. LUNDY, M.D.**
Rochester, Minn.

BY INVITATION

**The Choice of Anesthetics and Special Points of Technic in the Administration**

The course will cover the various technical points in connection with anesthesia and supportive therapy, with emphasis on technical points with regard to making difficult venipunctures easier. Among the agents to be considered for supportive therapy will be the standard solutions of normal saline, dextrose with and without saline, 5 per cent alcohol and 5 per cent dextrose in water, 6 per cent gelatine of large molecular size, Macrose which is a new polysaccharid of large molecular weight, blood and plasma. Endotracheal anesthesia will be emphasized as well as the advantages of tracheobronchial aspiration during and after operation. The problem of anesthetization in the presence of bilateral vocal cord paralysis will be discussed. Lantern slides will be used.

### COURSE 343

Room 724 Periods T-2, W-2, Th-2, F-2

**MERCER G. LYNCH, M.D.**
and
**F. E. LeJEUNE, M.D.**
New Orleans, La.

**Suspension Laryngoscopy and Its Uses**

The course will deal with a brief history of suspension laryngoscopy and its development to present day technic.

The question of anesthesia will be discussed. The type of anesthesia best suited for suspension, with variations as adaptable to different patients and the handling of different laryngeal lesions.

A description of the instrument will be given with special reference to the suspension hook and choice of spatulas as adaptable to different patients and different pathologic conditions. Following this, the technic of suspension laryngoscopy will be discussed and a motion picture shown to further emphasize the more important points in technic.

There are limitations and conditions preventing the use of the instrument, but also definite advantages to this type of procedure in many cases. These limitations will be discussed and the advantages of this procedure brought out in a discussion of intralaryngeal surgery.

Slides will be shown to illustrate the points and advantages using this type of procedure for intralaryngeal surgery.

### COURSE 344

Room 788 Periods T-1, W-1, Th-1

**JAMES H. MAXWELL, M.D.**
Ann Arbor, Mich.

**Surgery of the Parotid Gland**

After a brief discussion of the differential diagnosis of swellings in the parotid region, special consideration will be given to the surgical lesions of the parotid gland including inflammatory and neoplastic processes. In discussing surgical operations involving the parotid gland, methods of identification of the facial nerve and means of avoiding undue trauma to this structure will be described. Motion pictures showing surgical operations for the removal of different types of parotid gland tumors will be shown.

**COURSE 345**

Room 750 Periods Th-2, F-2

**CARL H. McCASKEY, M.D.**
Indianapolis, Ind.

**Neck Surgery in Otolaryngology**

If one is to do this type of surgery he should have a basic background, especially in anatomy, pathology, bacteriology, diagnosis and differential diagnosis, and surgical technic. Tumors, neck abscesses, tracheotomies, laryngofissures, laryngectomies, thyroglossal duct cysts, fixation of vocal cords, esophageal diverticulae and other problems involving neck surgery will be discussed.

**COURSE 346**

Room 784 Periods T-1, W-2, Th-2

**JOHN G. McLAURIN, M.D.**
Dallas, Tex.

**The Indications for Radical Sinus Surgery and Recommendations for Adequate Procedure for the Individual Case**

The instructor attempts to give a fairly complete list of sinus diseases that should be handled by so-called radical surgical procedures. He prefers that such operations be considered adequate to obtain maximum relief for each individual case where surgery is contemplated. He describes briefly the operation which has been most satisfactory in his hands in dealing with sinus diseases that will not respond to simple measures of drainage and ventilation.

He attempts to point out some reasons for failure where such operations are done and explains how some of these failures can be obviated.

Some remarks are made regarding the best type of anesthesia to be used in such operations.

**COURSE 347**

Room 784 Periods T-3, W-3, Th-3, F-3

**W. J. McNALLY, M.D.**
Montreal, Can.

**Practical Methods for Carrying Out Labyrinthine Tests**

This course is designed to present the basic or elementary facts which are necessary for an understanding of clinical labyrinthine tests. Much of the time will be taken up in studying the actual methods of carrying out the various tests. Time will also be given to working out the interpretation of reactions which follow the simple tests. The simplicity of the tests will be stressed because they can be made very complex. There is still so much speculation and so much to be learned about labyrinthine function that there is no universal agreement about many of the points which come up in the course of a labyrinthine examination. There are, however, a sufficient number of basic facts known to justify the general adoption of a labyrinthine test which should become part of the otologist's routine examination. As a brief introduction, the anatomy and physiology of the semicircular canals and the otoliths will be reviewed. The examination of spontaneous phenomena will be considered. Guides for the detection of spontaneous nystagmus will be given. An interpretation of past-pointing tests and its relation to vestibular stimulation will be studied. The technic of performing the caloric and rotation tests will be taken up in detail. The rules which govern labyrinthine reactions will be used to interpret the results of these tests. Finally the significance of the results of the tests in labyrinthine and intracranial diagnosis will be investigated.

**COURSE 348**

Room 739 Periods T-1, W-1, Th-1, F-1

**RALPH J. McQUISTON, M.D.**
Indianapolis, Ind.

**Deep Infections of the Neck**

This course considers the deep infections of the neck which are related to otolaryngology. The general surgical anatomy of the mouth, pharynx and neck are reviewed with special attention given to the fascial planes, muscles and other structures which aid in the formation of the various spaces and fossae in the neck. These structures may act either as pathways or barriers which greatly influence infections which invade the neck.

Suppurations which occur in the retropharyngeal, pharyngomaxillary, submaxillary, masticator, and parotid spaces are discussed as to their etioloy, clinical picture and their medical and surgical management.

The most common complications of deep infections of the neck, such as septicemia, hemorrhage and asphyxia, present a serious situation and are given consideration as to their etiology and management

The lecture is supplemented by lantern slides and illustrations.

**COURSE 349**

Room 784 Periods T-2, F-2

**ALDEN H. MILLER, M.D.**

Los Angeles, Calif.

**Tracheotomy in Children**

This course will be a practical discussion of the problems encountered in considering and doing tracheotomies on small children, including such subjects as:

**Indications:** The newer ones, bulbar poliomyelitis and bronchiolitis as well as the accepted indications for immediate and elective tracheotomy.

**Technic:** Anesthesia; kind and site of skin incision; tracheal incision; use of the bronchoscope.

**Postoperative Care:** Moisture, control of crusting and secretions; decannulization.

**Complications:** Pneumothorax; subcutaneous and mediastinal emphysema.

---

**COURSE 350**

Room 788 Periods T-3, W-3, Th-3

**PAUL M. MOORE, M.D.**

Cleveland, Ohio

**Lesions of the Mouth**

A Kodachrome illustrated discussion of many of the lesions seen in the mouth. This will include lesions of the mucous membranes, gums, tongue, bone, and adjoining structures and the oral manifestations of some of the general diseases.

---

**COURSE 351**

Room 738 Period T-1

**HARRIS P. MOSHER, M.D.**

Marblehead, Mass.

**Brain Abscess from Infections of the Skull**

The purpose of this paper is to answer the question whether the otolaryngologist who encounters a brain abscess complicating osteomyletis of the frontal bone or a brain abscess arising from infection of the middle ear, the mastoid of the lateral sinus, should treat it or transfer it to the neurological surgeon. What technic does the neurological surgeon use which the otolaryngologist does not employ or cannot easily acquire to make him equally competent to handle these cases?

A brief description of the pathways of infection, and the major signs and symptoms of brain abscess will precede the discussion of surgical measures.

---

**COURSE 352**

Room 795 Periods T-2, W-2, Th-2

**JOHN R. MYERS, M.D.**

Syracuse, N. Y.

BY INVITATION

**Nasal Allergy**

This course will include the various phases of the practical side of the subject of nasal allergy and its allied conditions.

The methods of diagnosis and treatment, both local and general, will be discussed from the practicing rhinologist's viewpoint.

---

**COURSE 353**

Room 738 Periods W-1, Th-1, F-1

**C. STEWART NASH, M.D.**

Rochester, N. Y.

**The Functional Diseases of the Nose**

It has been estimated that about 90 per cent of the diseases of the nose are physiologic in origin.

If the normal functions of the nose are to be maintained, the nasal mucosa must be normally active; it can neither hypofunction nor hyperfunction.

This course will include informal lectures and discussions on such hypofunctioning diseases as physical, psychic, and febrile rhinitis; and such hyperfunctioning diseases as irritative, infective (the common cold) and endocrine rhinitis. Emphasis is to be laid on conditions which are often unrecognized and consequently improperly treated.

---

**COURSE 354**

Room 734 Periods T-2, Th-1, F-2

**HENRY B. PERLMAN, M.D.**

Chicago, Ill.

**Quantitative Tests of Eustachian Tube Function**

Every patient with ear diseases requires an evaluation of his tubal function. Disturbance of the eustachian tube function may take the form of abnormal patency or varying degrees of obstruction. The latter is of importance in recently developed industries such as aviation, caison workers, etc., and is a primary factor in development of chronic middle ear deafness in childhood. The methods which have long been in use for testing this function are qualitative and difficult to evaluate. More exact methods should be developed if we are going to be able to define the normal and pathologic tube.

In this presentation an attempt will be made to point out the value and significance of some of the well-known maneuvers such as Valsalva, Toynbee, etc., and to demonstrate how such tests can be measured quantitatively. Obstruction of the tube produces a reduced air pressure in the middle ear. A simple method for determining the air pressure in the middle ear will be demonstrated. Details of the

method and its practical application will be presented. Results obtained on controlled experiments in normal and pathologic ears will be discussed.

Politzerization may be used to determine the degree of eustachian tube obstruction. An apparatus will be shown by which the resistance of the tube to inflation during swallowing can be measured. The practical value of such a method for tubal inflation is pointed out.

**COURSE 355**

Room 795 Periods T-1, W-1, Th-1, F-1

**JOEL J. PRESSMAN, M.D.**

Los Angeles, Calif.

**A Discussion of Problems in Bronchoscopy and Laryngeal Surgery**

The course, entirely for those of limited experience, consists of a brief resume of impressions of various factors involved in the practice of endoscopy and laryngology. Beginning with a discussion of the organization and material arrangement of a small bronchoscopic clinic it will include the author's conclusions as to the value of various types of instruments, methods of anesthesia, technic of treatment of a limited number of diseases, diagnostic problems, lipiodol instillation and experiences with some phases of laryngeal surgery. The course is intended to briefly present a potpourri of isolated individual facts and conclusions which stand out as highlights in the background of experience of the author. No theoretical consideration will be given to any problems but, on the contrary, the material presented will be of a very practical nature.

**COURSE 356**

Room 788 Periods T-2, F-1

**ROBERT E. PRIEST, M.D.**

Minneapolis, Minn.

**Indications and Technic of Tracheotomy**

This course will review the history, indications and technic of tracheotomy. A brief discussion of tracheotomy in bulbar poliomyelitis on a basis of experiences at the University of Minnesota Hospitals and the Minneapolis General Hospital during the 1946 Minneapolis poliomyelitis epidemic will be given. A short movie on the technic of tracheotomy and post-tracheotomic care will be presented.

**COURSE 357**

Room 738 Periods T-2, W-2, Th-2, F-2

**ARTHUR W. PROETZ, M.D.**

St. Louis, Mo.

**Treatment and Diagnosis by the Displacement Method**

The first half of the period will be devoted to a motion picture, a discussion of principles and details of technic, and a demonstration of the method on a subject.

The second half of the period will deal with solutions and indications for treatment, and with x-ray films showing the diagnostic points peculiar to the filling of sinuses with radiopaques by displacement.

In order to conserve time it will be assumed that the listener has some familiarity with the method, and the discussion will be kept on a practical clinical level.

**COURSE 358**

Room 750 Periods T-1, W-1, Th-1

**A. G. RAWLINS, M.D.**

San Francisco, Calif.

**Allergic Sinusitis**

The allergic membrane as the commonest cause of chronicity in chronic sinusitis. Mechanism of the allergic reaction. How physical allergy comes into the picture. About histamine. Pathology produced in allergy. About eosinophils. Diagnosis of allergic sinusitis. How to take history. Doing nasal and nasopharyngeal smears for eosinophils. Nasopharynx as choice spot for specimens. New method. Clinical examination of nose often practically negative in mild cases. Cause of the perennial allergic membrane is usually the perennial inhalants. We can eliminate many from patients' environment. Four that cannot be eliminated are: dust, mold, tobacco smoke, and paper. About dust, mold, tobacco smoke, and paper. Testing. Method of testing and dilutions. Treatment — general and specific. Newer concept of hyposensitization. Low and optimum dosage. Useful drugs. Treatment for physical allergy. How to simplify nasal allergy for rhinologist. Summary. Slides.

**COURSE 359**

Room 732 Periods T-2, W-2, Th-2

**SCOTT N. REGER, Ph.D.**

Iowa City, Ia.

**The Selection of Hearing Aids**

I. Obligation of the otologist to the patient in need of a hearing aid.
II. Types of hearing aids.
III. Indications for use of hearing aids.
IV. Psychological acceptance; social and economic considerations.
V. Interpretation of audiometric and controlled speech tests.
VI. The individually molded ear piece.

VII. Minimum practical selection procedures without the use of laboratory equipment.
VIII. Hearing aid clinic selection procedures; performance ideals.
IX. Use training; performance recheck; lip reading.
X. Recommendations and Reports of the Committee on Hearing Aids of the Council on Physical Medicine of the American Medical Association.

**COURSE 360**

Room 732 Periods T-3, W-3, Th-3

**SCOTT N. REGER, Ph.D.**
Iowa City, Ia.

**Technic and Interpretation of Functional Hearing Tests**

I. General considerations:
  a. Testing environment
  b. Calibration and maintenance of equipment
  c. Testing procedures
  d. Masking noise
  e. Factors influencing accuracy
II. Diagnostic differentials:
  a. Threshold profile for pure tones
  b. Degree of impairment
  c. Relationship between air and bone conduction acuity
  d. Mobility tests
  e. Response to pure tones above threshold levels; loudness level balancing and equal loudness level contours
III. Controlled intensity level speech tests
IV. Malingering
V. Recording and classification of audiometric measurements
VI. Evaluation of hearing disability
VII. Recommendations of the Audiometer Standardization Committee of the Council on Physical Medicine of the American Medical Association.
VIII. Group audiometry

**COURSE 361**

Room 732 Periods T-1, W-1, Th-1

**J. M. ROBB, M.D.**
Detroit, Mich.

**Proven Procedures in Otolaryngological Office Practice**

By "proven" is meant those procedures that have been used sufficiently often to obtain good results in ear, nose, and throat practice in the lake district of the Middle West over a period of twenty-five years.

The ear, nose, and throat will be taken up separately; conditions discussed; treatments recommended; and the drugs and instruments used to get the best results. Of special interest will be a presentation of a case of supposed vasomotor, hyperesthetic or allergic rhinitis.

**COURSE 362**

Room 739 Periods T-2, W-2, Th-2, F-2

**J. M. ROBISON, M.D.**
Houston, Tex.

**The Treatment of Maxillary Sinusitis**

This course will be limited to consideration of the more conservative measures used in the treatment of maxillary sinusitis. The radical maxillary sinus operations will not be discussed. The physiology and functional pathology of the sinus mucosa and the turbinates will be correlated. A classification of sinusitis based on the presence or absence of irreversible structural changes in the sinus mucosa will be offered, which encompasses the purulent, allergic, and mixed types. The pathologic changes in the sinus mucosa which are present in the various types of sinusitis will be illustrated and correlated with clinical history and symptomology. The theory and practice of the application of local pressure to the mucosa of the maxillary sinus by means of balloons filled with iodized oil will be described and illustrated. The technic of the application of local pressure and the criteria used in the selection of patients suitable for this procedure will be discussed. Students registering for this course will be given reprints covering its fundamentals.

**COURSE 363**

Room 733 Periods T-2, W-2, Th-2, F-2

**SAMUEL SALINGER, M.D.**
Chicago, Ill.

**Nasal Fractures: Recent and Old; Diagnosis and Treatment**

This presentation will include demonstration of types of fractures in adults and children; evaluation of local signs; correct projections for satisfactory x-ray films; discussion of methods of reduction; time of application and means of retention.

Complications such as external lacerations and fractures involving other facial bones.

Old fractures will be considered from both the functional and cosmetic point of view and methods of correcting the intranasal as well as the external deformity will be presented.

**COURSE 364**

Room 738 Periods Th-3, F-3

**LeROY A. SCHALL, M.D.**
Boston, Mass.

**Cancer of the Upper Respiratory Tract**

This lecture concerns itself with histopathology of the more common malignant diseases that involve the nose and the nasal accessory sinuses.

There will be a discussion of the cell types, behavior and characteristics of each individual group. Although this discussion will be somewhat elementary, it is necessary for a thorough appreciation of the treatment and behavior of malignant disease.

The symptoms of nasal and sinus malignancy will be considered, based upon the location and type of the growth, and there will be a discussion of proper methods of treatment, both surgical and radiological. There will be a review of the tumors that involve the nasopharynx and a discussion of their symptoms as well as the treatment of these tumors. An attempt will be made to differentiate tumors of the tonsil and the base of the tongue.

Concerning malignant disease of the larynx, there will be a discussion of the types of tumors, the location of the growth, and how treatment depends upon each individual case as to the type of growth, as to its location, and as to its extent, and somewhat upon the age of the patient. The various types of treatment of malignant diseases of the larynx will be presented.

**COURSE 365**

Room 739 Periods T-3, W-3, Th-3, F-3

**HARRY P. SCHENCK, M.D.**
Philadelphia, Pa.

**The Management of Tympanic Membrane Perforations**

Perforations in the tympanic membrane which fail to heal spontaneously may or may not respond to efforts made to bring about closure. A distinction is made between those perforations amenable to treatment and those resistant to any form of intervention. Failure to accomplish closure is inevitable in the presence of continuing infection in the middle ear. Reasonably accurate predictions of the result of treatment can be made on the basis of etiology, location, and size of the lesion. The technics most likely to succeed are described and the materials employed are discussed in detail.

**COURSE 366**

Room 736 Periods W-3, Th-3, F-3

**LYLE M. SELLERS, M.D.**
Dallas, Tex.

**Headaches of Otorhinologic Origin**

The otorhinologist who is able to treat headache successfully is indeed fortunate. A strikingly high percentage of headaches are of otorhinologic origin. Because most of the patients so afflicted surely find their way to his hands, the otorhinologist occupies a highly strategic and advantageous position in the fight against this affliction if he is able to recognize and treat these headaches.

The course here offered is designed to prepare the otorhinologist to recognize headaches of otorhinologic origin. Since there are a number of definite causes of such headaches, and since each cause produces a definite type of headache, each cause and its clinical manifestations will be correlated. The means of making definite differential diagnoses and the application of specific therapeutic measures to each type of case will be presented and illustrated with applicable case histories. Histamine-type headache will be discussed similarly and its control described in detail.

The course will be well outlined and will be illustrated with lantern slides.

**COURSE 367**

Room 733 Periods T-3, W-3

**JOHN J. SHEA, M.D.**
Memphis, Tenn.

**Modern Treatment of Infections of the Ear, Nose, and Throat**

The modern treatment as used in the office today has changed from the solvent packs to the use of antibiotics.

The technic of sinus irrigations will be considered.

The method of penicillin inhalation will be discussed.

The frequency of fungus infections of the external ear and its treatment will be outlined.

**COURSE 368**

Room 733 Periods Th-3, F-3

**JOHN J. SHEA, M.D.**
Memphis, Tenn.

**The Therapy of Headache**

The chief complaint of headache alone or associated with vertigo is the reason that the majority of patients seek relief from the otolaryngologist. The therapy of

histamine and nicotinic acid is the hopeful remedy for these individuals.

The therapy of histamine and nicotinic acid has rendered marvelous relief to many patients suffering from dysfunctions of the auditory nerve, manifested by deafness, tinnitus or vertigo, and to headache of the nonsurgical variety. These drugs are vasodilators and as such relieve vasoconstriction affecting the auditory nerve and cranial vessels. Nicotinamide does not produce the cerebral vasodilation like nicotinic acid, and the modus operandi of nicotinic acid accomplishing vasodilation is a result of the chemical combustion when it becomes an amide.

---

**COURSE 369**

Room 740 Periods T-1, W-1, Th-1, F-1

**J. D. SINGLETON, M.D.**
Dallas, Tex.

**Chemotherapy in the Field of Otolaryngology: Its Present Status**

The course will consist of a discussion of penicillin, streptomycin, and the sulfonamides as related to the field of otolaryngology in general. Their use in some of the more common, severe infections encountered in the specialty will be considered in more detail.

Penicillin and streptomycin will be discussed under such headings as:

1. Properties.
2. Pharmacology.
   a. Dosage.
   b. Absorption.
   c. Distribution in body.
   d. In cerebrospinal fluid.
   e. Excretion.
   f. Toxic effects.
3. Indications for use.
4. Methods of administration.
5. Causes of failures.

Basic general principles for guidance in the successful use of these drugs will be stressed.

---

**COURSE 370**

Room 754 Periods T-3, W-3, Th-3, F-3

**MAURICE F. SNITMAN, M.D.**
Chicago, Ill.

**Surgical Anatomy of the Neck**

I. Introduction:
   Head position
II. Skin and platysma myoides
III. Superficial veins
IV. Fasciae and loose connective tissue
   Viscero-vertebral angle
   Deep neck infections
V. Triangles of the neck
VI. Submental triangle.
   Thyroglossal cyst and fistula
VII. Submaxillary (digastric) triangle
   Removal of submaxillary salivary gland
   Ligation of lingual artery
   Approach to parapharyngeal abscess
VIII. Carotid Triangle
   Ligation of carotid arteries and internal jugular vein
   Removal of bronchial cyst
IX. Parotid region
   Removal of mixed tumor of parotid
X. Muscular triangle
   Tracheotomy
   Laryngofissure
   Laryngectomy
   Removal of pharyngeal diverticulum

---

**COURSE 371**

Room 731 Periods T-1, W-1, Th-1

**LaVERNE B. SPAKE, M.D.**
Kansas City, Mo.

**The Submucous Operation**

I. Anatomy
   a. Mucous membrane
   b. The septum—specimen, slides
   c. The blood and nerve supply
II. The type of deviation
   a. Congenital abnormalities in the new born
   b. Unilateral and bilateral choanal occlusion.
   c. Injuries
      1. Cartilaginous
      2. Bony
      3. Infection—abscess—gumma
III. Indication for submucous operation
   a. Age of the patient
   b. Symptomology
   c. In presence of purulent sinusitis
IV. The anesthesia
   a. Complete physical
      1. Blood pressure
      2. Urine analysis
      3. Coagulation and bleeding time
   b. Hospitalization
   c. Local or general
   d. Morphine, atropine 1/200, scopolamine 1/250
   e. Nembutal
      Antidote for cocaine and derivatives
   f. Novocain

g. Cocaine and andrenalin
   1. Air-way, amytal, intravenous
   2. Ether, oxygen, artificial respiration
   3. Coramine

h. Rate of absorption
   Adrenalin first

V. Operation
   a. Position of the patient
   b. Surgical preparation of patient
   c. Instruments used
   d. Type of incision
   e. The time element
   f. Perforation
   g. Nasal packs, splints, etc.
   h. The immediate postoperative treatment
   i. Postoperative care

---

**COURSE 372**

Room 733 Periods T-1, W-1, Th-1, F-1

**C. L. STRAITH, M.D.**
Detroit, Mich.
BY INVITATION
and
**WAYNE B. SLAUGHTER, M.D.**
Chicago, Ill.
BY INVITATION

**Plastic Surgery of Facial Injuries**

The treatment of all acutely injured persons must follow a rather definite course, the purpose of which is to give a maximum of benefit to the patient. To obtain this maximum benefit requires adherence to certain fundamental surgical principles, outlined as follows (illustrated with Kodachrome slides and movies):

1. Treatment of impending shock or actual shock.
2. Prevention of additional contamination and trauma to the wound. (Use of antiseptics.)
3. Estimation of the patient's general condition and recognition of the presence of multiple injuries.
4. Cleansing of the wound. (Prevention of "tattoo" marks, removal of foreign bodies, etc.)
5. Surgical care. In plastic surgery of the face certain problems are encountered peculiar to this region. These points will be stressed, together with emphasis on fixation of fractures, suturing of wounds and reconstructive procedures for recent or old injuries.
6. After care.

---

**COURSE 373**

Room 777 Periods Th-2, F-2

**JOSEPH A. SULLIVAN, M.D.**
Toronto, Can.

**Recent Advances in Surgical Repair of the Facial Nerve**

The surgical anatomy of the facial nerve within the temporal bone and parotid gland will be briefly described and demonstrated by means of slides and specimens.

The common sites for injuries to the facial nerve in the mastoid operation will be discussed, based on two hundred and fifty facial nerve grafting operations over a period of eleven years. The question of when and when not to use a nerve graft will be described.

The advances in repair of peripheral nerves as a result of experiences in this war and its application to the injuries to the facial nerve will be considered, particularly in respect to sutures versus plasma.

A brief view of Bell's palsy and what we can do for that particular lesion.

Finally, the maximum results we can expect from this advance in otologic surgery.

---

**COURSE 374**

Room 776 Periods T-3, W-3, Th-3

**THOMAS G. TICKLE, M.D.**
New York, N. Y.

**Clinical Observations in the Surgical Treatment of Facial Paralysis Over a Period of Fifteen Years**

History of experiments in facial nerve anastomoses from earliest recorded cases up to Ballance-Duel research. Signs and symptoms of facial paralysis—including electrical testing—in cases of injury and so-called "Bell's palsy." Selection of cases for nerve-grafting, end-to-end anastomosis and decompression. Present-day operative technic. Description of approved therapy and postoperative care. When and how to observe functional return. Report on end-results—slight facial tic and associated movements. Re-education. (Moving pictures.)

---

**COURSE 375**

Room 777 Periods T-2, W-2

**EDWARD H. TRUEX, JR., M. D.**
Hartford, Conn.

**A Simple and Safe Technic for Taking Earmold Impressions**

The impression material consists of a powdered plastic and a liquid. They are mixed in the proper proportions at the time of the operation and kneaded into the external canal and auricle which require no preliminary preparation. In ten min-

utes the mixture becomes rubbery in consistency and is extracted easily from the canal.

The finished impression can be transported to the dental technician for immediate processing or, if delay is necessary, a permanent positive can be made from the impression within a few minutes.

The method is simple in that it requires no unusual apparatus or space. It is safe, having been used at Deshon General Hospital about 5,000 times without a single complication or untoward effect.

A discussion and practical demonstration of the technic will be presented.

---

**COURSE 376**

Room 782 Periods T-3, W-3

**GABRIEL TUCKER, M.D.**
Philadelphia, Pa.

**Obstructive Dyspnea and Tracheotomy**

*Definition.* For the purposes of this discussion, obstructive dyspnea may be defined as "dyspnea due to mechanical obstruction of the airway through the pharynx, larynx and tracheobronchial tree.

*Etiology.* Obstruction of the airway may be caused by foreign body, inflammatory, neoplastic or traumatic lesions of the pharynx, larynx and tracheobronchial tree. The occurrence of obstructive dyspnea in the presence of such lesions creates an urgent indication for relief of obstruction and restoration of the airway. Where mechanical removal of obstruction by direct laryngoscopy or bronchoscopy will restore the airway, short-circuiting of the air-way by tracheotomy may not be required. Where this is not practical, however, the indication for tracheotomy becomes urgent.

*Tracheotomy.* Tracheotomy is one of the oldest operative procedures known to surgery. In spite of this fact, the indications for tracheotomy in obstructive dyspnea are frequently unrecognized. The modern methods of temporary relief of obstructive dyspnea, and the prevention of mediastinal complications by the use of the bronchoscope or the Mosher Life-Saver during tracheotomy, are not fully appreciated. Tracheotomy, indications, technic and after care will be demonstrated, with colored motion pictures and lantern slides.

---

**COURSE 377**

Room 777 Periods T-1, W-1

**O. E. VAN ALYEA, M.D.**
Chicago, Ill.

**Embryology of the Ear, Nose, and Throat**

I. Embryonic Stage:

During the first 8 weeks of intrauterine life, the structures which compose the ear, nose, and throat are in the formative stage of development. The major changes which take place at this time will be considered step by step by means of lantern slides and wax reconstructions.

II. Fetal Stage:

The changes which occur from the 8th week on are of especial interest to the otolaryngologist. In this course considerable time will be devoted to the study of serial sections made from fetal heads of various ages with special emphasis on those periods which deal with the origin and development of the nasal sinuses.

III. Congenital Defects:

These occur largely as a result of growth defects in the early formative stages of the embryo. Anomalies of the various types encountered in clinical practice will be studied by means of lantern slide projections of charts, x-rays, photographs and specimens from the cadaver.

---

**COURSE 378**

Room 777 Periods Th-1, F-1

**O. E. VAN ALYEA, M.D.**
Chicago, Ill.

**Management of Chronic Sinusitis**

Persistence of a sinus infection implies inadequate drainage. Factors contributing to drainage impairment may be local or systemic. The local factors may be anatomic or pathologic in nature and in some cases the sinusitis is maintained by a combination of contributing causes.

The goal of the rhinologist is to eliminate the causative factors while treating the infection. Most cases respond to simple measures, such as infraction of the middle turbinate, or the removal of a few polyps along with the control of allergy. In those cases which fail to respond readily to carefully directed treatment there is usually some overlooked contributing systemic or local factor. In certain cases thickened mucosa lines the sinus osteum to such an extent as to occlude, or nearly occlude, the drainage outlet and extra drainage openings (windows) may be required.

The radical removal of sinus mucosa in the treatment of sinusitis is ill advised and should be instituted with the knowledge that only temporary relief may be expected and that a return of the symptoms, even more pronounced than formerly, may result.

**COURSE 379**

Room 724 Period T-3
Room 716 Period Th-2
Room 732 Period F-3

**RICHARD WALDAPFEL, M.D.**
Grand Junction, Colo.

**Common Diseases of the Larynx**

Some important hints about examination, diagnosis, and differential diagnosis. Essentials in the treatment of the mucous membrane of larynx and trachea. Intralaryngeal methods of treatment. Inhalation, instillation, insufflation and prescribed drugs. Acute and chronic laryngitis. Edema. Croup and pseudocroup. Tuberculosis. Papilloma. Early carcinoma. Hoarseness in children and in adults. Do's and don'ts.

---

**COURSE 380**

Room 777 Periods T-3, W-3, Th-3, F-3

**THEODORE E. WALSH, M.D.**
St. Louis, Mo.

**Treatment and Prevention of Acute Rhinitis**

Acute rhinitis is considered under two heads:

a. Infectious.
b. Vasomotor.

*Infectious:*

Pathogenesis of infectious rhinitis is discussed in relation to the defense mechanisms. Viral and bacterial infections are compared:

Rationale of preventive therapy is outlined and experimental and clinical data presented.

The pathology of infectious rhinitis is discussed and therapeutic measures appropriate to the pathologic stages are outlined.

*Vasomotor:*

Vasomotor rhinitis in relation to allergy and endocrine imbalance is discussed briefly.

---

**COURSE 381**

Room 712 Period T-3
Room 727 Period W-3
Room 788 Period F-3

**WILLIAM C. WARREN, JR., M.D.**
Atlanta, Ga.

**Practical Therapeutics in Ear, Nose and Throat**

In this conference emphasis will be placed upon the treatment of office patients who present everyday signs and symptoms.

Impaired breathing will be discussed at length with special attention being called to the role the inferior turbinates play. A method of correcting same will be outlined. Likewise, the ever-present complaint of postnasal drip will get a lay. Also, nasal hemorrhages with their respective problems.

In the throat, a demonstration as to the best location for incising a peritonsillar abscess will be given. Listing the signs and symptoms which tell us it should be opened.

External otitis, an ever present disease, will be dealt with, and the use of Vinegar of Cantharides stressed.

---

**COURSE 382**

Room 795 Periods T-3, W-3, Th-3, F-3

**HENRY L. WILLIAMS, M.D.**
Rochester, Minn.

**Differentiation, Cause and Treatment of Certain Common Types of Headaches**

An attempt at a fresh approach to the differential diagnosis and treatment of headache will be made by considering the pain-sensitive structures in the head and the manner in which they may be stimulated in certain conditions. The more difficult differential diagnoses, especially those of major importance to the otologist and rhinologist, will be outlined. The treatment of those headaches which appear to be due to physical allergy will be stressed because they have suffered from considerable therapeutic neglect and they are of the type apt to cause the patient to consult the otorhinologist.

---

**COURSE 383**

Room 738 Periods T-3, W-3

**WAITMAN F. ZINN, M.D.**
Baltimore, Md.

**Diagnosis and Treatment of Cancer of the Larynx**

The early diagnosis of cancer of the larynx is of the utmost importance.

Hoarseness is the most predominant symptom. The diagnostic procedure includes the following:

1. Complete history.
2. Indirect examination.
3. External examination of neck for palpable glands.
4. X-ray of larynx.
5. Laboratory check-up.
6. Direct laryngoscopy for biopsy; histological report.
7. Complete physical examination before surgery.

The treatment will depend upon the extent of the disease. Intrinsic cases are operable and extrinsic cases inoperable.

Operative cases:

Anesthesia.

Type of surgical technic (explanation of steps).

Methods of closure of hypopharynx.

Type of drainage.

Application of proper dressing.

Inoperable cases:

Deep x-ray therapy should be considered for all cases in which surgery is contraindicated.

Consideration of best type of pseudovoice; methods of teaching.

**The Following are Condensed Schedules of Courses Previously Described.**

## CONTINUOUS COURSES — OPHTHALMOLOGY

| COURSE | SPEAKERS | TITLE | PERIODS USED |
|---|---|---|---|
| 1 or 2 | Allen | Bacteriology | 2 PERIODS M-4, M-5 or T-4, T-5 |
| 3 | Brown, et al. | Eye Muscles | 8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5 |
| 4 or 5 | Copeland | Streak Retinoscopy | 2 PERIODS W-4, W-5 or Th-4, Th-5 |
| 6 or 7 | Cordes | Acquired and Congenital Cataracts | 2 PERIODS M-5, M-6, or T-5, T-6 |
| 8 | Dunnington | Surgical Treatment of Heterophoria and Heterotropia | 4 PERIODS M-5, T-5, W-5, Th-5 |
| 9 | Falls | Heredity | 2 PERIODS Th-5, Th-6 |
| 10 | Fralick and Henderson | Anatomy of the Orbit | 4 PERIODS M-4, M-5, T-4, T-5 |
| 11 | Friedenwald and Hughes | Physiology and Pathology of Cornea in Relation to Chemical Burns | 3 PERIODS M-4, M-5, M-6 |
| 12 or 13 | Givner | Ophthalmological Bacteriology | 2 PERIODS W-4, W-5 or Th-4, Th-5 |
| 14 or 15 | Grant | Practical Orthoptics | 2 PERIODS M-5, M-6 or T-5, T-6 |
| 16 or 17 | Hardy and Rand | Color Vision Testing | 2 PERIODS M-4, M-5 or T-4, T-5 |
| 18 | Hardy, et al. | Orthoptics | 9 PERIODS T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6 |
| 19 or 20 | Jardon and Noelle | Plastic Prosthesis | 2 PERIODS M-4, M-5 or T-4, T-5 |
| 21 | Koch | Systemic Hypertensive Disease | 3 PERIODS M-6, T-6, W-6 |
| 22 or 23 | Kronfeld | Gonioscopy | 2 PERIODS W-4, W-5 or Th-4, Th-5 |
| 24 | Kuhn, et al. | Industrial Ophthalmology | 12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th.-4, Th.-5, Th.-6 |
| 25 | Lancaster and Linksz | Aniseikonia | 4 PERIODS W-5, W-6, Th-5, Th-6 |
| 26 or 27 | Lloyd | Ophthalmoscopy | 2 PERIODS M-5, M-6 or T-5, T-6 |
| 28 | Lyle | Neuro-ophthalmology | 3 PERIODS T-4, W-4, Th.-4 |
| 29 | Masters, et al. | Slit Lamp Biomicroscopy | 12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th.-4, Th.-5, Th.-6 |
| 30 | McLean and Guyton | Cataract Surgery | 8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5 |
| 31 | Meyer, et al. | Glaucoma | 8 PERIODS M-5, M-6, T-5, T-6, W-5, W-6, Th-5, Th-6 |
| 32 | Payne | Advanced Eye Pathology | 4 PERIODS M-6, T-6, W-6, Th-6 |
| 33 or 34 | Pischel | Retinal Detachment | 2 PERIODS M-4, M-5 or T-4, T-5 |
| 35 | Rones and Stow | Clinical Pathology | 12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6 |
| 36 or 37 | Rucker and Love | Lesions in Region of Chiasm | 2 PERIODS M-4, M-5 or T-4, T-5 |
| 38 or 39 | Scobee | Routine Check of Ocular Muscles | 2 PERIODS W-5, W-6 or Th-5, Th-6 |
| 40 | Sheard | Applied Physiological Optics | 4 PERIODS M-6, T-6, W-6, Th-6 |
| 41 | Theobald, et al. | Histopathology — Elementary | 12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6 |
| 42 | Woods and Owens | Medical Ophthalmology | 4 PERIODS M-6, T-6, W-6, Th-6 |

## SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Monday

**Key M - 4 Period: 2:00 p.m. to 3:00 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 101 | Alvaro | Uveitis |
| 104 | Bahn | Vitamins, Hormones and Enzymes in Ophth. |
| 106 | Baker | Neuralgias of the Face |
| 107 | Barkan | Goniotomy for Congenital Glaucoma |
| 117 | Bruner | Visual Aids |
| 119 | Burch, F. E. | Glaucoma — Choice of Surgery |
| 122 | Castroviejo | Lesions of Cornea |
| 141 | Gipner | Recent Advances in Ophth. |
| 146 | Hitz | Visual Failures in School Children |
| 149 | Hughes | Surgical Repair of Lids |
| 151 | Jobe | Physiological Optics |
| 156 | Kirby | Procedures for Cataract Surgery |
| 159 | Koenig | Ocular Tuberculosis |
| 168 | McAlester | Pigmentary Retinal Changes |
| 171 | Meek | Surgical Anatomy of Orbit |
| 188 | Simpson | Treatment of Myopia |
| 192 | Spaeth | Congenital Ptosis |
| 198 | Struble | Intraocular Foreign Bodies |
| 199 | Swan | Action of Drugs on Iris and Ciliary Body |

**Key M - 5 Period: 3:15 p.m. to 4:15 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 105 | Baird | Vitamins & Adjuncts to Diet |
| 108 | Beach | Therapeutic Value of Refraction |
| 112 | Billingsley | Headaches, Head Pains of Ocular Origin |
| 121 | Casten | Practical Perimetry |
| 132 | Dickey | Levator Paralysis |
| 135 | Gailey | Complications, Treatment in Cataract Surgery |
| 145 | Hilgartner | Treatment of Corneal Opacities |
| 162 | Lehrfeld | Ocular Manifestations of Allergy |
| 169 | McCaslin | Refraction of Children's Eyes |
| 176 | Paton | Corneal Surgery |
| 177 | Peck | Sight-Saving Classes for Poor-Sighted |
| 189 | Smith | Presbyopia |
| 201 | Tassman | Chemotherapy |
| 206 | Weisser | Diagnosis & Treatment of Iritis |

**Key M - 6 Period: 4:30 p.m. to 5:30 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 102 | Alvis | Operations for Glaucoma |
| 103 | Atkinson | Trends in Cataract Surgery |
| 111 | Berliner | Slit Lamp Microscopy |
| 116 | Brown | Focal Infection |
| 129 | Cutler | Reconstruction of Orbit |
| 136 | Gemeroy | External Diseases of Eye |
| 138 | Gibson | End Results in Retinal Detachment |
| 142 | Greear | Rehabilitation of Blind |
| 150 | Irvine | Manifest Refraction |
| 163 | Lemoine | Vitreous Opacities |
| 173 | Motto | Treatment of Muscle Problem |
| 175 | O'Rourke | Refraction: Cycloplegic and Manifest |
| 186 | Scheie | Injury to Iris & Ciliary Body |
| 187 | Schwartz | Mechanics of Lens Extraction |
| 202 | Thomas | Diabetes and Blood Dyscrasias |
| 203 | Town | Contact Lens Fittings |
| 204 | Townes | Hyperphoria |
| 210 | Wolpaw | Hyperopia |

## SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Tuesday

**Key T - 4 Period: 2:00 p.m. to 3:00 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 101 | Alvaro | Uveitis |
| 104 | Bahn | Vitamins, Hormones & Enzymes in Ophth. |
| 106 | Baker | Neuralgias of the Face |
| 107 | Barkan | Goniotomy for Congenital Glaucoma |
| 117 | Bruner | Visual Aids |
| 119 | Burch, F. E. | Glaucoma — Choice of Surgery |
| 123 | Castroviejo | Lesions of Cornea |
| 141 | Gipner | Recent Advances in Ophth. |
| 146 | Hitz | Visual Failures in School Children |
| 149 | Hughes | Surgical Repair of Lids |
| 151 | Jobe | Physiological Optics |
| 159 | Koenig | Ocular Tuberculosis |
| 168 | McAlester | Pigmentary Retinal Changes |
| 171 | Meek | Surgical Anatomy of Orbit |
| 188 | Simpson | Treatment of Myopia |
| 193 | Spaeth | Acquired Ptosis |
| 198 | Struble | Intraocular Foreign Bodies |
| 199 | Swan | Action of Drugs on Iris and Ciliary Body |

**Key T - 5 Periods: 3:15 p.m. to 4:15 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 105 | Baird | Vitamins & Adjuncts to Diet |
| 108 | Beach | Therapeutic Value of Refraction |
| 112 | Billingsley | Headaches, Head Pains of Ocular Origin |
| 121 | Casten | Practical Perimetry |
| 132 | Dickey | Levator Paralysis |
| 135 | Gailey | Complications, Treatment in Cataract Surgery |
| 145 | Hilgartner | Treatment of Corneal Opacities |
| 162 | Lehrfeld | Ocular Manifestations of Allergy |
| 169 | McCaslin | Refraction of Children's Eyes |
| 176 | Paton | Corneal Surgery |
| 177 | Peck | Sight-Saving Classes for Poor-Sighted |
| 189 | Smith | Presbyopia |
| 201 | Tassman | Chemotherapy |
| 206 | Weisser | Diagnosis & Treatment of Iritis |

**Key T - 6 Period: 4:30 p.m. to 5:30 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 102 | Alvis | Operations for Glaucoma |
| 103 | Atkinson | Trends in Cataract Surgery |
| 111 | Berliner | Slit Lamp Microscopy |
| 116 | Brown | Focal Infection |
| 129 | Cutler | Reconstruction of Orbit |
| 136 | Gemeroy | External Diseases of Eye |
| 138 | Gibson | End Results in Retinal Detachment |
| 142 | Greear | Rehabilitation of Blind |
| 150 | Irvine | Manifest Refraction |
| 163 | Lemoine | Vitreous Opacities |
| 173 | Motto | Treatment of Muscle Problem |
| 175 | O'Rourke | Refraction: Cycloplegic and Manifest |
| 186 | Scheie | Injury to Iris & Ciliary Body |
| 187 | Schwartz | Mechanics of Lens Extraction |
| 202 | Thomas | Diabetes and Blood Dyscrasias |
| 203 | Town | Contact Lens Fittings |
| 204 | Townes | Hyperphoria |
| 210 | Wolpaw | Hyperopia |

## SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Wednesday

**Key W - 4 Period: 2:00 p.m. to 3:00 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 110 | Berke | Exophthalmos |
| 113 | Boeder | Aniseikonia |
| 114 | Bogart | Eye Shortening Procedures |
| 120 | Carroll | Optic Neuritis |
| 124 | Castroviejo | Lesions of Cornea |
| 130 | Danielson | Diagnosis of Retinoblastoma |
| 144 | Haessler | Divergence Excess |
| 148 | Hughes | Photography — Motion Pictures |
| 153 | Judd | Optic Neuritis |
| 160 | Krause | Biochemistry of Eye |
| 167 | Marshall | Chronic Infections of Lid |
| 178 | Perera | Retinal Detachment |
| 183 | Riser | Fundus Lesions |
| 184 | Roper | Chemotherapy |
| 185 | Rychener | External Diseases of Eye |
| 194 | Spaeth | Ectropion & Entropion |
| 197 | Stine | Retinal Detachment |
| 200 | Tait | Accommodation & Convergence Reflex Mechanisms |
| 205 | Veasey | Refraction |
| 208 | Whitney | Lacrimal Apparatus |

**Key W - 5 Period: 3:15 p.m. to 4:15 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 115 | Boyes | Questions & Answers in Surgical Cases |
| 126 | Clark | Preparation of Patient for Cataract Surgery |
| 127 | Crisp | Testing Astigmatism, Dials & Cross Cylinders |
| 131 | Davies | Optic Discs |
| 133 | Falls | Embryology |
| 139 | Gifford | Dacryocystorhinostomy |
| 143 | Gump | Iridocyclitis |
| 155 | Keyes | Retinal Arteriovascular Disease |
| 157 | Knighton | Analysis of Glaucoma Cases |
| 164 | Lepard | Juvenile & Soft Cataract |
| 170 | McDonald | Tinted Lenses |
| 172 | Morrison | Lacrimal Glands |
| 179 | Pfeiffer | Value of X-ray Studies |
| 181 | Regan | Astigmatic Dials in Refraction |
| 196 | Sterling | Lens—Practical Optics |
| 209 | Wiener | Points in Surgery |

**Key W - 6 Period: 4:30 p.m. to 5:30 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 109 | Bellows | Lens Changes |
| 118 | Burch, E. P. | Anesthesia in Ophth. |
| 128 | Cushman | Endocrine Disorders |
| 134 | Fink | Oblique Muscles |
| 137 | Gemeroy | Anterior Segment Photography |
| 140 | Gillette | Chronic Uveitis |
| 147 | Hoffman | Infectious Lesions of Lid |
| 152 | Johnson | Pigmented Lesions of Choroid |
| 154 | Katz | Interpreting Field Defects |
| 161 | Lebensohn | Physiology of Vision |
| 165 | Mangold | Fitting of Glasses |
| 166 | Mann | Refraction of Cataract Patient |
| 174 | Nethercut | Chorioretinitis |
| 180 | Ravdin | Psychiatric Problems |
| 182 | Richardson | Office Management |
| 190 | Snell | Medicolegal Ophth. |
| 191 | Snydacker | Vertical Phoria |
| 207 | Whalman | Traumatic Lesions of Cornea |

## SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Thursday

**Key Th.- 4 Period: 2:00 p.m. to 3:00 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 110 | Berke | Exophthalmos |
| 113 | Boeder | Aniseikonia |
| 114 | Bogart | Eye Shortening Procedures |
| 120 | Carroll | Optic Neuritis |
| 125 | Castroviejo | Lesions of Cornea |
| 130 | Danielson | Diagnosis of Retinoblastoma |
| 144 | Haessler | Divergence Excess |
| 153 | Judd | Optic Neuritis |
| 158 | Koch | Fundus Photography |
| 160 | Krause | Biochemistry of Eye |
| 167 | Marshall | Chronic Infections of Lid |
| 178 | Perera | Retinal Detachment |
| 183 | Riser | Fundus Lesions |
| 184 | Roper | Chemotherapy |
| 185 | Rychener | External Diseases of Eye |
| 195 | Spaeth | Scar Tissue Resections |
| 197 | Stine | Retinal Detachment |
| 200 | Tait | Accommodation & Convergence Reflex Mechanisms |
| 205 | Veasey | Refraction |
| 208 | Whitney | Lacrimal Apparatus |

**Key Th.- 5 Period: 3:15 p.m. to 4:15 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 115 | Boyes | Questions & Answers in Surgical Cases |
| 126 | Clark | Preparation of Patient for Cataract Surgery |
| 127 | Crisp | Testing Astigmatism, Dials & Cross Cylinders |
| 131 | Davies | Optic Discs |
| 139 | Gifford | Dacryocystorhinostomy |
| 143 | Gump | Iridocyclitis |
| 155 | Keyes | Retinal Arteriovascular Disease |
| 157 | Knighton | Analysis of Glaucoma Cases |
| 164 | Lepard | Juvenile & Soft Cataract |
| 170 | McDonald | Tinted Lenses |
| 172 | Morrison | Lacrimal Glands |
| 179 | Pfeiffer | Value of X-ray Studies |
| 181 | Regan | Astigmatic Dials in Refraction |
| 196 | Sterling | Lens—Practical Optics |
| 209 | Wiener | Points in Surgery |

**Key Th.- 6 Period: 4:30 p.m. to 5:30 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 109 | Bellows | Lens Changes |
| 118 | Burch, E. P. | Anesthesia in Ophth. |
| 128 | Cushman | Endocrine Disorders |
| 134 | Fink | Oblique Muscles |
| 140 | Gillette | Chronic Uveitis |
| 147 | Hoffman | Infectious Lesions of Lid |
| 152 | Johnson | Pigmented Lesions of Choroid |
| 154 | Katz | Interpreting Field Defects |
| 161 | Lebensohn | Physiology of Vision |
| 165 | Mangold | Fitting of Glasses |
| 166 | Mann | Refraction of Cataract Patient |
| 174 | Nethercut | Chorioretinitis |
| 180 | Ravdin | Psychiatric Problems |
| 182 | Richardson | Office Management |
| 191 | Snydacker | Vertical Phoria |
| 207 | Whalman | Traumatic Lesions of Cornea |

# CONTINUOUS COURSES — OTOLARYNGOLOGY

| COURSE | SPEAKERS | TITLE | PERIODS USED |
|---|---|---|---|
| 51 or 52 | Bordley | Nasopharynx, Eustachian Tube, Middle Ear | 2 PERIODS T-2, T-3 or W-2, W-3 |
| 53 or 54 | Brown | Facio-Maxillary Surgery | 2 PERIODS T-1, T-2 or W-1, W-2 |
| 55 | Carhart | Audiometric Technic; Fitting of Hearing Aids | 3 PERIODS T-1, W-1, Th-1 |
| 56 | Clerf | Causes of Hoarseness — Treatment | 3 PERIODS T-3, W-3, Th-3 |
| 57 | Converse | Plastic Surgery of the Nose | 6 PERIODS T-1, T-2, W-1, W-2, Th-1, Th-2 |
| 58 or 59 | Cullen | Anesthesia in Otolaryngic Surgery | 2 PERIODS Th-2, Th-3 or F-2, F-3 |
| 60 or 61 | Fishbein | Medical Writing | 2 PERIODS T-1, T-2 or W-1, W-2 |
| 62 | Fowler, E. | Histology of the Ear | 6 PERIODS T-1, T-2, W-1, W-2, Th-1, Th-2 |
| 63 or 64 | Fowler, W. | Blood Dyscrasias in Otolaryngology | 2 PERIODS T-2, T-3 or W-2, W-3 |
| 65 or 66 or 67 or 68 | Huffman | Plastic Reconstruction of Nasal Septum | 2 PERIODS T-2, T-3 or W-2, W-3 or Th-2, Th-3 or F-2, F-3 |
| 69 | Jackson | Surgery of Larynx and Hypopharynx | 3 PERIODS T-1, W-1, Th-1 |
| 70 or 71 or 72 or 73 | Lempert | Modern Temporal Bone Surgery | 2 PERIODS T-2, T-3 or W-2, W-3 or Th-2, Th-3 or F-2, F-3 |
| 74 or 75 or 76 or 77 | Mueller | Practical Audiometry | 2 PERIODS T-1, T-2 or W-1, W-2 or Th-1, Th-2 or F-1, F-2 |
| 78 or 79 | Mysel | Labyrinthitis Secondary to Ear Suppurations | 2 PERIODS Th-1, Th-2 or F-1, F-2 |
| 80 or 81 or 82 or 83 | Orton | Surgery of the Neck | 2 PERIODS T-1, T-2 or W-1, W-2 or Th-1, Th-2 or F-1, F-2 |
| 84 or 85 or 86 or 87 | Scheldrup | Anatomy of the Neck | 2 PERIODS T-2, T-3 or W-2, W-3 or Th-2, Th-3 or F-2, F-3 |
| 88 or 89 | Turchik | Plastic Reconstruction of the Nasal Septum | 2 PERIODS W-2, W-3 or Th-2, Th-3 |

## SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Tuesday

**Key T-1 Period: 9:00 a.m. to 10:00 a.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 301 | Anthony | Salivary Glands and Ducts |
| 304 | Boies | Problems in Otology |
| 306 | Burchell | Temporal Bone |
| 309 | Cone | Direct Laryngoscopy |
| 320 | Farrior | Ear Surgery |
| 321 | Furstenberg | Neurological Considerations in Rhinolaryngology |
| 324 | Gilliland | Sinusitis in Children |
| 325 | Goodyear | Neck Infections |
| 327 | Hansel | Allergy of Nose & Paranasal Sinuses |
| 333 | Holinger & Cassels | Anesthesia for Peroral Endoscopy |
| 336 | Hunnicutt | Histopathology in Office Practice |
| 344 | Maxwell | Surgery of the Parotid Gland |
| 346 | McLaurin | Radical Sinus Surgery |
| 348 | McQuiston | Deep Infections of the Neck |
| 351 | Mosher | Brain Abscess from Infections of the Skull |
| 355 | Pressman | Bronchoscopy & Laryngeal Surgery |
| 358 | Rawlins | Allergic Sinusitis |
| 361 | Robb | Procedures in Office Practice |
| 369 | Singleton | Chemotherapy in Otolaryngology |
| 371 | Spake | The Submucous Operation |
| 372 | Straith & Slaughter | Plastic Surgery of Facial Injuries |
| 377 | Van Alyea | Embryology of ENT |

**Key T-2 Period: 10:15 a.m. to 11:15 a.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 302 | Birsner | Surgical Anatomy of the Skull |
| 303 | Blaisdell | Bronchography |
| 313 | Davis | Problems in Surgery of the Face |
| 315 | Dearmin | Chronic Mastoiditis |
| 317 | Eagle | Uncommon Otolaryngic Syndromes |
| 319 | Erich | Fractures of Maxilla & Malar Bones |
| 326 | Hall | Acute Neck Infections |
| 334 | Horton | Headaches and Head Pains |
| 337 | Ireland | Surgical Emergencies |
| 339 | Kinney | Office Audiometry |
| 343 | Lynch & LeJeune | Suspension Laryngoscopy |
| 349 | Miller | Tracheotomy in Children |
| 352 | Myers | Nasal Allergy |
| 354 | Perlman | Eustachian Tube Function |
| 356 | Priest | Tracheotomy |
| 357 | Proetz | Displacement Method |
| 359 | Reger | Selection of Hearing Aids |
| 362 | Robison | Maxillary Sinusitis |
| 363 | Salinger | Nasal Fractures |
| 375 | Truex | Technic for Earmold Impressions |

**Key T-3 Period: 11:30 p.m. to 12:30 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 305 | Bryant | Sphenopalatine Ganglion |
| 307 | Campbell | Differential Diagnosis of Hearing Loss |
| 308 | Canfield | Auditory Rehabilitation Clinic |
| 310 | Costen | Mandibular Joint Neuralgia |
| 329 | Hart | Clinical Aspects of Bronchiectasis |
| 331 | Hill | Management of Antro-Oral Fistulae |
| 338 | Jones | Office Procedures |
| 342 | Lundy | Choice of Anesthetics |
| 347 | McNally | Labyrinthine Tests |
| 350 | Moore | Lesions of the Mouth |
| 360 | Reger | Functional Hearing Tests |
| 365 | Schenck | Tympanic Membrane Perforations |
| 367 | Shea | Treatment of Infections |
| 370 | Snitman | Surgical Anatomy of the Neck |
| 374 | Tickle | Surgical Treatment of Facial Paralysis |
| 376 | Tucker | Obstructive Dyspnea & Tracheotomy |
| 379 | Waldapfel | Diseases of the Larynx |
| 380 | Walsh | Acute Rhinitis |
| 381 | Warren | Practical Therapeutics in ENT |
| 382 | Williams | Headache in Otolaryngology |
| 383 | Zinn | Cancer of the Larynx |

## SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Wednesday

### Key W-1 Period: 9:00 a.m. to 10:00 a.m.

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 301 | Anthony | Salivary Glands and Ducts |
| 304 | Boies | Problems in Otology |
| 306 | Burchell | Temporal Bone |
| 309 | Cone | Direct Laryngoscopy |
| 311 | Cunning | Laryngeal Tumors |
| 316 | Decker | Early Deafness |
| 320 | Farrior | Ear Surgery |
| 321 | Furstenberg | Neurological Considerations in Rhinolaryngology |
| 324 | Gilliland | Sinusitis in Children |
| 325 | Goodyear | Neck Infections |
| 327 | Hansel | Allergy of Nose & Paranasal Sinuses |
| 335 | House | Endaural Approach to Temporal Bone Surgery |
| 336 | Hunnicutt | Histopathology in Office Practice |
| 340 | Kos | Interpretation of Vestibular Test |
| 344 | Maxwell | Surgery of Parotid Gland |
| 348 | McQuiston | Deep Infections of the Neck |
| 353 | Nash | Functional Diseases of the Nose |
| 355 | Pressman | Bronchoscopy & Laryngeal Surgery |
| 358 | Rawlins | Allergic Sinusitis |
| 361 | Robb | Procedures in Office Practice |
| 369 | Singleton | Chemotherapy in Otolaryngology |
| 371 | Spake | Submucous Operation |
| 372 | Straith & Slaughter | Plastic Surgery of Facial Injuries |
| 377 | Van Alyea | Embryology of ENT |

### Key W-2 Period: 10:15 a.m. to 11:15 a.m.

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 303 | Blaisdell | Bronchography |
| 312 | Curtis | Diagnosis of Common Oral Lesions |
| 313 | Davis | Problems in Surgery of the Face |
| 315 | Dearmin | Chronic Mastoiditis |
| 317 | Eagle | Uncommon Otolaryngic Syndromes |
| 319 | Erich | Fractures of Maxilla & Malar Bones |
| 326 | Hall | Acute Neck Infections |
| 334 | Horton | Headaches and Head Pains |
| 337 | Ireland | Surgical Emergencies |
| 339 | Kinney | Office Audiometry |
| 341 | Lindsay | Vertigo |
| 343 | Lynch & LeJeune | Suspension Laryngoscopy |
| 346 | McLaurin | Radical Sinus Surgery |
| 352 | Myers | Nasal Allergy |
| 357 | Proetz | Displacement Method |
| 359 | Reger | Selection of Hearing Aids |
| 362 | Robison | Maxillary Sinusitis |
| 363 | Salinger | Nasal Fractures |
| 375 | Truex | Technic for Earmold Impressions |

### Key W-3 Period: 11:30 p.m. to 12:30 p.m.

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 305 | Bryant | Sphenopalatine Ganglion |
| 307 | Campbell | Differential Diagnosis of Hearing Loss |
| 308 | Canfield | Auditory Rehabilitation Clinic |
| 310 | Costen | Mandibular Joint Neuralgia |
| 314 | Day | Hydrops of the Labyrinth |
| 318 | Ellis | Congenital Anomalies |
| 322 | Galloway | Respiratory Obstruction, etc. |
| 328 | Harkness | Private Practice |
| 329 | Hart | Clinical Aspects of Bronchiectasis |
| 331 | Hill | Management of Antro-Oral Fistulae |
| 342 | Lundy | Choice of Anesthetics |
| 347 | McNally | Labyrinthine Tests |
| 350 | Moore | Lesions of the Mouth |
| 360 | Reger | Functional Hearing Tests |
| 365 | Schenck | Tympanic Membrane Perforations |
| 366 | Sellers | Headaches of Otorhinologic Origin |
| 367 | Shea | Treatment of Infections |
| 370 | Snitman | Surgical Anatomy of the Neck |
| 374 | Tickle | Surgical Treatment of Facial Paralysis |
| 376 | Tucker | Obstructive Dyspnea & Tracheotomy |
| 380 | Walsh | Acute Rhinitis |
| 381 | Warren | Practical Therapeutics in ENT |
| 382 | Williams | Headache in Otolaryngology |
| 383 | Zinn | Cancer of the Larynx |

## SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Thursday

**Key Th.-1 Period: 9:00 a.m. to 10:00 a.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 301 | Anthony | Salivary Glands & Ducts |
| 304 | Boies | Problems in Otology |
| 306 | Burchell | Temporal Bone |
| 309 | Cone | Direct Laryngoscopy |
| 311 | Cunning | Laryngeal Tumors |
| 316 | Decker | Early Deafness |
| 320 | Farrior | Ear Surgery |
| 321 | Furstenberg | Neurological Considerations in Rhinolaryngology |
| 325 | Goodyear | Neck Infections |
| 327 | Hansel | Allergy of Nose & Paranasal Sinuses |
| 335 | House | Endaural Approach to Temporal Bone Surgery |
| 336 | Hunnicutt | Histopathology in Office Practice |
| 340 | Kos | Interpretation of Vestibular Test |
| 344 | Maxwell | Surgery of the Parotid Gland |
| 348 | McQuiston | Deep Infections of the Neck |
| 353 | Nash | Functional Diseases of the Nose |
| 354 | Perlman | Eustachian Tube Function |
| 355 | Pressman | Bronchoscopy and Laryngeal Surgery |
| 358 | Rawlins | Allergic Sinusitis |
| 361 | Robb | Procedures in Office Practice |
| 369 | Singleton | Chemotherapy |
| 371 | Spake | Submucous Operation |
| 372 | Straith & Slaughter | Plastic Surgery of Facial Injuries |
| 378 | Van Alyea | Chronic Sinusitis |

**Key Th.-2 Period: 10:15 a.m. to 11:15 a.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 302 | Birsner | Surgical Anatomy of the Skull |
| 312 | Curtis | Diagnosis of Common Oral Lesions |
| 313 | Davis | Problems in Surgery of the Face |
| 315 | Dearmin | Chronic Mastoiditis |
| 317 | Eagle | Uncommon Otolaryngic Syndromes |
| 323 | Gill | External Otitis |
| 333 | Holinger & Cassels | Anesthesia for Peroral Endoscopy |
| 334 | Horton | Headaches and Head Pains |
| 337 | Ireland | Surgical Emergencies |
| 339 | Kinney | Office Audiometry |
| 341 | Lindsay | Vertigo |
| 343 | Lynch & LeJeune | Suspension Laryngoscopy |
| 345 | McCaskey | Neck Surgery |
| 346 | McLaurin | Radical Sinus Surgery |
| 352 | Myers | Nasal Allergy |
| 357 | Proetz | Displacement Method |
| 359 | Reger | Selection of Hearing Aids |
| 362 | Robison | Maxillary Sinusitis |
| 363 | Salinger | Nasal Fractures |
| 373 | Sullivan | Surgical Repair of Facial Nerve |
| 379 | Waldapfel | Common Diseases of the Larynx |

**Key Th.-3 Period: 11:30 p.m. to 12:30 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 307 | Campbell | Differential Diagnosis of Hearing Loss |
| 310 | Costen | Mandibular Joint Neuralgia |
| 314 | Day | Hydrops of the Labyrinth |
| 318 | Ellis | Congenital Anomalies |
| 322 | Galloway | Respiratory Obstruction, etc. |
| 328 | Harkness | Private Practice |
| 329 | Hart | Clinical Aspects of Bronchiectasis |
| 330 | Havens | Repair of Deformities of Face |
| 331 | Hill | Management of Antro-Oral Fistulae |
| 332 | Holinger | Photography |
| 338 | Jones | Office Procedures |
| 347 | McNally | Labyrinthine Tests |
| 350 | Moore | Lesions of the Mouth |
| 360 | Reger | Functional Hearing Tests |
| 364 | Schall | Cancer of Upper Respiratory Tract |
| 365 | Schenck | Tympanic Membrane Perforations |
| 366 | Sellers | Headaches of Otorhinologic Origin |
| 368 | Shea | Therapy of Headache |
| 370 | Snitman | Surgical Anatomy of the Neck |
| 374 | Tickle | Surgical Treatment of Facial Paralysis |
| 380 | Walsh | Acute Rhinitis |
| 382 | Williams | Headache in Otolaryngology |

## SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Friday

**Key F-1 Period: 9:00 a.m. to 10:00 a.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 301 | Anthony | Salivary Glands and Ducts |
| 304 | Boies | Problems in Otology |
| 311 | Cunning | Laryngeal Tumors |
| 316 | Decker | Early Deafness |
| 320 | Farrior | Ear Surgery |
| 321 | Furstenberg | Neurological Considerations in Rhinolaryngology |
| 324 | Gilliland | Sinusitis in Children |
| 327 | Hansel | Allergy of Nose & Paranasal Sinuses |
| 335 | House | Endaural Approach to Temporal Bone Surgery |
| 336 | Hunnicutt | Histopathology in Office Practice |
| 340 | Kos | Interpretation of Vestibuiar Test |
| 348 | McQuiston | Deep Infections of the Neck |
| 353 | Nash | Functional Diseases of the Nose |
| 355 | Pressman | Bronchoscopy and Laryngeal Surgery |
| 356 | Priest | Tracheotomy |
| 369 | Singleton | Chemotherapy |
| 372 | Straith & Slaughter | Plastic Surgery of Facial Injuries |
| 378 | Van Alyea | Chronic Sinusitis |

**Key F-2 Period: 10:15 a.m. to 11:15 a.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 302 | Birsner | Surgical Anatomy of the Skull |
| 312 | Curtis | Diagnosis of Common Oral Lesions |
| 313 | Davis | Problems in Surgery of the Face |
| 315 | Dearmin | Chronic Mastoiditis |
| 323 | Gill | External Otitis |
| 333 | Holinger & Cassels | Anesthesia for Peroral Endoscopy |
| 334 | Horton | Headaches and Head Pains |
| 341 | Lindsay | Vertigo |
| 343 | Lynch & LeJeune | Suspension Laryngoscopy |
| 345 | McCaskey | Neck Surgery |
| 349 | Miller | Tracheotomy in Children |
| 354 | Perlman | Eustachian Tube Function |
| 357 | Proetz | Displacement Method |
| 362 | Robison | Maxillary Sinusitis |
| 363 | Salinger | Nasal Fractures |
| 373 | Sullivan | Surgical Repair of Facial Nerve |

**Key F-3 Period: 11:30 a.m. to 12:30 p.m.**

| Course No. | SPEAKER | SUBJECT |
|---|---|---|
| 305 | Bryant | Sphenopalatine Ganglion |
| 307 | Campbell | Differential Diagnosis of Hearing Loss |
| 314 | Day | Hydrops of the Labyrinth |
| 318 | Ellis | Congenital Anomalies |
| 322 | Galloway | Respiratory Obstruction, etc. |
| 328 | Harkness | Private Practice |
| 329 | Hart | Clinical Aspects of Bronchiectasis |
| 330 | Havens | Repair of Deformities of Face |
| 331 | Hill | Management of Antro-Oral Fistulae |
| 332 | Holinger | Photography |
| 338 | Jones | Office Procedures |
| 347 | McNally | Labyrinthine Tests |
| 364 | Schall | Cancer of Upper Respiratory Tract |
| 365 | Schenck | Tympanic Membrane Perforations |
| 366 | Sellers | Headaches of Otorhinologic Origin |
| 368 | Shea | Therapy of Headache |
| 370 | Snitman | Surgical Anatomy of the Neck |
| 379 | Waldapfel | Common Diseases of the Larynx |
| 380 | Walsh | Acute Rhinitis |
| 381 | Warren | Practical Therapeutics in ENT |
| 382 | Williams | Headache in Otolaryngology |

MEDICAL LIBRARY 51 #5 JUL 7 1947

# TRANSACTIONS

## AMERICAN ACADEMY
*of*
## OPHTHALMOLOGY
*and*
## OTOLARYNGOLOGY

MAY - JUNE
*1947*

Published bimonthly, February, April, June, August, October, and December, by the Douglas Printing Company, 109 North 18th Street, Omaha 2, Nebraska, for the American Academy of Ophthalmology and Otolaryngology. Editorial and business office, 100 First Avenue Building, Rochester, Minnesota. Subscription price $3.00 yearly to members, $6.00 yearly to non-members. Entered as Second-Class Matter November 28, 1938, at the Post Office at Omaha, Neb., under Act of March 8, 1879.

ANNUAL MEETING OCTOBER 12-17, 1947, CHICAGO

# HYPOSENSITIZATION THERAPY IN CHRONIC SINUS DISEASE

REA E. ASHLEY, M.D.
SAN FRANCISCO, CALIF.

HYPOSENSITIZATION by means of vaccines, serums, bacteriophages, and bacteriolysates in the treatment of chronic infective sinusitis has proved most disappointing in our experience. Treatment by the establishment and maintaining of proper aeration and drainage plus the administration of penicillin and the sulfa drugs has proved much more effective.

Hyposensitization by means of injections of house dust and certain molds and fungi has given universally good results in selected cases of chronic allergic sinusitis.

It is with the allergic type of sinusitis that the main body of this paper deals.

Until recently it was believed that all edematous swellings and polypi occurring in the nose and sinuses were secondary to infection, and the recognized treatment was surgical. Temporary results were frequently good following operation, but later results were often very disappointing.

This failure on the part of surgery to effect permanent cures in a considerable percentage of these patients stimulated research aimed at a better understanding of the physiology, pathology, and immunology of the nose and perinasal sinuses.

During the past two decades much investigative work has been undertaken with the result that our knowledge of these subjects has increased materially. However, the general belief still persists that failure to effect a cure in many of these patients is the result of poor or incomplete surgery. While this may be true in the treatment of pure cases of infective sinusitis, it is definitely not true in chronic sinusitis due to allergy. Adequate allergic management will often render surgery unnecessary. The all important point is to recognize the presence of allergy and treat it as such.

Foremost among these investigators has been Hansel.[4] He was first to point out the importance of allergy as an etiologic factor in chronic sinusitis. He it was who described the cytologic examination of the nasal secretion as a means of differentiating sinusitis due to allergy from sinusitis due to infection. He also was first to describe a comprehensive method of diagnosis and an effective method of treatment.

So convincing is Hansel's work that many rhinologists now believe:

1. That a great majority of all so-called chronic sinus infections are allergic manifestations.
2. That the great majority of all mucous membrane thickenings in the nose and perinasal sinuses, including polypi, are of allergic origin and not of infectious origin.
3. That a large percentage of sinus infections, if uncomplicated by allergy, will clear up spontaneously.
4. That pure infection of a sinus is a local disease and requires local treatment, and that a pure allergic sinusitis is a general disease with local nasal symptoms, and requires general immunologic treatment.
5. That combined infectious sinusitis and allergic sinusitis requires local

Presented before the Academy Section on Otolaryngology and the Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 18, 1946, Chicago, Ill.



UNIVERSITY OF MICHIGAN LIBRARIES

and general treatment with much emphasis on the general treatment and little on the local treatment. The greater the allergic element the less local treatment is indicated.

6. That many of the symptoms formerly attributed entirely to foci of infection have in fact proved to be allergic manifestations, and have responded to allergic treatment after repeated nasal operations have failed to give relief. Some of the more important of these symptoms, in which allergy should be suspected, are nasal obstruction, excessive nasal discharge, frequent acute head colds, certain types of headache, arthritis, neuritis and myositis, chronic bronchitis, vertigo, chronic sore throat, tubotympanal catarrh, mental haziness, chronic laryngitis, bronchial asthma, and many others.

Before discussing the diagnosis and treatment of chronic allergic rhinitis I should like to make a plea to rhinologists to diagnose and treat their own allergic patients. It is only by observing the results of treatment that one can become "allergy conscious," and can become proficient in the diagnosis of these conditions. Only by close observation of the changes which occur in the nasal mucosa following allergic management can the effectiveness of the treatment be determined.

The rhinologist alone is competent to make these examinations and to adequately watch and evaluate the treatment accurately.

Until recently most rhinologists have been loath to undertake the treatment of nasal allergy because it was considered most complicated, tedious and time consuming, and results following treatment were for the most part far from satisfactory; such is not now the case.

Successful management of allergy of the nose and perinasal sinuses depends upon, first an accurate diagnosis, and secondly treatment conducted along general and immunological lines.

## DIAGNOSIS

The factors necessary for an accurate diagnosis of chronic allergic sinusitis are: a careful allergic history, proper evaluation of symptoms, cytologic examinations of the nasal secretion, rhinologic examination, skin tests, and the x-ray.

*History:* A complete allergic history must include inherited predisposition; other manifestations of allergy, such as urticaria, hayfever, asthma, migraine and dermatitis; the relationship of symptoms to the ingestion of food; occupational contacts to dust and fumes; environmental contacts, and contacts with animals and birds. All of this information can quickly be obtained from the history.

*Symptoms:* The cardinal symptoms of chronic allergic sinusitis are: nasal obstruction, excessive nasal discharge, and sneezing. In complicated cases all of the cardinal symptoms may be missing. These are the patients in which allergy is usually missed.

The obstruction is usually bilateral, but may affect one side only. The amount of obstruction varies greatly and may change rapidly with sudden changes of temperature and humidity. The obstruction may be transitory or continuously present for years.

Increased nasal secretion varies greatly in amount and consistency. It may be thin, watery and excessive in amount, or thick and glary and very small in amount.

Other important symptoms which are frequently associated with nasal manifestations of allergy, and which may give a lead to diagnosis are: lacrimation, itching of the skin, excoriations about the nares, dry and nonproductive cough,

hoarseness, recurrent head colds, frequent sore throat, mental haziness, and croup in children.

*Cytology:* Cytologic examinations should be made routinely when allergy is even suspected and allergy should always be suspected in all patients of all ages complaining of nasal obstruction or excessive nasal discharge until it is definitely ruled out as an etiologic factor.

Secretions are obtained from the nose by requesting the patient to blow his nose on a piece of glazed paper, or from the pharynx by means of a swab or forceps. The secretion is transferred to a slide and stained for eosinophils by using Hansel's quick stain — time required is about two minutes. The slides are examined under low power.

The secretion from a normal nose shows epithelial cells and debris with a few neutrophils and an occasional eosinophil. The secretion from a nose harboring infection shows a few epithelial cells, many polymorphonuclear leucocytes and an occasional eosinophil.

The secretion from a nose in which combined infection and allergy exist shows many neutrophils and an increased number of eosinophils.

Hansel believes that the presence of large numbers of eosinophils, or eosinophils occurring in clumps, is pathognomonic of allergy.

It is interesting to note that when allergic patients develop nasal infections, the eosinophils decrease in number and the polymorphonuclear leucocytes appear in great numbers. When the infection subsides, however, the polymorphonuclear leucocytes decrease and the eosinophils again appear. Frequent cytologic examination is often necessary to establish a positive diagnosis in complicated cases of suspected allergy.

*Rhinologic Examination:* In the past a pale, boggy-appearing nasal mucosa was thought to be indicative of allergy, while a red and engorged membrane indicated an infectious process. These ideas must now be re-evaluated since it has been abundantly demonstrated by cytologic studies that in the early stages of allergy, and in the presence of combined allergy and infection the nasal mucosa is quite frequently red and inflamed, and that the characteristic pale, boggy appearance of the allergic membrane develops only with chronicity.

*Skin Tests:* Skin tests in nasal allergy are not accurate, but when positive may be of value in outlining specific treatment. Some of our best results in hyposensitization to house dust have been obtained in patients whose skin tests with high concentrations of this allergen have been negative.

*X-ray:* X-ray findings can be most misleading. The presence of thickened membrane in all of the sinuses does not necessarily mean sinus infection. On the other hand, allergic thickening may be demonstrated in only one sinus, and marked thickening may disappear under proper allergic management. Radiograms should never be made during an acute exacerbation, because it has been frequently noted that thickenings shown under these circumstances may disappear within a very short period of time.

The important points in making a diagnosis of allergy of the nose and perinasal sinuses in the order of their importance then are:

1. A comprehensive history.
2. Evaluation of complaints and symptoms.
3. Positive cytologic evidence.
4. Appearance of the nasal mucosa.
5. Skin tests.
6. X-ray findings.

If this plan of procedure is followed, not only can a definite diagnosis of allergy be made, but a lead to specific treatment will frequently be indicated.

TREATMENT

While hyposensitization is the main subject of this paper several other elements must be briefly mentioned since they all play a part in obtaining satisfactory results.

Although the treatment of each patient must be on an individual basis, we have tried to develop a routine which begins with the simplest method of treatment which gives promise of relief and resorts to more complicated procedures only after simpler methods have failed. These procedures may be summarized as follows:

1. Elimination of the offending allergen.
2. Use of certain drugs.
3. Conservative surgery in those patients who continue to have symptoms either because of permanent hypertrophic changes in nasal and sinus mucosa, or in whom infection remains after control of the allergy has been accomplished.
4. Hyposensitization.

*Elimination of Offending Allergen:* The elimination of the offending allergen is usually not possible, but prophylactic measures can be of great aid in treatment. These should include:

1. Stop all nose drops, jellies, nasal irrigations, and nasal sprays.
2. Dust-proof covers for pillows and mattresses.
3. Elimination of all animal and bird contacts.
4. Dust-free sleeping quarters, i.e., removal of all wool rugs and ozite floor coverings; discontinue use of insect repellents and sprays in sleeping quarters; frequent thorough cleaning of bedroom woodwork and floors with an oiled cloth.
5. Elimination diet when indicated.

*Drugs:* Ephedrine compounds are of inestimable value in controlling symptoms in acute allergic exacerbations, and in chronic manifestations before hyposensitization has become effective. A valuable combination is:

Ephedrine .....gr. 3/8 to 1/4
Phenobarbital .....gr. 1/4 to 3/8
Atropine .....gr. 1/400
Thiamine chloride ..Units 1000

The advantage of this prescription over some of the proprietary preparations is that the ephedrine and phenobarbital ratio can be adjusted to suit the nervous as well as the phlegmatic patient (Arylcaps, Tedral and Novaline have also been of value in controlling some of the allergic symptoms). Our experience with Benadryl and Pyrobenzamine (two promising drugs) has as yet been too limited to draw any definite conclusions as to their value in chronic allergic sinusitis.

*Surgery:* Operative procedures should be palliative before the allergy has been brought under control. Experience has shown that much less radical surgery is required, and much better results are obtained if corrective surgery is postponed until hyposensitization has been established.

*Hyposensitization:* The great majority of nasal allergies are caused by inhalents, and if those specific allergens which cause hayfever are excluded, the most common inhalent causing nasal symptoms is house dust. House dust contains many known allergens to which the patient may be sensitive, and some specific allergen, the nature of which is not know. Unless the patient can be definitely proved to be allergic to a specific allergen, house dust is by all odds the allergen of choice in the treatment of chronic allergic sinusitis. According to Davidson,[3] the chief constituents of house dust are: cotton, flax, jute, wool, silk, animal hair, kapok, orris root, Pyrethrum and tobacco. Cooke,[2] Cohen[1] and his co-workers all presented

evidence that house dust contains a specific antigen.

In 1938 Hansel[5,6,7] made two very important observations which revolutionized the treatment of nasal allergy. His first observation was that hyposensitization is accomplished in a high percentage of allergic patients by the injection of very dilute solutions of antigen.

The doses recommended by Hansel were so fantastically small as to tax one's credulity. Clinical evidence over a period of years however, has substantiated this observation. Hansel's second observation was that much better results are obtained in treating allergic patients if the dose of allergen is held at the effective optimum dose; i.e., the smallest dose which will relieve symptoms and not produce reactions, rather than if the dose is increased to tolerance, i.e., to the maximum dose.

Following these observations and guided by Hansel's[8] plan of treatment a routine method of hyposensitization was adopted, which has given most gratifying results in the management of chronic allergic sinusitis. The following is a brief outline of the method.

Establishing the size of the proper dose is the all important factor in obtaining satisfactory results.

The patients are treated according to symptoms and not according to skin tests, i.e., the more severe the symptoms the smaller the dose of antigen.

The antigen used is house dust to which Wittich[9,10] antigen (consisting mainly of molds, fungi and smuts) has been added in the proportions of one part of Wittich to nine parts of dust.

Dilutions of 1:100,000,000 and 1:10,000,000, 1:1,000,000,000 and 1:10,000,000,000 are prepared.

The starting dose is decided according to the severity of the symptoms. I repeat, the more severe the symptoms, the smaller the dose. The starting dose in severe cases is usually 0.1 cc. of 1:100,000,000 dilution (1:1,000,000,000 in very severe cases and those with asthmatic history) in young children and, in mild cases, 0.1 cc. of 1:10,000,000 dilution.

Injections are given subcutaneously twice weekly, and the dose is increased 0.1 cc. each visit until definite improvement in the symptoms is obtained, or until 0.5 cc. is reached.

If no improvement is manifest at 0.5 cc. of 1:100,000,000, then 0.1 cc. of the next lower dilution, 1:10,000,000, is given, and the same routine carried out. Trial injections of stronger or lower dilutions may be necessary before the effective optimum dose can be established. Once improvement is noted the dose is held at *that* dilution and size, and the time between injections is increased; first to one injection per week, then one injection every two weeks, and finally to one injection every three weeks. When the patient is symptom free for three twenty-one day periods, he is instructed to return for further treatment only if symptoms return. Two or three injections at that time usually render the patient symptom free for another varying period of time.

Occasionally a dilution of 1:1,000,000,000 has been found to improve symptoms when lower dilutions have failed to give results. On the other hand, a dose which has given improvement in symptoms but which does not carry the patient symptom free through to the time of the next injection, is too small and should be increased by 0.1 of 1 cc. of the dilution then being given.

My experience in treating chronic allergic sinusitis has been at variance with that of some other rhinologists doing this same type of work in that I have rarely found it necessary to increase the dose above 0.5 cc. of 1:10,000,000 dilution in order to render the patient symptom free. In fact, my best results

have been with the 1:100,000,000 dilutions.

## RESULTS

1. A high percentage of allergic patients treated according to this routine have become symptom free for varying lengths of time (months to years). If symptoms return two or three injections are usually all that are required to again relieve symptoms.

2. The severe and dangerous local and constitutional symptoms which were occasionally seen when much larger doses of antigen were injected do not occur with the low dosage treatment.

3. The only untoward symptoms which have been observed since this method of treatment was adopted were in patients with an asthmatic background. On two or three occasions dilutions of 1:100,000,000 have thrown the patient into an acute attack of asthma. All were able to tolerate dilutions of 1:1,000,000,000 however, and their general nasal allergic symptoms were subsequently brought under control by means of the low dosage plan of treatment.

4. Subjective improvement of symptoms is frequently manifest after a few injections—often after the first injection.

5. Objective improvement in the nasal mucosa is frequently observed after four or five injections.

I am quite cognizant of the fact that there are many statements in this paper, and many points in this method of procedure which have only clinical experience as their basis. I take the position however, that patients suffering from chronic allergic sinusitis should not be denied the relief which follows hyposensitization by this method because experimental justification for such treatment is as yet lacking.

A few of these unknown factors which need clarification are here listed in the hope that some one may have the answers, or that he may be stimulated to undertake some investigative work on them.

1. More accurate methods of diagnosis are needed.
2. To date, no accurate method of assaying the potency of house dust has been described.
3. The exact character of the specific allergen found in house dust has never been established.
4. A better explanation as to why hyposensitization gives relief is needed. Is the reaction similar to the formation of antibodies produced by bacterial vaccines? Or, may it not be some entirely different reaction, not related in any way to the formation of immune bodies?
5. No method of testing the antibody titer following hyposensitization to house dust has yet been described.
6. What is the part played by the eosinophils?
7. In what way does histamine tie into the picture?

These are only a few of the many questions which need answering, but when they are answered we may be better able to explain why the clinical results following low dosage therapy in chronic allergic sinusitis are so satisfactory.

## SUMMARY

1. Hyposensitization by means of bacterial antigens has failed to produce results in chronic sinusitis due to infection.
2. Hyposensitization by means of house dust has given good results in selected cases of chronic sinusitis due to allergy.
3. Failure to recognize allergy as primary causative factor in chronic sinusitis has led to frequent failures following nasal surgery.
4. It is still the common belief that many of these operative failures have been due to poor or incomplete

surgery—in reality many of them are due to failure to recognize allergy and treat it as such.

5. It is important for rhinologists to become "allergy conscious" by treating their own allergic patients. It is the only way to become proficient in diagnosis as well as treatment.
6. Diagnosis of nasal allergy in the order of their importance depends on complete allergic history, evaluation of symptoms, cytologic examinations of nasal secretion, rhinologic examination, skin tests, and x-ray.
7. Satisfactory treatment depends on elimination of causative allergen, use of ephedrine and similarly acting drugs, surgery only after hyposensitization has been obtained—finally hyposensitization.
8. Most chronic nasal allergy is caused by inhalents.
9. If those specific allergens causing hayfever are eliminated the most common allergen causing nasal symptoms is house dust.
10. In 1938, Hansel made two suggestion which revolutionized the treatment of allergy of the nose and perinasal sinuses. These were fantastically small doses of allergen and the buildup of dosage only to optimum effective dose instead of to the maximum dose.
11. Important points in hyposensitization treatment of chronic allergic sinusitis are:
    (a) Small doses of very dilute antigen.
    (b) Build up dose only to point at which relief of symptoms is obtained then lengthen time between injections—if symptoms are aggravated drop the dose.
    (c) When patient is symptom free stop treatment.
12. A very high percentage of patients treated have become symptom free for varying lengths of time—months to years.
13. No severe local or constitutional symptoms have been observed except in a few patients with an asthmatic background, and these were subsequently successfully treated by lowering the initial dose of antigen.
14. Improvement of symptoms is often noted after the first injection, and improvement in the appearance of nasal mucosa is frequently observed after the fourth or fifth injection.
15. Many points in method of procedure have abundant clinical basis, but no scientific basis.
16. The position is taken that patients should not be denied relief of symptoms by this treatment because of lack of experimental justification for the treatment.
17. Many unknown factors need clarification.

## REFERENCES

1. Cohen, M. B., Nelson T. and Reinarz, B. H.: Observations on the nature of the house dust allergen, J. Allergy, 6:517-524 (Sept.) 1935.
2. Cooke, R. A.: Studies in specific hypersensitiveness. New etiologic factors in bronchial asthma, J. Immunol., 7:147-162 (March) 1922.
3. Davidson, M. T.: The source of the allergic activity of house dust, J. Allergy, 14:244-255 (March) 1934.
4. Hansel, F. K.: Allergy of the nose and paranasal sinuses, St. Louis, The C. V. Mosbey Company, 1936.
5. Hansel, F. K.: Personal communication, 1938.
6. Hansel, F. K.: The treatment of allergy of the nose and paranasal sinuses by hyposensitization with dust extracts, Tr. Am. Laryng., Rhin & Otol Soc., 1940, 156-179.
7. Hansel, F. K.: Principles of diagnosis and treatment of allergy as related to otolaryngology, Laryngoscope, 53:260-275 (April) 1943.

8. Hansel, F. K.: Some experience with small dosage dust and pollen therapy, South. M. J., 38:608-613 (Sept.) 1945.
9. Wittich, F. W.: Further observations on allergy to smuts, Lancet, 59:382-388 (Sept.) 1939.
10. Wittich, F. W.: The nature of various mill dust allergens, Lancet, 60:418-421 (Sept.) 1940.

## DISCUSSION

HENRY L. WILLIAMS, M.D., Rochester, Minn.: In a discussion such as this, in which terminology is so important, it is essential to give a clear definition of the subject and the terminology used.

Von Pirquet's definition of allergy as including all alterations in the state of reactivity of an organism due to contact with any organic living or nonviable substance is now too broad to be of the greatest value.

Urbach suggested the term "pathergy" as the comprehensive term for all allergic and nonallergic processes of hypersensitiveness and hyposensitiveness. He subdivided pathergy into allergic, hetero-allergic and nonallergic pathergies.

Allergy is defined as embracing alterations in the reactivity of an organism as the result of the result of the production of specific antibodies usually occurring after exposure to a substance, the antigen. Nonallergic hypersensitiveness embraces those cases of altered reactivity of the organism to actinic, thermal, mechanical, chemical, or emotional stimuli acting directly or intermediately through the autonomic nervous system in which the mechanism is not that of the antigen-antibody reaction.

The hetero-allergies are subdivided into paraallergy and metallergy. Parallergy is defined as the condition in which during the development of an allergy or during states of considerable allergic fluctuation, a specifically sensitized organism will react more easily and more rapidly to a given living or nonviable excitant than will a nonallergic organism. Metallergy denotes the fact that a specifically sensitized organism will respond to a subsequent exposure of a different nature with specific allergic reactions presenting the same clinical picture as that elicited by the first allergen.

I believe this concept of pathergy with its subdivisions makes the classification and understanding of the various phenomena with which we are confronted much simpler.

I feel that Dr. Ashley is a little overenthusiastic in his embracement of the concept of allergy when he implies that an allergic background is present in most if not all cases of chronic suppurative sinusitis. I believe that, while the rhinologist should be allergy-conscious, he need not be inhibited from doing obviously necessary surgical work by a fear that the whole condition is allergic after all. In most cases in which allergy is a factor, its diagnosis is rendered fairly obvious by a properly taken history and a properly done examination. I believe that Ashley has also overemphasized the value of cytologic examination of the nasal secretion. Since repeated cytologic examinations are often necessary before a definite diagnosis can be established by this means, time can often be saved, when history and examination indicate a pathergic causation, by a clinical trial of antipathergic methods, particularly with the advent of nonspecific antipathergic methods of therapy which offer such great promise of further development.

It is of great satisfaction to me as a rhinologist in reading Ashley's paper to realize that many of the advances in the therapy of rhinopathy, especially the contributions of Hansel, have been the result of studies by men in my own specialty who have taken special interest in the allergies as they influence nasal and sinal pathologic conditions. This gives point to Ashley's plea that rhinologists carry out or at least supervise their own "allergic" management of cases in which allergic or at least pathergic rhinopathy is suspected. Rhinology is one of the few well-rounded specialties in medicine in that the same individual is responsible for both the medical and surgical management of a given case and is in a position both by training and by experience to decide when surgical intervention is to be instituted and when it is to be avoided. Best of all, the rhinologist is able to effect a judicious combination of medical and surgical management when such is indicated.

As far as the rhinologist is concerned allergy is the most important branch of medicine. It is imperative therefore, as Ashley states, for all rhinologists to familiarize themselves thoroughly with allergic procedures. Ashley has pointed out that with the exception of pollinosis, routine skin testing is of little if any value in regard to the diagnosis in the rhinopathies and elaborate diagnostic equipment is unnecessary.

Because my experience in rhinology covers only the past twenty-five years, I am not familiar with the era of destructive surgical treatment for rhinopathy which Ashley describes, but I am in full accord with his plea that, when infection occurs secondarily to, or in conjunction with, allergic or pathergic rhinopathy, only such surgical treatment be done as would be indicated

if the altered reactivity were not present. As he points out, "allergic" management will often so reduce the congestion present that in a patient in whom surgical drainage seemed required before such management was instituted it will be obvious afterward that surgical intervention is unnecessary. It must not be forgotten, however, that parallergy exists, so that a secondary sensitization to bacterial products will at times interfere with and vitiate attempts at hyposensitization. Surgical management and "allergic" management must be joined to secure success in certain cases.

The value of the low dosage schedules which Ashley, following Hansel, recommends is unquestioned. It is our experience at the Mayo Clinic that dosages carried up to the point of reaction are contraindicated although we have not tried hyposensitization in unquestioned allergic conditions such as pollinosis with such dilute solutions as Hansel recommends. In therapy with histamine, following Hansel's dosage schedule, we have secured good results using solutions of the same order of magnitude. These results with histamine have been confirmed by Farmer and by Urbach. The fact that the use of the same dosage schedule and dilutions of histamine that Hansel used for house dust produce similar results led me to suspect that house dust might contain histamine. I secured a sample of house dust from Hansel and submitted it to Code for testing. He was unable to demonstrate any histamine content in the sample of house dust submitted. In spite of this it is difficult to believe that using dilutions such as this we are producing hyposensitization by influencing an allergic condition. In skin testing with house dust it is my experience that well over 50 per cent of patients will give positive reactions even though from grounds of clinical history and examination there is no reason to suspect allergy to exist in them. Likewise, I have found patients who gave negative reactions to house dust who from history and clinical examination were pathergic and could be relieved by nonspecific antipathergic methods. Therefore, I am inclined to class treatment with house dust among the nonspecific antipathergic methods of hyposensitization.

I believe the correctness of the hypothesis receives substantiation from the successful clinical results which Ashley, Hansel, Craddock and others report they have secured with house dust in patients giving negative skin reactions to this substance. These allergists have been unable to free themselves from concentration on the antigen-antibody concept of resistance when securing results which apparently do not depend on an antigen-antibody type of reaction.

The specialty of allergy has suffered from the neglect of the tremendous mass of evidence which Selye and his co-workers have piled up indicating that there is another more primitive and therefore more fundamental mechanism by which the organism can protect itself from noxious stimuli—the general adaptation syndrome. This mechanism does not depend on an antigen-antibody reaction for its protective effects and yet disorders of the "alarm reaction" are associated with the release of histamine and produce a picture clinically indistinguishable from that produced by an allergic reaction. In my experience not more than 10 per cent of cases of vasomotor rhinitis (pathergic rhinitis) can be classified by the clinical means available as allergic. In my opinion, excluding pollinosis, the majority of cases of rhinopathy should be classified as pathergies. I believe that the generally disappointing results obtained in the treatment of the rhinopathies by routine allergic methods depend on the failure to realize that an antigen-antibody type of mechanism is not a factor in producing the presenting symptoms in many instances. Because alteration of the general adaptation mechanism depends on disorders of endocrine and autonomic balance; progress in relieving many of the so-called allergic patients will be achieved only when attention is focused on the contributory factors of endocrine imbalance and emotional maladjustment.

It is for this reason that the taking of an adequate history is of such great importance. To be adequate, however, it must cover a much wider field of inquiry than the specimen history presented by Ashley. In addition to inquiries in regard to contacts, foods and the like, an adequate history should include an inquiry into social and family adjustments, reaction to atmospheric changes, and physiologic functions related to the endocrine system.

ABELARDO IRIGOYEN FREYRE, M.D., Santa Fe, Argentina: I regret that I was unable to obtain a copy of Dr. Ashley's paper, so that I might have made a careful analysis of it.

However, may I congratulate Dr. Ashley on his interesting work, which constitutes a valuable contribution to the treatment of chronic allergic sinusitis, and I would only like to remind you that, actually, among the causes capable of producing an alteration of the mucosa of the sinuses, as a consequence of parasympathetic stimulation, are those which produce edema and an overlarge amount of secretion, such as occurs, for example, in the edema of the bronchial mucosa in the "status asthmaticus"; also in edema due to urticaria, at least in its manifestations in the sinuses. For this reason, we believe that a large

percentage of sufferers from chronic sinusitis of the allergic type can be benefited by desensitization, which results in a diminution of the edema of the mucosa, and consequently, improves aeration of the paranasal sinuses.

I would a'so like to say that one cannot speak of definite cures, where allergy is concerned, since we believe that an individual is born allergic and dies allergic. We always state that allergic sinusitis is simply an allergic reaction which happens to occur in the sinuses, but which is not an isolated sinusitis: rather, it is a local manifestation of a generalized sensitivity.

I want to emphasize the necessity of careful x-ray studies, in accordance with the criteria estab!ished by MacMillan, of Boston. In these x-rays we can see, in the case of the frontal sinuses, a general thinning of the walls of the sinuses, particularly of the intersinus septum, as well as in the olfactory fissure, which does not disappear in those cases of true allergic sinusitis.

This data concerning allergic sinusitis, which is furnished by the x-rays, serves as a guide (in addition to the information found in the clinical history, the symptoms, etc.) to enable us to start, with some possibility of success, the treatment laid out by Dr. Ashley, or to consider surgical treatment.

Treatment: As far as the use of drugs is concerned, we wou!d like to say that we prefer not to use any local medication of the ephedrine or similar type, because of the inconstancy of the results obtained, without first making a complete allergic study, and in some cases, as, for instance, those in which one is dealing with a sensitivity to a large number of allergens, we use large doses of vitamin $B_1$, a method of treatment which will be the subject of a work to be presented tomorrow. As Hansel has already stated, we also use the method of desensitization by small doses of powder.

As far as surgery is concerned, we have adopted a conservative stand, and we operate in cases where it is necessary to aerate the sinuses because of existing degeneration and exaggerated hyperplasia.

Fina!ly, we wish to emphasize the necessity of incorporating, in our service of otorhinolaryngology and broncho-esophagology, a division of allergy, which, along the lines advocated by Dr. Ashley, will be of inestimable value in the study and treatment of these disorders.

FRENCH K. HANSEL, M.D., St. Louis, Mo.: (Discussion not available.)

# CHANGES IN THE NASAL FUNCTION ASSOCIATED WITH VARIATIONS IN EMOTIONAL STATE AND LIFE SITUATION

THOMAS H. HOLMES, M.D., HELEN GOODELL, STEWART WOLF, M.D., *and* HAROLD G. WOLFF, M.D.
BY INVITATION
NEW YORK, N. Y.

IT is the purpose of this presentation to offer, for your consideration, data from recent experiments dealing with disturbances in nasal function productive of troublesome symptoms and even possibly serious disease.

Nasal disturbances are among the commonest of man's complaints and yet comparatively little is known of their pathogenesis. In these experiments we have been able to demonstrate that much nasal difficulty is related to stressful life situations.

Our study involved the direct examination of the noses of a large number of people under a wide variety of circumstances. Examinations were carried out under standard conditions of lighting. A warmed nasal speculum was used for inspection of the structures but no further instrumentation. The range of nasal functions, including degree of redness of the membranes, swelling, secretion, and obstruction, were roughly quantitated in terms of 0 to 4+.

It was found that individuals vary greatly in their nasal reactivity. Some displayed wide variations from time to time in color, swelling, and secretion, while others varied but little.

Figures 1 and 2 compare the nasal changes in the subjects studied. Daily observations are plotted on the abscissa, and on the ordinate is represented the degree of hyperemia, swelling, secretion, obstruction and color. Here you see the wide variations in some individuals contrasted with the comparatively narrow ones in other individuals.

Having established that important changes in nasal function occur from time to time and that these changes can be satisfactorily observed, our experimental subjects were exposed to a series of environmental threats. The first of these consisted of noxious stimulation of the nose with fumes of ammonium carbonate. We observed the reaction which you might expect; namely, after inhalation a sudden hyperemia, swelling of the nasal structures with hypersecretion, and obstruction. Associated with these nasal changes there occurred weeping, tearing, and spasm of the eyelids as well as strenuous coughing.

In figure 3 is observed the reaction time in minutes on the abscissa and quantity of secretion, color, swelling, and obstruction on the ordinate. Here, after inhalation, there occurred a sudden acceleration of all functions. This was obviously a reaction of defense on the part of the organism, an effort at shutting-out and washing-away and ejection of the offending substance.

Another type of assault against the person comes from the inhalation of pollens to which he may be sensitive. Accordingly, we studied a subject in an attack of hay fever from its very inception. Prior to the attack the septum and turbinates were of normal appearance.

From the New York Hospital and the Departments of Medicine and Psychiatry, Cornell University Medical College, New York, N. Y.

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

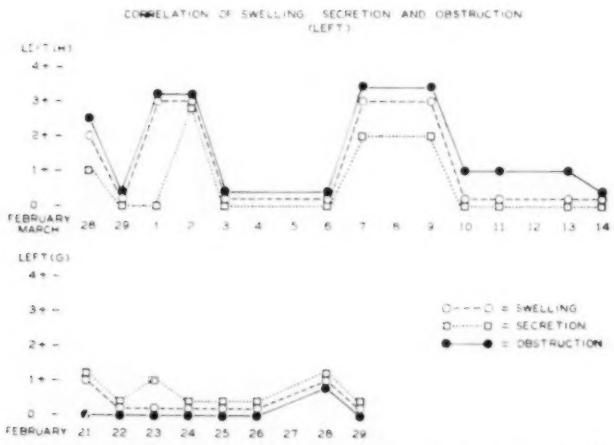



FIG. 1. — Correlation of swelling, secretion and obstruction in the left nostril of two individuals showing varying degrees of nasal reactivity.

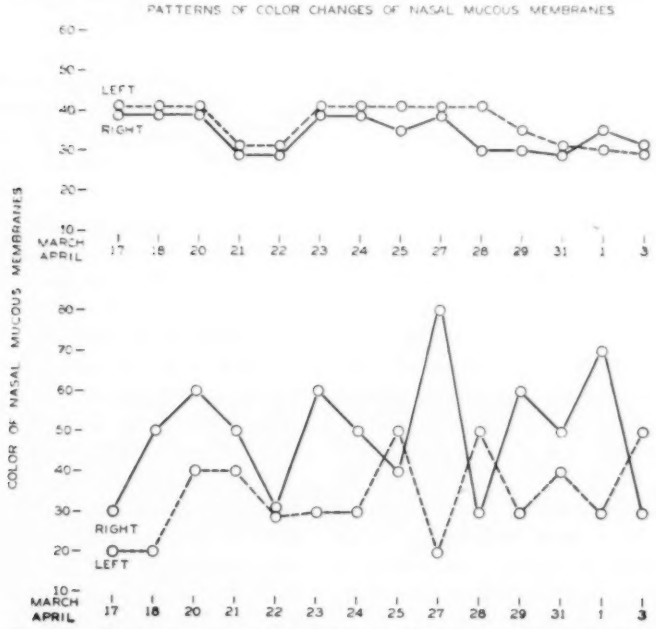



FIG. 2. — Relatively minor color changes of the nasal mucous membranes observed during 2½ weeks in one individual contrasted with marked variations in color during the same period in another. In the latter the right side of the nose was usually redder than the left.

Immediately upon beginning to cut flowers in his garden, however, the subject began to cry and sneeze. His membranes were promptly examined and the results are recorded in figure 4.

It is clear that immediately upon inhaling the offending pollens there occurred an extreme hyperemia with hypersecretion, swelling, and obstruction in the nose. This particular attack was an abortive one but frequently, when the swelling of the membranes is sustained, the hyperemia subsides leaving the membranes pale but still swollen, wet,

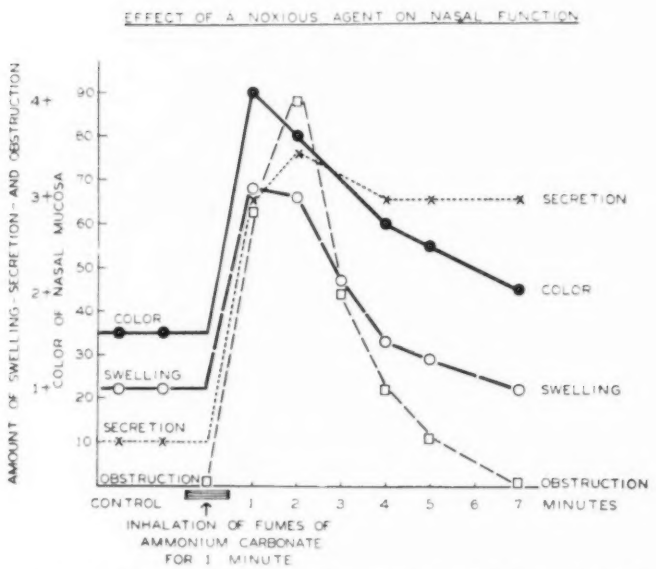



FIG. 3. — Hyperemia with swelling, hypersecretion and obstruction of the nose following inhalation of noxious chemical agent — ammonium carbonate.

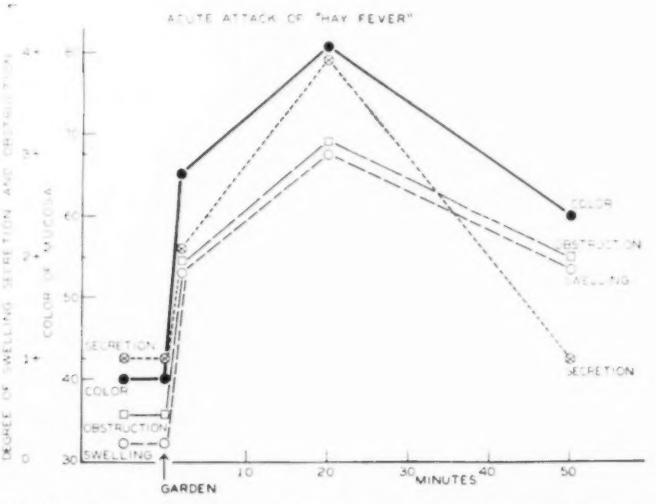



FIG. 4. — Hyperemia, swelling, hypersecretion and obstruction in the nose during an abortive acute attack of hay fever.

nd edematous-looking. This pale, swol-
en state is the usual appearance of the
ose of the hay fever sufferer when he
omes to the doctor's office for treat-
ent. It is clear, however, that, as in
he case of inhalation of irritating
umes, the hyperemia comes first. This is another instance of the bodily reaction of shutting-out and washing-away.

Our next step was to inflict upon the experimental subject a nonspecific threat, not directed at his respiratory passages. Accordingly, an intense headache was induced by constricting the

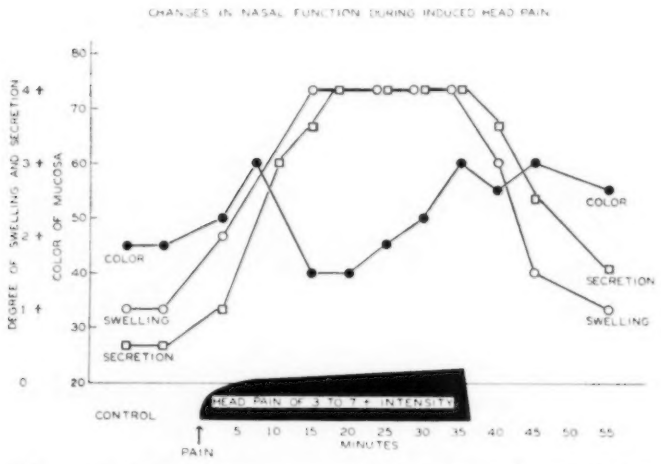



FIG. 5. — Hyperemia, hypersecretion, and obstruction in the nose during induced headache—nasal reaction to pain.

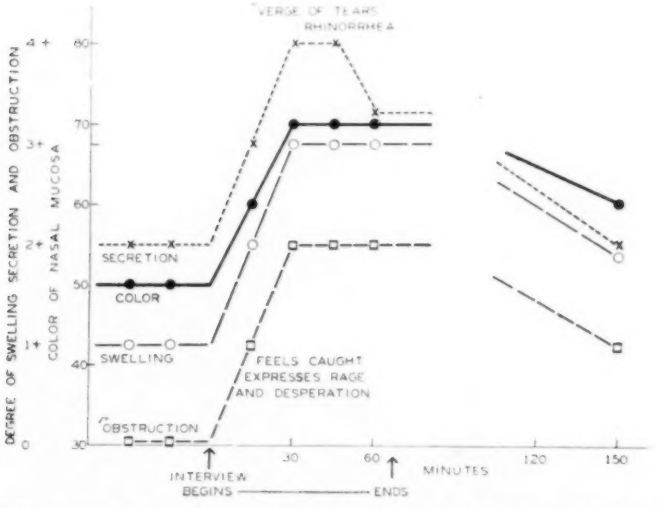



FIG. 6. — Experimentally induced hyperemia, swelling, hypersecretion, and obstruction in the nose during an interview in which the subject experienced anger and resentment.

head in a tight-fitting steel crown. This was a highly unpleasant experience associated with feelings of apprehension on the part of the victim and it provoked the same reaction of defense as that described already for more specific threats to the nose. (Fig. 5.)

The next step was to learn whether or not situational threats which did not involve the application of physical trauma would induce such a pattern of defense with nasal changes. (Fig. 6.)

A subject whose nasal structures at the time of observation were normal was forcibly reminded that he was caught in the toils of an unfavorable marriage, that his wife was using him for a meal ticket and giving him nothing in return. After the discussion was ended the subject was reassured

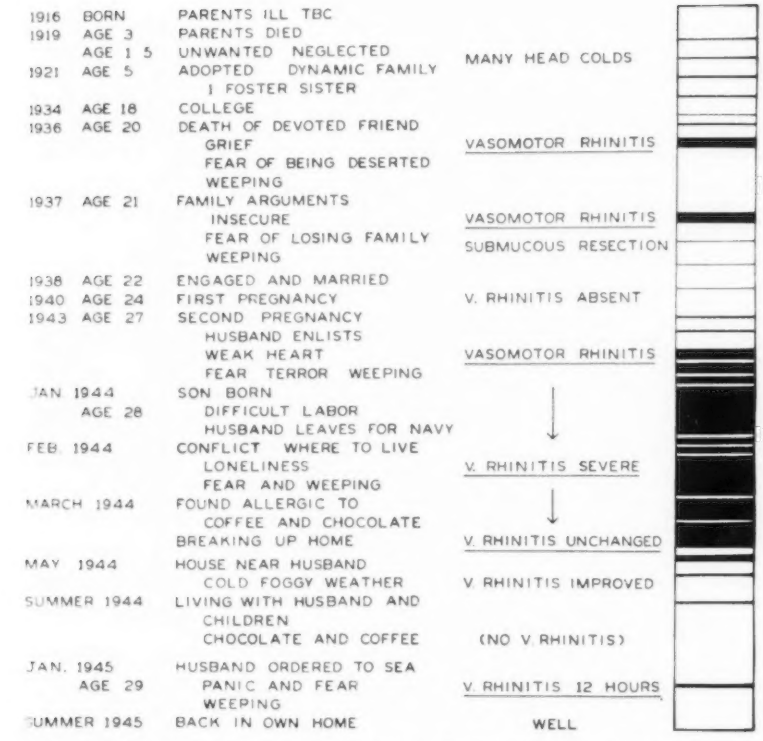



FIG. 7. — Life chart illustrating coincidence of situational threats and nasal disturbances in an insecure, anxious woman.

and diverted and after another hour the nasal functions had returned toward normal.

The changes in nasal function which occur under stress and which were observed in this patient after initiation of the unsympathetic interview are in themselves often sufficient to produce both obstruction and pain.[2] Added to these, however, may be the complicating factor of infection from the ever-present bacterial and virus organisms on the mucous membrane. Given an edematous mucosa with improper drainage, a pyogenic reaction may easily be set up and, once established, the infective process might perpetuate itself.

We then examined and inquired into the life stories of several patients who came to our hospital with nasal complaints including frequent colds and sinusitis.

In a high percentage of these individuals, as reported elsewhere,[1] it was possible to correlate the onset and recurrence of nasal troubles with episodes of situational stress and conflict; furthermore, a fairly clear-cut pattern of attitude and behavior was recognized among these subjects. Their actions in meeting threats were predominantly defensive. The shutting-out pattern in their noses reflected a general reaction of non-participation, rather than aggression in dealing with problems. In figures 7, 8 and 9 are illustrated the correlation of life situations, reactions and symptoms in three of these patients.

While our temporal correlations of nasal disease with adverse life situations

FIG. 8. — Life chart illustrating coincidence of situational threats and nasal disturbances in an insecure, dissatisfied, resentful woman.

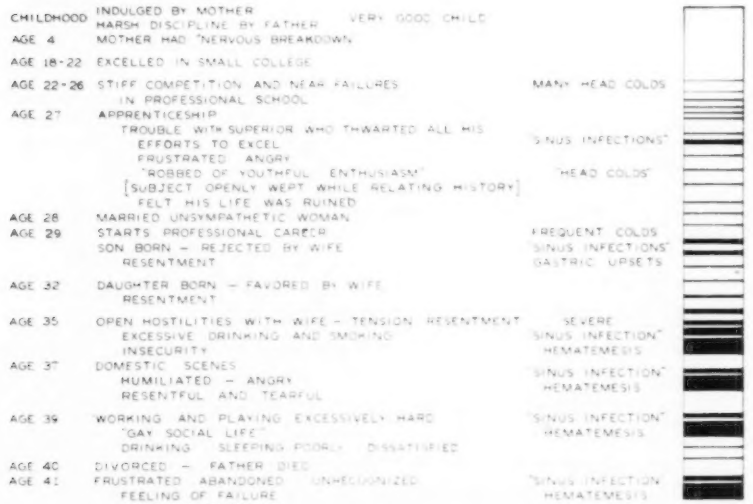

CHILDHOOD INDULGED BY MOTHER
HARSH DISCIPLINE BY FATHER VERY GOOD CHILD
AGE 4 MOTHER HAD "NERVOUS BREAKDOWN"
AGE 18-22 EXCELLED IN SMALL COLLEGE
AGE 22-26 STIFF COMPETITION AND NEAR FAILURES MANY HEAD COLDS
IN PROFESSIONAL SCHOOL
AGE 27 APPRENTICESHIP
TROUBLE WITH SUPERIOR WHO THWARTED ALL HIS
EFFORTS TO EXCEL "SINUS INFECTIONS"
FRUSTRATED ANGRY
"ROBBED OF YOUTHFUL ENTHUSIASM" "HEAD COLDS"
[SUBJECT OPENLY WEPT WHILE RELATING HISTORY]
FELT HIS LIFE WAS RUINED
AGE 28 MARRIED UNSYMPATHETIC WOMAN
AGE 29 STARTS PROFESSIONAL CAREER FREQUENT COLDS
SON BORN — REJECTED BY WIFE "SINUS INFECTIONS"
RESENTMENT GASTRIC UPSETS
AGE 32 DAUGHTER BORN — FAVORED BY WIFE
RESENTMENT
AGE 35 OPEN HOSTILITIES WITH WIFE — TENSION RESENTMENT SEVERE
EXCESSIVE DRINKING AND SMOKING "SINUS INFECTION"
INSECURITY HEMATEMESIS
AGE 37 DOMESTIC SCENES "SINUS INFECTION"
HUMILIATED — ANGRY HEMATEMESIS
RESENTFUL AND TEARFUL
AGE 39 WORKING AND PLAYING EXCESSIVELY HARD "SINUS INFECTION"
"GAY SOCIAL LIFE" HEMATEMESIS
DRINKING SLEEPING POORLY DISSATISFIED
AGE 40 DIVORCED — FATHER DIED
AGE 41 FRUSTRATED ABANDONED UNRECOGNIZED "SINUS INFECTION"
FEELING OF FAILURE HEMATEMESIS
BITTER RESENTMENT

FIG. 9. — Life chart correlating nasal and gastric disturbances with life situations.

are interesting, the most convincing evidence of the relation of life situation to nasal disorders lies in the fact that it was possible with certain subjects to manipulate the situation during an interview under experimental conditions with the nasal structures in view.

Such a case (Fig. 10) was that of a 36 year old orthodox Jewess, whose nasal obstruction and discharge had begun four years before when she found herself caught in a serious dilemma having to do with her failure to have vindicated her position when she broke away from her family tradition to marry a Roman Catholic. She had given up hope of her husband measuring up and had placed all her hopes and ambitions on her children. Her nasal symptoms began when her older child was discovered to have

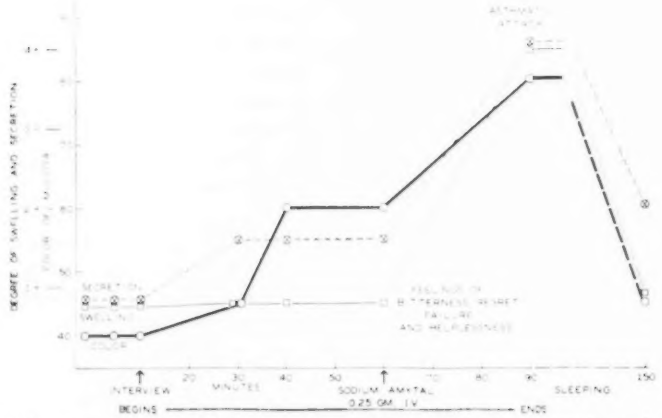



FIG. 10. — Experimentally induced hyperemia, swelling, hypersecretion, and obstruction in the nose associated with narrowing of the bronchial tubes during a discussion of topics provocative of feelings of remorse, guilt, and resentment.

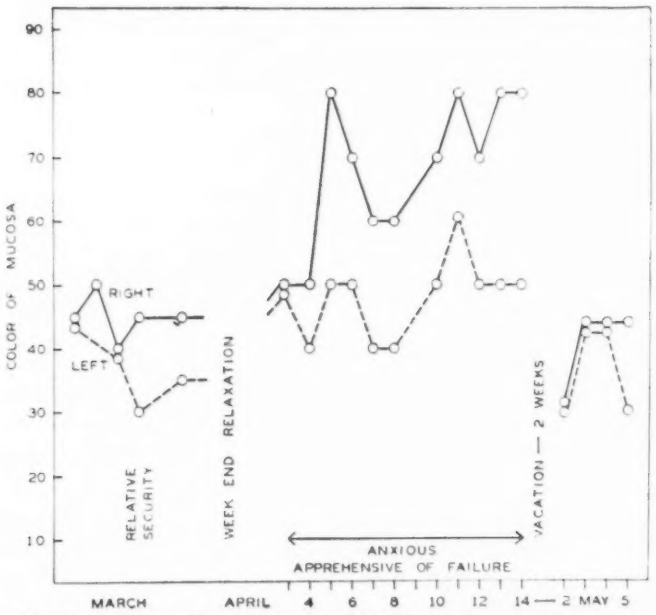



FIG. 11. — Hyperemia more on the right side of the nose than on the left during a period of conflict in which the subject's "independence", "career" and "approval from his senior colleague" were threatened.

diabetes and two years later, when her younger child became ill, she developed asthma.

In this woman, in three interviews under sodium amytal, it was possible to turn on and off at will her nasal symptoms and asthma. During relaxation and diversion she was free of symptoms but when sensitive topics were discussed unsympathetically her nasal membranes began to swell and secrete, creating partial obstruction of the nose. Even her deeper respiratory structures constricted, resulting in a typical attack of

asthma. It is clear that in response to situational threats the same changes may occur in nasal function as in response to locally applied noxious stimuli.

In 2 other subjects similar changes in the respiratory passages, especially the nose, were brought about during manipulation of the situation experimentally while the nasal structures were under observation.

Transitory disturbances of the character of hyperemia, hypersecretion, swelling, and obstruction in the nose in response to adverse situational stimuli probably occur from time to time in all of us and have comparatively little importance. Nasal infection is more likely to complicate sustained nasal obstruction. This has been repeatedly observed as exemplified in an instance when there occurred sustained conflict concerning the problems created by our subject's mother-in-law moving in and taking over the household.

Figure 11 portrays, in the center, nasal hyperemia, hypersecretion, swelling, and obstruction sustained for several days during such a situation. On the right and left are shown the control periods which immediately preceded and followed the period of tension, conflict and resentment.

This aforementioned reaction of nasal hyperfunction which, according to evidence from nerve cutting, appears to be due to parasympathetic predominance, was only one of two nasal patterns observed following situational stimuli. As noted, it accompanied feelings characterized by emotional conflict. More pure feeling states such as sudden abject fear, sadness and dejection were accompanied by opposite changes in the nasal mucosa, namely shrinkage of the membranes with pallor, hyposecretion, and increase in the size of the air passages. This is more of an adrenergic effect and is characteristic of the total reaction of Cannon in which all the bodily systems are mobilized for fight or flight. The heart rate and blood pressure increase. Digestion stops. Blood is shunted to the muscles. Ventilation of the lungs increases and is facilitated by the increase in size of the nasal passages. In this the organism is doing something very different from what is implied in the earlier described pattern of shutting-out by swelling of nasal structures.

An example of reaction of increased air space in a setting of fear was provided by a physician who usually reacted to situational threats with blocking off of nasal passages but who, on one occasion, when his pregnant wife had a sudden hemoptysis, was terrified and responded with shrinkage, pallor, and dryness of the nasal mucosa. (Fig. 12.)

## SUMMARY

Functional alterations in the structures in the nose of man have been studied and correlated with a wide variety of circumstances including noxious stimulation inside the nose by chemical agents, noxious stimulation of other portions of the head, interruption of nerve pathways, weeping, and numerous threatening life situations with their accompanying affective states.

In general, two patterns of disturbance of nasal function were recognized. The first involved vasoconstriction in the nose with shrinkage of the membrane and increase in the size of the air passages. Such changes in reaction to threats accompanied feelings of fear, sadness, and other emotions which, although strong, involved minimal conflict. The second type of disturbance in the nose was characterized by the initial hyperemia associated with turgescence of the erectile tissues in the turbinates and nasal septum, engorgement of the nasal mucosa and increased secretion.

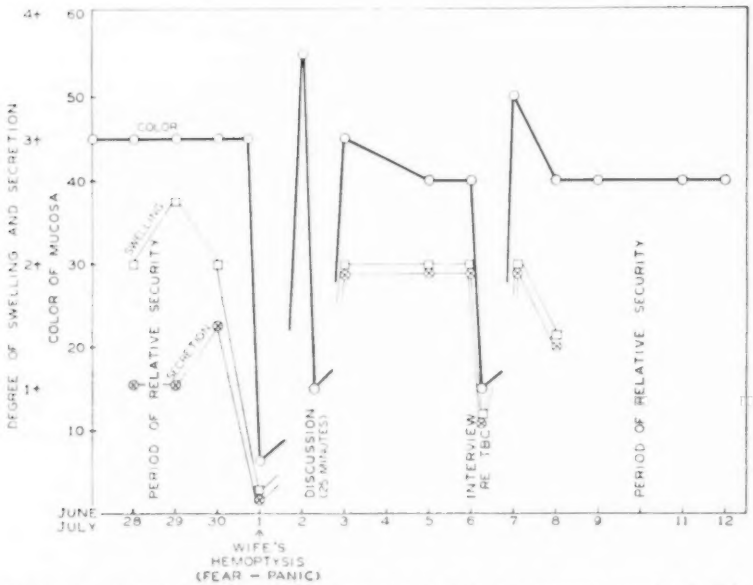



FIG. 12. — Blanching of nasal membranes with wide air passages associated with fear and panic.

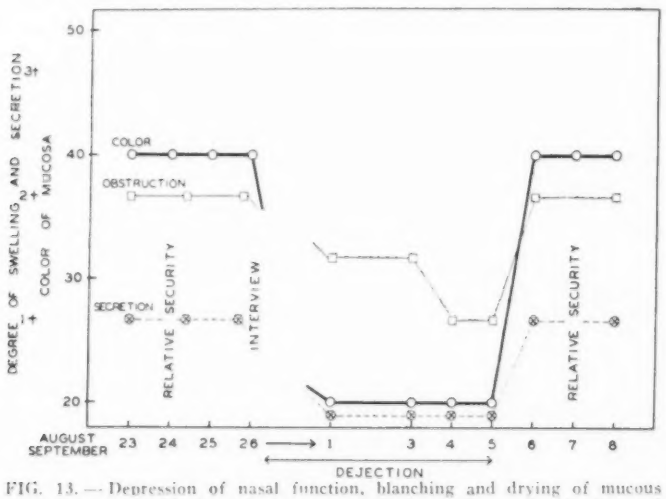



FIG. 13. — Depression of nasal function, blanching and drying of mucous membranes and widening of air passages during feelings of dejection and defeat.

These changes were accompanied by obstruction to breathing. Often, after the subsidence of hyperemia, secondary pallor ensued with the mucous membranes of the nose remaining boggy and edematous. The appearance of the nose under these circumstances resembled that of vasomotor rhinitis. In fact, it was demonstrated in an attack of hay fever which was observed from its onset, that the initial changes following inhalation of pollen involved intense hyperemia of the mucous membrane covering the septum and turbinates with

broad dilatation of the superficial vessels creating a prominent red network. Hypersecretion, swelling and obstruction quickly followed. Later the hyperemia subsided leaving the membranes pale but still swollen and edematous-looking.

As previously observed in the skin, stomach, and bladder, turgidity and hyperemia of the mucous membranes of the nose often brought about a lowering of the pain threshold.

This type of disturbance in the nose characterized by hyperemia, hypersecretion, swelling, and obstruction, occurred in reaction to local noxious stimulation as well as to other threats inducing significant conflict with resultant anxiety, resentment, anger, hostility, guilt, and feelings of frustration. Against local intrusion by dust, for example, or noxious fumes, the reaction of shutting-out and washing-away proved highly effective. Against situational threats involving interpersonal relations, however, the nasal changes afforded incomplete relief and were often productive of discomfort.

There appears to be some broad biological significance to this reaction of shutting-out and washing-away of the head end of the organism. During weeping, not only the nose but also the eyes were found to participate in this reaction, and in 3 individuals in this study, deeper structures including the bronchi constricted during threatening situations, thus participating in the bodily reaction of exclusion.

When the nasal changes in the bodily pattern of shutting-out and washing-away were unduly sustained, they gave rise to troublesome symptoms including pain and, especially when coupled with infection, to chronic nasal disease.

## REFERENCES

1. Holmes, T. H., Goodell, H., Wolf, S., Wolff, H.G.: The nose: An experimental study of reactions within the nose in human subjects during varying life experiences, Springfield, Ill., Charles C. Thomas, 1947.
2. McAuliffe, G. W., Goodell, H. and Wolff, H. G.: Experimental studies on headache: Pain from the nasal and paranasal structures, A. Research Nerv. & Ment. Dis., Proc. (1942) 23:185-208, 1943.

## DISCUSSION

EDMUND P. FOWLER, JR., M.D., New York, N. Y.: I consider it a great honor to be asked to discuss this most interesting paper. Of course, we have known for a long time that the emotions affect the nose, but I am afraid that we forget it entirely too often.

The war, if it didn't do anything else, emphasized the frequency of this type of case that has been discussed in both Dr. Semenov's and Dr. Wolf's papers. On a morning sick call one's first approach to the patient was to examine him carefully to see whether there were any flecks of red shining through the gold plate of the "brick." In civilian life I believe the doctor should always bear in mind that, even in a private office, the patient may or may not be what he seems. I read the material in Dr. Wolf's paper some months ago and since that time it is very remarkable how many cases I have found in which all of the nasal trouble was psychic in origin and how many in which a very large part of it was psychic in origin.

Dr. Wolf's and Dr. Semenov's papers were highly scientific. With that background, I think that a few cases out of my small experience might amuse you. For example, there was a shop girl who came to me with a very tightly closed nose, 3+ hypersecretion, tears in congested eyes, severe headache, a mucous membrane corresponding to 80 per cent on my Tallquist scale, and pus in the antrum. After a couple of washings, the antrum cleared. In less than a week she was well. About two months later she came up again with her nose completely closed up, but this time there was no pus in the antrum, and I thought I saw a few "little flecks of red in the gold of the brick." So I asked her, "You didn't by any chance have an emotional upset the day before yesterday when this 'cold' started, did you?"

She looked at me rather peculiarly, as patients always do when you bring up this type

of thing, but she said, "Yes, as a matter of fact, I did."

I said, "Well, what was it?"

She said, "It was a very stupid thing. The butcher tried to soak me 20 cents a pound extra for my meat. I had a fight with him."

I said, "By the way, the last time you came up here—can you remember when that 'cold' started? How did it start?"

"Oh," she said, after a pause, "I had hysterics."

I said, "What did you have hysterics about?"

"Well, I didn't want to go to Coney Island with my boy friend. He insisted on going there. It was a cold day. I didn't want to go and I had a fight with him."

This brings up the problem of what to do with the psychogenic nose. I am convinced that this girl's nasal symptoms started with emotional storms. I am also convinced that, because it lasted several days in the beginning, the bacterial flora that were normally present in the nose passed into her antrum. The therapy in this case may amuse you. I wish it were always so easy. I suggested to her that it would be better to pay the butcher 20 cents more for the meat than to pay $5.00 to me every time she came to me for a treatment. She thought this was a very good idea.

I cannot overemphasize the difficulty of therapy. Unfortunately, the psychiatrist does not help us very much. They either have to go through a couple of years of psychoanalysis or they find that the psychic disturbances are so ingrained they are unable to do much about them.

Let me mention that I think that narcosynthesis is rarely the answer in civilian life. Narcosynthesis was very simple in the Army. In the services we had a normal individual in an abnormal environment. In civilian life we are often dealing with an abnormal individual who is in a relatively normal environment. It is harder to make a change in an individual than to make a change in his environment.

There is one other aspect of the psychic nose which I think is extremely important. Suggestion works on it and often not only on the patient but also the doctor who does not realize that this is the case. We must avoid being autosuggestive ourselves. Surgery is a very strong medium of suggestion. Surgery occasionally helps these psychic cases for a time. If there is a septum with a big spur that sticks out against the side of the nose, why of course, when the nose swells up in the way that Dr. Wolf shows that they do under emotional stress, there is going to be pain. If you take the spur off there isn't going to be as much pain, and the patient will claim he is a little better. But by and large, surgery does not work on this type of case for long, and these patients love surgery. They travel around from doctor to doctor demanding that something, "Anything!" be done.

In the therapy let's not forget the power of the pink pill. The old pink pill often works very well. So does rubbing the back of the neck the way the osteopaths do it. That is mostly suggestion too. We must practice the art of medicine, and use suggestion when it is indicated, but let's not fool ourselves with pink pills, fancy machines, or with surgery.

I don't think Dr. Wolf brought out the very interesting way and the very simple way that he measures the color of the mucous membrane. He simply uses a Tallquist hemoglobinometer and reflects the light from a head mirror into the nose. So that instead of reporting a "pale" mucous membrane or a "red" mucous membrane, he can say that it resembles 20 per cent or 30 per cent, 40 per cent, or 60 per cent on the Tallquist scale. You all know that the Tallquist scale varies from printer to printer, and unfortunately, we will not be able to compare each other's results within 20 per cent, but any Tallquist scale is better than no scale.

I suggest that if you try this method you cut off some mailing tubing, three-quarters of an inch or so in size, and paste it along the hemoglobin scale, so that when you reflect your light, you get the same light on the Tallquist that you get on the septum.

Thank you.

WILLIAM C. WARREN, JR., M.D., Atlanta, Ga.: This is a very impressive and timely paper and I am delighted at the opportunity to discuss it, not that I can add anything to it from the standpoint of research, but I do want to emphasize the fact that it contains much food for thought, and we ear, nose and throat men can take back to our practice many excellent ideas if we will.

First, the role environment plays in nasal conditions. This may be the beginning of a new era when some of these more delicate problems of the nose will be solved by means of research, and I assure you there are many.

Second, mental states produce definite changes within the nose. Knowing this, we should be careful to take better histories and to give the patients a break by listening to their stories more attentively, and by weighing them, do our best to direct them in the proper channels. We can only accomplish this by deliberate thought and careful advice. Too many of us are prone

to prescribe for patients who have purely physiological symptoms. To name a few of this type of complaints: postnasal drip, dependent drainage, air-conditioning reactions which cause the nose to act like a thermostat. Handling these patients borders on psychosomatic medicine, but nevertheless the ear, nose and throat men should treat or advise the patient whenever possible because of the uncalled-for stigma so many feel when referred to a psychiatrist.

This same condition used to exist when you told a patient he had to have a blood transfusion or x-ray treatment. People thought the patient was on his last legs and was going to die. In the near future they will probably become educated that going to the psychiatrist is not so bad after all.

I should like to add to the group discussed in this paper some of the everyday cases we see in office practice which present the same signs and symptoms. Nearly all are entities within themselves. The first that comes to my mind is what many have termed "honey-moon coryza." I feel that the obstruction complained of in the nose goes hand-in-hand with the stimulation of the gonads. Similar signs and symptoms occur in some patients at the time of menstruation. Likewise, the intake of alcohol in any form in some individuals will produce the classical picture of vasomotor-rhinitis or allergy. The same can be said of pregnancy.

In closing I want to put in a good plug for research done by this same group of doctors and called to your attention about two years ago in this assembly hall, in a book published by Williams and Wilkins Company of Baltimore, called "Pain."

The primary force in this book is the Association for Research in Nervous and Mental Disease, which comprises about 400 individuals, as I understand it. Chapter 12 deals with headache mechanisms and chapter 13 deals with experimental studies on headache, pain from the nasal and paranasal structures. You will find this book most interesting and instructive, and I highly recommend it.

I feel about the nose in our line of work like the man who had just accumulated so much money he did not know what to do with it, and so, for lack of something else, he thought he should first become cultured. As the only way to do this, he considered it mandatory that he go to the old country. He finally arrived at the Louvre, in Paris, and was standing in front of Leonardo da Vinci's great painting, "The Mona Lisa." After looking at it for quite a while he began to criticize it very severely—the color was not what it should be, no luster to the eyes, the smile he could not understand, etc. The criticism became so harsh that finally his guide, a little Italian boy, said to him, "Mister, when you stand in front of the Mona Lisa, the picture is not on trial; you are on trial."

Fellows, the nose is not on trial. We are. Thank you.

STEWART WOLF, M.D., New York, N. Y.: I want to thank Dr. Fowler and Dr. Warren for their kind and illuminating remarks.

With regard to Dr. Warren's mention of the relationship of nasal swelling to sexual excitement, we had some fragmentary quantitative observations on that and they bear out precisely what he said, that during sexual excitement the noses of all the individuals whom we have observed undergo hypersecretion to a varying degree. Following orgasm, the change subsided very quickly. Without orgasm the change was sustained. I suggest that the relationship of sustained nasal hyperemia to the honeymoon may be related to the failure of adequate sexual adjustment with proper orgasm during the early days of marriage. A small series of pregnant women whom we observed showed hyperemia and hypersecretion to a varying degree. Of 20 pregnant women about half had rather marked changes. On psychological examination of these individuals a remarkable correlation displayed itself, which I don't think has any statistical significance because of the small number, but it may be suggestive. The half of the pregnant women who had troublesome changes in their noses were the half who didn't want their children.

# APPLICATION OF LOCAL PRESSURE TO THE MUCOSA OF THE MAXILLARY SINUS

J. MATHEWS ROBISON, M.D.
HOUSTON, TEXAS

THE therapeutic application of pressure to the mucosa of the maxillary sinus is based on the same fundamental physiologic principles which are utilized in other parts of the body to promote the absorption or prevent the accumulation of stagnant tissue fluid. This discussion considers some principles of the physiology and functional pathology of the sinus mucosa; the pathogenesis of allergic and purulent sinusitis; some of the related aspects of the practice of the application of local pressure to the sinus mucosa by balloons distended with iodized oil; and the selection of the patients suitable for this therapy. Pressure by a balloon was used first in the maxillary sinus by Ferris Smith to hold a skin graft in place.[10] This type of pressure has also been used in the maxillary sinus by Shea to hold its walls in position following fracture reduction.[29]

## PHYSIOLOGY AND FUNCTIONAL PATHOLOGY OF THE SINUS MUCOSA

The normal sinus mucosa is "seldom more than 0.02 mm. in thickness,"[4] and consists of a very thin stroma of loosely spaced fibroblasts surmounted by ciliated stratified columnar epithelium. The normal sinus mucosa is a nonabsorbing structure except in that it is permeable to substances which gain entrance by molecular diffusion. Dyes, particulate matter, etc., when placed in the normal sinus are carried away through the ostium on the mucous layer covering the mucosa unless some of their component molecules diffuse through into the stroma of the mucosa or some irritation produces an edema of the mucosa which dilates its interstitial spaces and opens the pathways of lymphatic absorption. The functional pathology of the sinus mucosa is related to the capability of the potential interstitial tissue spaces of its stroma to become widely dilated by edema fluid and then return to normal without losing elastic mobility and inherent contractility. The necessity for this proclivity of the stroma of the mucosa is based on the tendency of all air-containing cavities of the body to lose their air when they become severely irritated or infected.[5] This filling of air-containing cavities of the body by secretion and/or edematous membrane, and the swelling of the tissues against the integument covering the part is nature's method of increasing the tissue pressure with an incompressible fluid medium. Arterial pulsations can be transmitted better through this medium to the dilated tissue spaces and lymph channels and thereby promote lymph flow and meet the increased need for a constantly changing interstitial fluid and pericellular environment for the body cells in the involved area. A constant change in pericellular environment is necessary for efficient cell physiology. This is accomplished normally by arterial capillary filtration of water, salts, and protein; and venous capillary absorption of

This study was aided by a grant from the Longview Foundation, Longview, Texas.
From the Department of Otolaryngology, University of Texas School of Medicine, Galveston, Texas.
Presented before the Academy Section on Otolaryngology and the Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 18, 1946, Chicago, Ill.

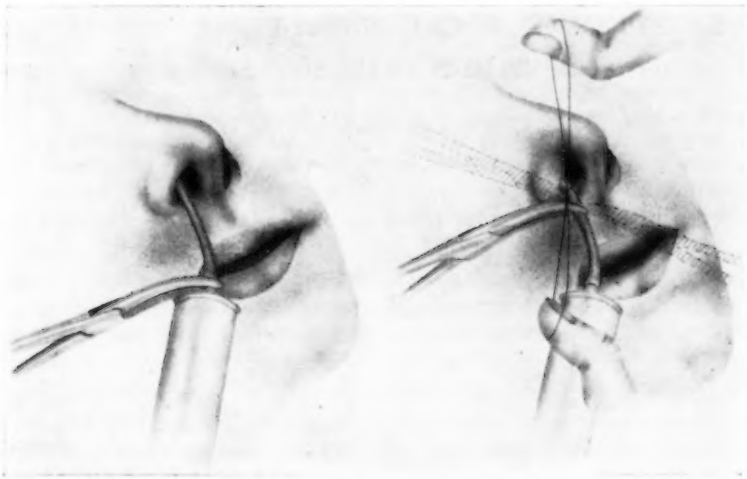


FIG. 1. — Drawings showing technic of inflating balloon in the maxillary sinus. After clipping the vibrissae from the nasal vestibule, the catheter is slipped on over the tip of the syringe and held in place with a small hemostat. The balloon is then distended with iodized oil until the patient experiences a sensation of pressure in the maxillary sinus which is neither painful nor uncomfortable. The hemostat is then clamped on the catheter near the nasal vestibule and the catheter is tied on the side away from the nasal septum with a loop of No. 7 braided silk cord. During this procedure it is important not to rotate the hemostat and catheter as shown by the dotted lines. If the catheter is rotated before it is tied the knot may impinge upon the septum where it can by pressure produce a small painful ulcer.

water, salts, and certain diffusable products; while the lymphatic system carries from the cell bed substances not readily absorbed by the blood such as products of cell metabolism, cellular detritus, bacterial products, particulate matter, and food (protein) unused by the body cells.[3] The formation of extravascular fluid from the plasma and its absorption by the veins and lymphatic system is related to the tissue pressure and the relative osmotic pressures of the plasma and extravascular fluid. Tissue pressure is gauged by the resistance a tissue offers to dilatation of its interstitial spaces by edema fluid. According to the magnitude of the tissue pressure it resists arterial capillary filtration and promotes venous capillary absorption and lymph flow. Tissue pressure increases as the tissue swells against the fascial planes of the body or the integument covering the part. Also it becomes greater as the dilated interstitial spaces become filled with fibroblasts and may be increased by the application of physical pressure to the surface of the part. Tissue pressure decreases as the tissue loses its elasticity due to long continued dilatation of its interstitial spaces by edema fluid.[3]

Retention of excess fluid in the interstitial spaces is usually due to some form of lymphatic blockage, increased venous pressure (passive congestion), lowered colloidal osmotic pressure of the plasma (hypoproteinemia), or the presence of unremovable molecules in the edema fluid. These retained molecules raise the osmotic pressure of the edema fluid to a magnitude which, under the influence of reduced tissue pressure, prevents its absorption. This latter cause for retention of excess fluid in the interstitial tissue spaces apparently is a part of the pathogenesis of certain types of sinus disease.

PATHOGENESIS OF ALLERGIC SINUSITIS

Allergic disease of the nose and paranasal sinuses is manifested clinically and pathologically by the presence of an excessive amount of fluid in the interstitial tissue spaces of the nasal and/or sinus mucosa.[7] In classifying allergic disease, emphasis should be placed on the presence or absence of irreversible changes in the involved tissue rather than the period of time the process has been present. This is particularly important in allergic disease of the sinuses because of the varying rapidity of progress of the disease in different patients. Certain patients may harbor stagnant tissue fluid in their sinus mucosa for many years with comparatively little structural change, while others will rapidly develop fibrosis and polypoid degeneration. This variable reaction found in different patients apparently is related to their sensitivity to allergens and their resistance to infection. The increased susceptibility of stagnant tissue fluid to recurrent infection forms the basis for the progressive pathologic changes which characterize allergic sinusitis.[2,7]

The patient's symptomatology, the amount of tissue fluid present in the mucosa, the length of time it is retained, and the pathologic changes which occur in the mucosa as a result of this retention form the basis for the following classification:

1. *Acute Allergic Rhinitis and Sinusitis*

Acute allergic rhinitis often is the beginning of a series of pathologic changes in the nasal and sinus mucosa which may begin in childhood or later and is characterized by nasal congestion, increased nasal secretion, and mild edema of the mucosa of the maxillary sinus and probably of the other sinuses. So long as this condition occurs episodically and the excess fluid in the mucosa is absorbed between attacks, progress of the changes in the mucosa may be retarded or delayed for many years or for the entire lifetime of the patient. However, if following an attack of acute allergic rhinitis, the sinus mucosa remains edematous, the susceptibility of this stagnant tissue fluid to recurrent infection may initiate the subacute and chronic forms of the disease.[2] This retention of fluid in the sinus mucosa apparently may begin at any age and may occur in patients who cannot be proved to be skin sensitive to any specific allergen. In the transition from acute allergic rhinitis to acute allergic rhinitis and sinusitis, three stages occur: 1) Allergic rhinitis or hayfever in which congestion of the intranasal tissues is the only pathology present. 2) In addition to severe nasal congestion, the maxillary sinus mucosa is mildly edematous and the sinus may contain a small amount of mucoid secretion. 3) Severe constant nasal congestion and edema of the maxillary sinus mucosa which, with its secretion, may approximate the capacity of the sinus. In the two latter types, infection may not be demonstrable, but recovery of the patient is definitely hastened by irrigation of the maxillary sinus. When the reaction in the sinus is mild, irrigation through the natural ostium may suffice; but if the edema of the sinus mucosa is severe and tissue fluid has been trapped in the mucosa, fistulization of the sinus mucosa by needle puncture and irrigation definitely accelerates recovery and aids in avoiding the subacute and chronic stages of the disease. Patients suffering from acute allergic rhinitis and sinusitis have not developed structural changes in their tissues and therefore should be curable. Even though some of these patients have asthmatic bronchitis, bronchial asthma, and rales, these symptoms may entirely disappear if the allergy and sinuses are properly treated. However, if the treatment is limited to anti-allergic therapy and topical applications to the nasal mucosa these patients may easily become candidates for the

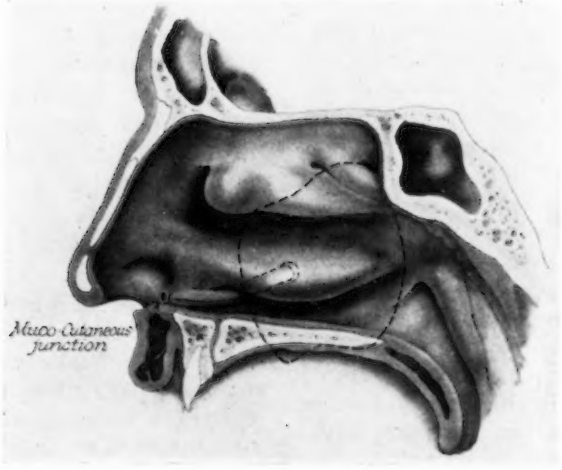



FIG. 2. — Drawings showing proper position of intranasal end of the catheter end of balloon. Since frequently it is necessary for the patient to wear these balloons for 2 or 3 months, comparatively minor errors of technic may cause considerable discomfort. If the catheter is too long it may interfere with respiration or irritate the tip of the vestibule. The catheter should be cut so that its free end rests at the mucocutaneous junction with its cut edge resting against the squamous epithelium instead of against the ciliated epithelium of the nasal septum. Occasionally patients may complain of slight odor from crusting around the catheter. This is usually easily controlled by having the patient cleanse the catheter with peroxide of hydrogen and may be minimized by applying a small amount of vaseline around the catheter. If these balloons are properly applied many patients are grateful for their improved nasal respiration and state that they are not conscious of the presence of the balloons.

subacute and chronic forms of the disease.

Two types of pathologic states occur when the sinus mucosa remains edematous following acute allergic rhinitis, and its stagnant tissue fluid manifests susceptibility to recurrent infection. These patients form two groups classed as those with subacute allergic rhinitis and subacute allergic sinusitis. They are classed as subacute, even though the pathology might have been present over a period of many years, because irreversible structural changes have not occurred in the mucosa of the nose, sinuses, or lungs. Important in the prevention and treatment of the subacute stage is the recognition of the difference between improvement or relief, and cure of the preceding acute condition. This differentiation may prove to be a problem which taxes the fortitude of the patient and physician due to the natural tendency of the patient to "let well enough alone" when he is reasonably symptom free compared to his condition during the acute stage.

### II. *Subacute Allergic Rhinitis and Sinusitis*

1. Subacute allergic rhinitis is manifested clinically by nasal stenosis due to turgescent turbinates which varies in its degree of severity and constancy. Patients with this condition often have been classed as having chronic allergic rhinitis because of the long period of time they may suffer from their allergic rhinitis. Here it is classed as subacute

because irreversible changes have not occurred in the sinus mucosa. This form of the disease may begin in certain patients early in life and require the entire life span to progress gradually to the chronic stage, while in other patients, their first nasal symptoms may not occur until middle age and the progress of structural changes in their nasal mucosa, sinus mucosa, and lungs may be so rapid that they arrive at the chronic stage with its irreversible structural changes in a comparatively short period of time. One pathologic finding which all of these patients have is retention of an excess of extravascular fluid in the mucosa of the maxillary sinuses. This edema of the sinus mucosa is always bilateral and often is not fully appreciated by x-ray examination with or without contrast media. The sinuses of these patients rarely contain excess secretion unless they are passing through an episode of infection. Although these patients may have repeated attacks of allergic rhinitis from some allergen, as long as the mucosa of their maxillary sinuses remains even slightly edematous they may suffer from repeated attacks of bacterial infection due to the susceptibility of stagnant tissue fluid to recurrent infection.[2] Theoretically, the application of pressure to the maxillary sinus mucosa of these patients during the early stages of edema before structural changes have occurred should give ideal results. Practically, the results of this procedure have been most promising. Certain patients in this category, suffering from constant nasal stenosis, nasal intonation of the voice, excessive sneezing, and frequent "catching cold" have been relieved of their symptoms completely by the application of local pressure to the mucosa of their maxillary sinuses. Pressure has been effective in patients whose symptoms were not relieved by previous treatment for their allergy or by intranasal antrotomy.

2. Subacute allergic sinusitis is manifested clinically by the same symptoms present during subacute allergic rhinitis with the added symptoms of cough, rales, and chronic bronchitis. Many of these patients have bronchial asthma. The maxillary sinus mucosa of these patients is found in two pathologic states: 1) Early in the disease it may be so edematous that it approximates the capacity of the sinus. 2) As fibroblasts proliferate to fill the interstitial spaces, the mucosa presents alternating areas of fibrosis and dilated tissue spaces. As the areas of fibrosis contract, the mucosa diminishes in volume and may remain in this redundant form for years.[8] Apparently the susceptibility to recurrent infection in these areas of dilated tissue spaces containing stagnant tissue fluid not only forms the basis for the progress of the disease, but this stagnant tissue fluid may be the source of some noxious substance, the absorption of which produces pulmonary symptoms. If this type of mucosa cannot be returned to normal by the application of local pressure, frequently it is possible to apply pressure, promote absorption of the stagnant tissue fluid, reduce the patient's susceptibility to recurrent infection, and relieve the patient's symptoms.

III. *Chronic Allergic Sinusitis*

1. Chronic allergic sinusitis follows the subacute stage as fibrosis of the mucosa becomes more extensive. Following repeated episodes of infection as the tissue pressure rises, polypi protrude from the areas of dilated tissue spaces and the volume of the mucosa may approximate the capacity of the sinus. This formation of polypi is nature's effort to drive air from the sinus cavity with an incompressible fluid medium through which pulsations can be transmitted from the arteries to the interstitial tissue spaces and lymph channels, thereby promoting absorption and lymph flow and

creating from the stagnant tissue fluid a constantly changing tissue fluid. The beneficial results following application of local pressure to the mucosa of the maxillary sinuses during chronic allergic sinusitis are related to the extent of the irreversible structural changes which have occurred in the sinus mucosa. Often it is impossible to determine by x-ray, with or without contrast media, the amount of fibrous tissue present in the edematous sinus mucosa. The best guide probably is derived from biopsy and study of the condition of the ciliated epithelium. Usually if the ciliated epithelium appears good then the application of pressure is worthy of trial. When one takes into consideration that the normal mucosa consists of a thin fibrous stroma surmounted by ciliated epithelium and the mucosa present in chronic allergic sinusitis consists of a stroma containing alternating areas of fibrosis and dilated tissue spaces containing stagnant tissue fluid, it becomes apparent that if activity of the cilia is good and the stagnant tissue fluid is removed by the application of pressure, the mucosa which remains is not unlike the normal mucosa except for its thickened stroma and its inability to become edematous and approximate the capacity of the sinus. This is particularly true if the fibrosis is not sufficiently intense to form periarteriolar whorls and interfere with proper arterial expansion and nutrition of the glands and ciliated epithelium. A sinus lined with such a mucous membrane may carry out its physiology very well except that, due to the inability of its mucosa to swell, the sinus will tend to retain secretion following infection.

FIG. 3. — Drawing showing preparation for extraction of balloon by removal of oil from balloon with curved suction canula introduced through catheter. After the balloon has been distended for 2 or 3 months with iodized oil the rubber may lose much of its contractibility. This and the high viscosity of the oil precludes the balloon from emptying itself of much of its oil when the cord around the catheter is cut. If the oil is not aspirated before traction is made on the catheter the oil may form a globular mass behind the small antrotomy opening. This may evenuate in the balloon becoming separated from the catheter when traction for removal is made, or the balloon may rupture, and portions of the thin rubber of the balloon may be left in the sinus. The balloon is removed best by anesthetizing the inferior meatus, aspirating the oil in the balloon, and exerting gentle traction on the catheter until the balloon presents itself under the inferior turbinate. The balloon is then grasped with a small hemostat and delivered.

### PRACTICAL CONSIDERATIONS

The physiology of the body is closely governed by the permeability of the natural membranes. The variables of membrane permeability as they affect osmotic pressure and molecular diffusion are: 1) time, 2) permeability and area of the membrane, 3) concentration difference across the membrane, 4) size and shape of the diffusing molecules, 5) temperature, and 6) pressure.[1]

Of these variables, local pressure and time can best be utilized to accelerate molecular diffusion in, and promote absorption of, stagnant tissue fluid. Application of local pressure to the mucosa of the maxillary sinus is accomplished by making a relatively small intranasal antrotomy under the inferior turbinate. After the postoperative reaction has subsided, usually about 2 weeks, a No. 12 Fr. self-retaining soft latex catheter

which has had its self-retaining head enclosed in a small distensible, natural rubber balloon is inserted through the antrotomy opening into the sinus. After the balloon has been in place for 48 hours, it is distended with iodized oil until the patient feels a sensation of pressure which is neither painful nor uncomfortable. Iodized oil is used for distending the balloon because its specific gravity (1.35) is higher than the specific gravity of the blood plasma (1.027). This allows the balloon to displace the secretion in the sinus and produce a certain amount of pressure on the edema fluid in the sinus mucosa by its own weight. The patient's sensation of pressure is the only guide used to determine how much pressure to apply. The balloons should not be distended immediately upon its insertion because infection may be carried into the sinus with the balloon or its presence may irritate the sinus mucosa. This may cause the mucosa to swell. If the balloon occupies all of the air space in the sinus, the added swelling of the mucosa may raise the pressure sufficiently to produce pain. The balloon should be distended slowly to allow it to displace any secretion present in the sinus. The secretion may be so viscid that it may not be displaced promptly, or possibly not until hours later, at which time the intrasinus pressure may be lowered. For this reason, oil should be added again within 48 hours after the first instillation. If possible, the balloon is distended further with iodized oil at one or two week intervals, and may be left in place, if well tolerated by the patient, for six to twelve weeks. The optimum time for leaving the balloon in place is probably about 8–12 weeks, followed by a rest period of four to six weeks. It may be reinserted if stagnant tissue fluid is still present in the sinus mucosa. Most of the difficulty patients experience while wearing these balloons arises from irritation of the nasal septum and vestibule by the intranasal end of the catheter. This may be minimized by cutting the catheter so that its intranasal end will rest about the mucocutaneous junction. As oil is added the intranasal end of the catheter may tend to protrude toward the vestibule and might have to be cut off again. The knot in the No. 7 braided silk cord used to tie the end of the catheter should be tied on the side of the catheter away from the nasal septum to avoid the knot producing a pressure ulcer on the septum. Much of this type of difficulty will be obviated when these balloons are manufactured commercially from thin walled catheters with less resilience than those used by the urologist, which are the type being used at present.

The mucosa of the maxillary sinus becomes edematous following operative trauma, infection, or mechanical irritation. The degree of increase in volume of which the mucosa is capable following these instances, may be used as a criterion of the physiologic efficiency retained by the mucosa. It may give one a reasonable idea of the amount of fibrosis present in the stroma of the mucosa and provides information of prognostic value in determining the ultimate results possible to obtain by the application of pressure. For instances of the two extremes: as pressure is applied to the sinus mucosa of a patient who has subacute allergic rhinitis and sinusitis, the mucosa of his maxillary sinuses may be so mildly edematous that the thickening is not discernable by x-ray with contrast media; however, the postoperative reaction, the irritation of the balloon or secondary infection may cause the mucosa to swell until it approximates the capacity of the sinus. If the reaction is severe the balloon should be removed and reinserted after the reaction subsides. In this instance the marked swelling of which the mucosa is capable indicates a good prognosis be-

cause it means that extensive fibrosis has not occurred in the stroma of the mucosa. Another patient who has chronic allergic or purulent sinusitis or both, may show mild thickening of his sinus mucosa by x-ray with contrast media. If the sinus mucosa of this patient shows no tendency to swell following surgical trauma or insertion of a balloon in his sinuses it may indicate extensive fibrosis of the stroma of the mucosa with resulting irreversible structural changes in its cilia and glands.

Two questions are often asked concerning the application of pressure to the maxillary sinus mucosa. 1. How does the application of pressure to the maxillary sinus mucosa relieve similar pathology in the other sinuses? The answer is that it does not relieve irreversible structural changes in the other sinuses. However, the beneficial effect on the other sinuses of relieving certain pathology present in the maxillary sinus is a fact well known clinically. The maxillary sinus frequently is as large as all of the other sinuses combined and when it is pathologic it may demand more blood supply and more movement of extravascular fluid than any or all of the other sinuses. This results in greater turgescence of the intranasal structures and a more constant effort on the part of the lymph pump mechanism to maintain a constantly changing pericellular environment for the cells in the mucosa. Acute purulent pansinusitis usually subsides promptly if the maxillary sinus alone is treated. However, during this disease, if treatment is administered solely to one of the other sinuses not only is the treatment usually ineffective in relieving the maxillary sinus, but it also may fail to relieve the sinus toward which the treatment is directed, as long as the maxillary sinus remains involved. The other sinuses are normally smaller than the maxillary sinus, and though the stroma of the maxillary sinus mucosa is usually slightly thicker than the stroma of the other sinuses, the mucosa of the smaller sinuses can more easily swell against their surrounding bony walls, and thereby increase the tissue pressure in the mucosa and prevent the accumulation of stagnant tissue fluid. When normal sinuses become severely infected, although some of their secretion may be removed by ciliary action, they depend for drainage upon displacement of the air and secretion in the sinus cavity by edematous membrane. The edema occurs first and to its greatest extent, in the portion of the sinus away from the ostium which forces the secretion toward the ostium. This explains why large sinuses tend to develop chronic infection; their size may be so great that their mucosa cannot swell sufficiently to effectively approximate their capacity and thereby increase the tissue pressure of the mucosa. This leads to tissue fluid stagnation and retention of secretion.

2. What beneficial results are obtained from application of pressure which are not obtained by intranasal antrotomy alone? Important in answering this question is the differentiation between improvement or relief of the patient's symptoms, although the patient retains some of the pathology such as edematous sinus mucosa; and cure of patient's symptoms with return of the patient's sinus mucosa to normal or near normal and interruption of the progressive characteristics of the disease. If the patient's symptoms and pathology can be cured by less radical procedures and the progress of the pathologic process in the sinus halted, then the application of pressure to the sinus mucosa would be superfluous. Clinical experience has shown that certain patients are not relieved of certain symptoms and pathology by intranasal antrotomy until pressure has been applied to the mucosa of their maxillary sinuses. Some of these

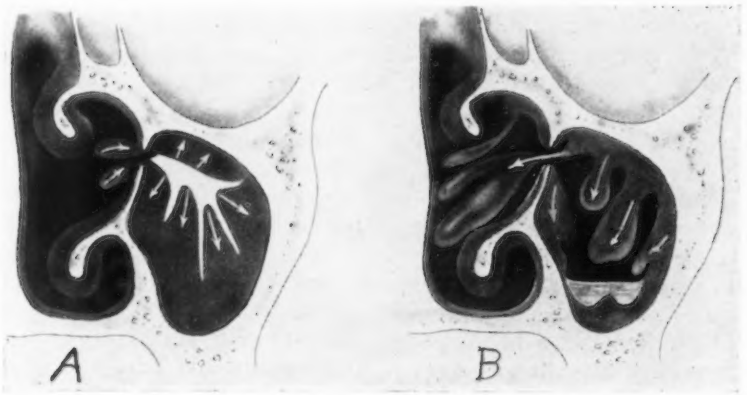



FIG. 4. — Drawings showing the lines of force exerted during acute and chronic edema of the mucosa of the maxillary sinus. A) Drawing showing the recently normal sinus mucosa which, due to acute infection has become edematous and is raising its tissue pressure by swelling against the sinus secretion and surrounding bony walls. Acutely formed polypi are shown as mechanical protrusions of the sinus mucosa through the ostium of the sinus. The white arrows represent the direction of force exerted by the normal elasticity and contractility of the acutely inflamed mucosa. The inherent contractility of the normal mucosa is the force which promotes the absorption of excess extravascular fluid, spontaneously reduces acute polypus formation and returns the sinus mucosa to its normal thickness during resolution from acute infection. B) Drawing showing the sinus mucosa which has been edematous for a protracted time and contains alternating areas of fibrosis and dilated tissue spaces. During recurrent infections in the stagnant tissue fluid the tissue pressure rises and polypoid protrusions of the mucosa occur from the areas of dilated tissue spaces. Since this mucosa, due to its long continued edematous distention, has lost its inherent elasticity and contractility there is little force exerted to promote absorption of the excess tissue fluid until the polypi and secretion completely fill the sinus cavity. The direction of the dominant lines of force exerted by the increased tissue pressure due to recurrent infection is represented by the white arrows.

patients have had their intranasal antrotomy openings present for months and one for four years without relief of symptoms.

## SELECTION OF PATIENTS FOR THE APPLICATION OF PRESSURE

Since the application of pressure to the mucosa of the maxillary sinuses was designed to promote the absorption of stagnant tissue fluid from the mucosa of the maxillary sinus, and since allergic disease of the sinuses is characterized by accumulation in the sinus mucosa of an abnormal amount of fluid in its interstitial spaces it follows that many of the patients to whom this treatment is applicable can be proved allergic by skin tests. If they are negative to skin tests they may be judged allergic by their history, clinical symptoms, nasal cytology, etc. The difficulties encountered in selecting patients suitable for the application of pressure are related to the training and experience of the otolaryngologist in allergy and the training and experience of the allergist in otolaryngology. The entire problem would be simplified and the interest of the patient served much better if all otolaryngologists were well trained in allergy and diseases of the chest.

The application of pressure to the sinus mucosa is not designed to supplant any less radical standard practice but rather is an adjunct to more conservative treatment, to be used only when these measures fail. If the following order of treatment is used in patients suffering from allergic sinusitis, those pa-

tients not relieved by the more conservative measures will automatically become candidates for the application of local pressure to the mucosa of their maxillary sinuses.

1. Treatment for their allergy. The allergist's treatment of patients with allergic rhinitis and sinusitis is often unsatisfactory, and may be attributed to lack of cooperation of the patient, delay of the patient in seeking treatment and lack of mastery by the medical profession of the knowledge necessary to cure successfully all allergic disease. The lack of training of the otolaryngologist in allergy has been attested by the numerous operations which have been done on the nose and sinuses in a futile effort to correct an allergic condition which might have been controlled by treatment for allergy. The best interest of the patient is served by close cooperation between the allergist, internist, and otolaryngologist.

2. When treatment for allergy is ineffective, these patients should have their nasal secretion examined, their maxillary sinuses irrigated via the natural ostium or by needle puncture, and an x-ray study with contrast media in their maxillary sinuses. If treatment by irrigation of the maxillary sinuses with or without the use of antibiotics suffices to relieve the patient's symptoms, returns the sinus mucosa to normal, and arrests the progress of the disease, then additional treatment is unnecessary.

3. Intranasal antrotomy is the next therapeutic procedure offered these patients and alone is sufficient to afford many of them so much relief that they are loathe to accept further treatment because their condition is satisfactory compared to their status before the antrotomy opening was made. There are certain patients in this category who although they may be improved by intranasal antrotomy, continue to have excessive drainage for their sinuses, constant or intermittent nasal stenosis, nasal intonation of their voice, recurrent formation of nasal polypi, and the pulmonary symptoms of cough, excessive expectoration, "wheezing in the chest," episodes of "catching cold" and paroxysms of bronchial asthma. Patients suffering from these symptoms comprise a large percentage of those who think that "once you have sinus trouble you always have sinus trouble." This type of patient usually has run the gauntlet of the allergists and otolaryngologists in his community and has been advised that there is no relief for him except radical maxillary sinus surgery.

4. Local pressure should be applied to the mucosa of the maxillary sinuses of patients in this category in an effort to promote absorption of stagnant tissue fluid. Adequate doses of autogenous, stock, and virus vaccines should be administered to increase their resistance to infection.

5. Radical maxillary sinus surgery should be reserved for these patients whose maxillary sinus mucosa has developed such extensive fibrosis and/or polypoid degeneration that the physiologic functions of the cilia and glands have become so attenuated that the sinus cavity and mucosa is a constant harbor for infection in spite of the application of all of the more conservative therapeutic measures.

6. The application of local pressure to the mucosa of the maxillary sinus during chronic purulent sinusitis has not been tried sufficiently to formulate an opinion concerning its value. Pressure has been applied in enough patients with primary chronic purulent sinusitis to note that in some of them, whose structural changes are not far advanced, the application of pressure evidently has a favorable effect on the nutrition of the mucus secreting glands of the mucosa. Ordinarily the clinician judges the progress of infection of any mucous mem-

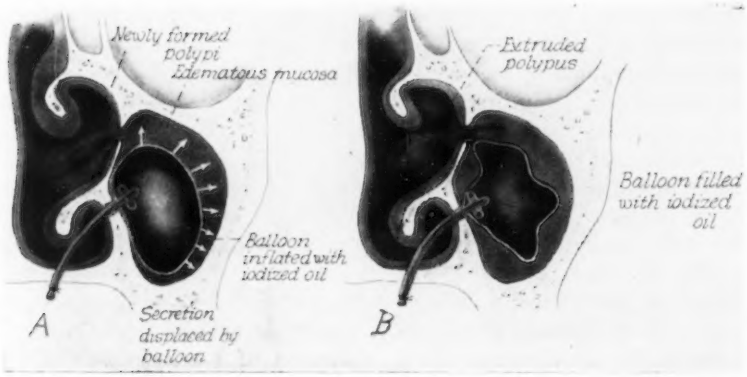



FIG. 5. — Drawings showing mechanism of formation of polypi which are present in the middle meatus following the application of local pressure to the mucosa of the maxillary sinus. A) Newly formed polypi appear in the middle meatus following the application of pressure to the uniformly edematous sinus mucosa. These polypi may form over a period of a few weeks and histologically show disruption of the septa between the dilated tissue spaces. The white arrows show the direction of the lines of force exerted by the balloon. These lines of force are in the same direction as those exerted by the inherent contractility of the previously normal recently infected mucosa. Compare with Fig. 4A. B) A polypus which has been present in the maxillary sinus for some time may be prolapsed through the middle meatus as the balloon is filled with oil. This may result in passive congestion, strangulation and hemorrhage into, or gangrene of the polypus. This has not proved to be a serious complication but may cause moderate pain which has been easily relieved by removal of the polypus without removing the balloon.

brane by the progressive changes in the secretion as it passes through the catarrhal, serous, purulent, and mucoid stages. If the mucoid stage does not appear and the secretion continues to be purulent, the mucosa usually is considered to be beyond repair. When this occurs during maxillary sinusitis, radical surgery often is advised. Following the application of pressure certain of these patients who previously had secretion which contained little or no mucus would have secretion which contained large amounts of mucus and the secretion would then entirely disappear after several irrigations. Application of local pressure to the mucosa during maxillary sinusitis of dental origin has not been tried and there is very little reason why it should be beneficial in such patients since most of them either get well following proper attention to their teeth or have irreversible structural changes in their mucosa or alveolus which cannot be affected by the application of pressure.

## RESULTS OF TREATMENT

Allergic disease of the maxillary sinuses frequently is characterized by slowly progressive pathologic changes in the sinus mucosa which may encompass most of the patient's lifetime. Therefore it is difficult and often erroneous to draw final conclusions and make accurate observations concerning the ultimate beneficial effects of any type of therapy administered for a short period of time to these patients. This becomes more apparent when one takes into consideration that certain patients harbor excessive amounts of tissue fluid in the mucosa of their sinuses for many years with minimal symptoms, while other patients will manifest severe signs and symptoms with apparently the same pathology. The symptoms of these patients may consist of intermittent or constant nasal stenosis, nasal intonation of the voice, frequent "catching cold," excessive nasal secretion, "sinus trouble," cough, bronchitis, and bronchial asthma. Examination may reveal turbinate tur-

gescence, nasal polypi, discoloration of the nasal mucosa, an increase in eosinophils or polymorphonuclear leucocytes in the nasal secretion, mucopurulent secretion in the maxillary sinuses, thickening of the mucosa of the maxillary sinus, and rales in the chest. Early in the disease many of these symptoms and signs occur episodically with symptom free intervals; however, following recurrent infections, the patient's difficulties often become continuous. The most beneficial result of the application of pressure is found in the lessening of the patient's susceptibility to repeated respiratory infections and constant nasal symptoms. Certain patients who have suffered for years from one or all of the symptoms and signs enumerated have been relieved for protracted periods. In some patients this relief has continued until they developed another infection, following which pressure was again applied. In others, complete relief of symptoms has been constant until the present time, and apparently the progressive characteristics of the pathology have been arrested or at least interrupted.

Certain patients with mild edema of their sinus mucosa may be relieved completely following one application of pressure while others who have more marked edema may require two to four applications of pressure at intervals over a period of six months to two years. The symptoms, relief for which the patient is most grateful, are: nasal stenosis due to turgescence of the turbinates, recurrent formation of nasal polypi, excessive nasal secretion, and the bronchopulmonary symptoms of cough, chronic bronchitis, and bronchial asthma. When constant turgescence of the turbinates accompanies edema of the sinus mucosa it is a manifestation of the effort of the lymph pump mechanism to remove from the lymph bed of the sinuses products of cell metabolism, bacterial products, excess protein, and other substances present in the extravascular fluid. The turbinate tissue accomplishes this by increasing its volume, and raising its tissue pressure by impinging against the nasal walls so that arterial pulsations can be transmitted to the lymph vessels by hydrostatic pressure. When pressure is applied to the edematous sinus mucosa and its tissue pressure raised, the turgescence of the turbinates may subside. This reduction in the swelling of the turbinates following the application of pressure to the maxillary sinus mucosa is evidenced in the improvement in nasal respiration which these patients enjoy in spite of the presence of the catheter in the inferior meatus. After pressure has been applied to the edematous mucosa of the maxillary sinus for a few weeks newly formed polypi may appear in the middle meatus, or polypi in the sinus may be forced through the meatus. These polypi may be the cause of nasal stenosis and should be removed either before or after the balloons are removed. The lack of consistency in the formation of polypi in different individuals and the varying time element necessary for their formation render it impossible to evaluate definitely the effect of the application of pressure upon the interruption or stopping of polypus formation in the maxillary sinus and middle meatus. However, certain patients who have had polypi in their middle meatuses for years have been free of polypi and have had normal appearing middle meatuses for a protracted time following removal of the newly formed polypi. Histologically, polypi formed slowly over a long period of time by nature show fluid-filled, dilated tissue spaces separated by thin connective tissue fibrils. The arrangement of these tissue spaces gives a "chicken wire" appearance to the tissue. Newly formed polypi produced by the application of pressure for a few weeks to the edematous mucosa show a disrup-

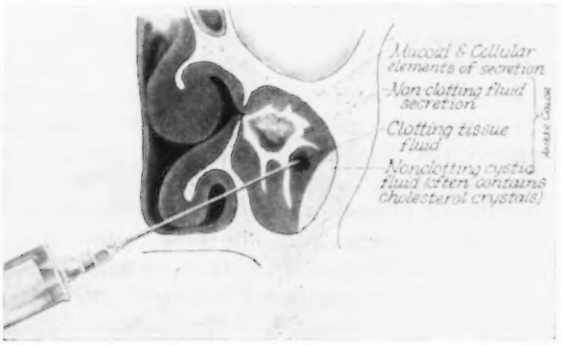

Mucoid & Cellular elements of secretion
Non-clotting fluid secretion
Clotting tissue fluid
Nonclotting cystic fluid (often contains cholesterol crystals)

FIG. 6. — Drawing showing various fluids which may be obtained from the maxillary sinus by aspiration and fistulization of the sinus mucosa during certain acute pathologic processes. During acute empyema of the maxillary sinus the secretion may be homogenous. Later, as the sinus drains, the secretion may separate into two parts. The mucoid and cellular elements of the secretion become clumped together and may be surrounded by the more fluid, amber-colored, non-clotting component. During acute purulent and allergic maxillary sinusitis, the sinus mucosa raises its tissue pressure by swelling against the inherent elasticity of the mucosa, the sinus secretion and the surrounding bony walls. As this occurs the interstitial tissue spaces in the stroma of the mucosa become widely dilated with fluid which may be free fluid and often may be aspirated following fistulization of the mucosa by needle puncture. This tissue fluid differs from the clear amber-colored component of the secretion and cystic fluid in that its cytology is different and it clots promptly after being withdrawn. Fistulization of the sinus mucosa is a valuable therapeutic procedure particularly during acute allergic sinusitis. During this disease the tissue fluid often resists absorption and tends to remain trapped within the sinus mucosa for a protracted period of time. Local pressure should not be applied to the sinus mucosa during any acute inflammatory process. It is applied best during the subacute stage before irreversible structural changes have occurred in the mucosa.

tion of the septa around the interstitial tissue spaces so that the tissue and its contained fluid appear homogeneous. These newly rapidly formed polypi so far have appeared only after application of pressure to patients with marked edema of the sinus mucosa and are comparable in the mechanism of their formation to the polypi occurring in the middle meatus during acute sinusitis. These latter polypi are the result of herniation of the maxillary sinus mucosa through the ostium as the mucosa becomes edematous and approximates the capacity of the sinus in an effort to raise its tissue pressure. Acute polypi due to the unimpaired elasticity of the stroma of the mucosa, usually disappear as the acute process subsides, but the newly formed polypi produced by the application of pressure applied to the sinus mucosa remain in the middle meatus because the tissue has lost its inherent elasticity due to long dilatation of its interstitial spaces by edema fluid.

## DIFFICULTIES ENCOUNTERED DURING TREATMENT

1. Temperament of the patient. Patients react to balloons in their sinuses in direct proportion to the length of time they have had allergic sinusitis and how severe their symptoms have been. If their symptoms are of recent origin and consist principally of subacute allergic rhinitis they may complain of comparatively trivial discomforts such as an occasional slight odor from crusting

around the tube in the nose. Crusting around the intranasal end of the catheter may be minimized by cleaning the nasal vestibule and around the tube with hydrogen peroxide. If their symptoms are of long duration and are associated with asthma, cough, rales, and nasal polypi, they rarely complain of any discomfort, and often state that they are not conscious of the presence of the balloons. This is particularly true of those who have had extensive treatment for allergy and sinusitis without relief and are faced with the alternative of radical sinus surgery.

2. Application of excessive pressure. Usually this occurs when the physician fails to take into consideration the degree of swelling of which the sinus mucosa is capable when irritated or infected. If a pressure balloon is inflated to comfortable pressure in a maxillary sinus, the mucosa of which is capable of greatly increased swelling, the irritation of the balloon may cause enough additional edema and pressure to produce discomfort or pain. Therefore it is important, unless one knows the degree of fibrosis present in the mucosa of a certain maxillary sinus, not to inflate the balloon until after it has been in the sinus for two or three days. When the balloon is inflated, thick viscid secretion which may not be displaced readily may be displaced after a few hours, reduce the intrasinus pressure and defeat the purpose of the inflation.

3. Infection and closure of the antrotomy opening. Infection may be carried into the maxillary sinus with the balloon when it is inserted. This becomes manifest by development of sensations of marked pressure and at times pain in the sinus with purulent secretion from the sinus. If the inflammatory reaction is severe and the antrotomy too large, the balloon may be extruded. This accidental infection often subsides promptly but may frequently be avoided by instilling 2 cc. of saline containing 5000 units of penicillin in the sinus before the balloon is inserted. A small antrotomy opening may tend to close before the balloon is inserted. When this occurs, it is usually permissible to slightly enlarge the opening to admit the balloon. However, this should only be done if the first operation was performed as long as two to four weeks previously. There is no more dangerous intranasal procedure than frequently repeated operations on the same area in the nose. Repeating operations in this area at short intervals is one of the most frequent causes of osteomyelitis of the bones surrounding the sinuses. Antibiotics should be administered as a prophylactic measure before and after the antrotomy, particularly if the patient is having asthma, and at any other time infection occurs during the application of pressure.

4. Herniation of the balloon into the nose occurs when the opening of the maxillary sinus into the middle meatus is large, the intranasal antrotomy opening is too large, or too much pressure is applied in the balloon. Herniation produces nasal stenosis and may, when it occurs through the middle meatus, produce lymphatic and venous congestion of the anterior group of sinuses. This is usually manifested by mild symptoms of frontal sinusitis. Herniation through the antrotomy opening may be minimized by using a balloon which contains a self-retaining catheter head which is of the proper size to completely occlude the intranasal window, and avoiding the application of too much pressure.

5. Nasal stenosis due to swelling of the turbinates usually subsides after pressure has been applied to the sinus mucosa long enough to accelerate molecular diffusion in the stagnant tissue fluid. Continuation of nasal stenosis is usually due to boggy turbinate tissue which has lost its contractility, to un-

removed or newly formed polypi or to the intranasal end of the catheter being too long. As pressure is applied in the maxillary sinus, intrasinus polypi may be forced through the ostium into the middle meatus. This may cause strangulation of, or hemorrhage into the polypi, either of which may be associated with pain and nasal stenosis which may not occur until some time after pressure is applied. This complication has not proved troublesome and is easily relieved by removal of the polypi. Small granuloma which occasionally form around the catheter in the inferior meatus may be removed after the balloon is removed. The rubber used for constructing these balloons has shown little tendency to disintegrate due to long contact with iodized sesame, or peanut oil. When these balloons are produced by various manufacturers, the rubber should be observed for its oil resisting qualities.

## SUMMARY

1. Some aspects of the physiology and functional pathology of the sinus mucosa are discussed with particular emphasis on the ability of the previously normal sinus membrane, when severely infected, to become edematous, displace the air and secretion in the cavity, and approximate the capacity of the sinus. By impinging against the bony walls of the sinus, the mucosa is able to raise its tissue pressure, which according to its magnitude retards capillary filtration and promotes capillary absorption, molecular diffusion, and lymph flow. This is one of nature's methods of producing a constantly changing pericellular environment and preventing accumulation of stagnant tissue fluid in the interstitial spaces of the sinus mucosa.

2. Allergic disease of the paranasal sinuses is characterized by accumulation in the interstitial spaces of the mucosa of an abnormal amount of fluid. As this fluid becomes stagnant and manifests increased susceptibility to recurrent infection, it participates in a series of progressive pathologic changes recognized clinically as subacute and chronic allergic rhinitis and sinusitis.

3. A classification of allergic rhinitis and sinusitis is offered based on the presence or absence of irreversible structural changes in the sinus mucosa without respect for the length of time the pathologic process has been present.

4. The application of local pressure to the mucosa of the maxillary sinus by balloons distended with iodized oil is considered in its theoretical and practical aspects. Local pressure applied to the mucosa of the maxillary sinus is most efficient in promoting absorption of stagnant tissue fluid and relieving the patient's symptoms if the pressure is applied early during the edematous stages of allergic disease before proliferation of fibroblasts in the stroma of the mucosa has occurred. Accordingly, the most spectacular results are obtained following the application of pressure during subacute allergic rhinitis and sinusitis. The results obtained following the application of pressure during chronic purulent and allergic sinusitis are related to the degree and extent of irreversible structural changes present in the sinus mucosa. The efficiency of the application of local pressure to the sinus mucosa in reducing the patient's susceptibility to recurrent sinus infection is described.

5. The order of treatment used in selection of patients suitable for the application of local pressure to the mucosa of their maxillary sinuses is detailed, and the results of, and difficulties encountered in this therapy are considered.

## REFERENCES

1. Davson, Hugh and Danielli, J. F.: The permeability of the natural membranes, New York, The Macmillan Company, 1943.

2. Drinker, C. K., Field, M. E., Ward, H. K. and Lyons, C.: Increased susceptibility to local infection following blockage of lymph drainage, Am. J. Physiol., 112:74-81 (May) 1935.
3. Drinker, C. K. and Yoffey, J. M.: Lymphatics, lymph and lymphoid tissue, Cambridge, Mass., Harvard University Press, 1941, pp. 4, 26, 53, 55, 58, 80, 83, 189-190, 284, 286, 289, 290, 291, 293, 300, 301, 305, and 307-309.
4. Piersol, G. A.: Normal histology, Philadelphia, J. B. Lippincott Company, ed. 2, 1894, p. 403.
5. Robison, J. M.: Lymphangitis of the retropharyngeal lymphatic system, Arch. Otolaryng. 40:385-395 (Nov.) 1944.
6. Robison, J. M.: Physiology and functional pathology of the lymphatic system applied to allergy of the nose and paranasal sinuses, J. Allergy, Vol. 17, No. 2, 53-69 (March) 1946.
7. Robison, J. M.: Pressure treatment of allergic sinusitis, To be published in Arch. Otolaryng.
8. Robison, J. M.: Lymphangitis of the mucosa of the paranasal sinuses, Texas State J. Med., 40:193-199 (July) 1944.
9. Shea, John J.: Management of fracture involving paranasal sinuses, J.A.M.A., 120: 745-749 (Nov.) 1942.
10. Smith, Ferris: Plastic surgery, its interest to otolaryngologist, J.A.M.A., 75:1554-1559 (Dec.) 1920.

## DISCUSSION

FLETCHER D. WOODWARD, M.D., Charlottesville, Va.: I have followed Dr. Robison's work since its inception with great interest I have envied its original concepts and his ability and persistence in his study of this problem; so today, I am very glad to be able to hear his mature and sound conclusions, and to compliment him on having accomplished such a fine piece of investigative work. The clinical aspects are most interesting and helpful to those patients who are suitable for its application. We are using his method now at the University of Virginia Hospital, and can verify the soundness of his observations. Our greatest trouble has been in the making of makeshift balloons. Whenever they become available commercially, clinical application will be simplified and the results improved.

In spite of the valuable clinical aspects, I still feel that his greatest contribution has been in a newer concept of physiology and functional pathology. When the complete paper can be studied, after publication, I feel sure that the stimulus and knowledge received from this work will lead many other investigators to further discoveries in this much neglected field.

In recent years there has been a tendency to irrigate all maxillary sinuses through the natural ostia, and although this approach is still the method of choice in the majority of cases, his argument for the use of the straight needle when fistulization of the sinus mucosa is indicated, is most interesting.

The decision as to when the pathologic changes in the sinus mucosa have become irreversible has always been most difficult to make, for reversible changes are amenable to medical and surgical treatment with conservation of the mucosa; whereas irreversible changes necessitate an external operation with removal of the mucosa. Since an intact epithelium and the ability to swell and later return to normal are the criteria for making this decision, the application of balloon pressure before deciding on an external operation is an important contribution in determining the status of the mucosa, and will no doubt save many patients from such a procedure.

Additional work on the anatomy of the lymphatics and on the function of the sympathetic and parasympathetic nervous systems of the head and neck is needed, to correlate with the physiologic and pathologic concepts as brought out by Dr. Robison; then, as biochemistry advances, so will our understanding of these complex problems advance, and therapy will be both logical and more effective.

ERNESTO RAFFO, M.D., Lima, Peru: (Discussion not available.)

J. M. ROBISON, M.D., Houston, Texas: Thank you, Dr. Woodward and Dr. Raffo, for your very constructive discussion of my paper. At this stage during the development of this type of treatment for maxillary sinusitis, apparently the best results are obtained in allergic sinusitis when the pathologic change in the sinus mucosa is limited to simple edema. Beneficial results also have been obtained in primary purulent maxillary sinusitis with delayed resolution. The symptoms relieved by the application of local pressure to the mucosa of the maxillary sinuses have all been related to the susceptibility of the stagnant tissue fluid in the sinus mucosa to recurrent infection. If by applying pressure to the mucosa of the maxillary sinuses, the incidence of infection is reduced then the patient may be relieved of those symptoms which are so often present during or associated with sinus infection, such as nasal stenosis, cough, bronchitis and bronchial asthma.

# THE MESOPHARYNX IN DIAGNOSIS

W. Burbank Woodson, M.D.
TEMPLE, TEXAS

In this paper I intend to present some results of observation of the mesopharynx. In spite of or perhaps because of its obvious nature and position, very little attention has been given in textbook descriptions of this area and no attempt made to differentiate certain signs and symptoms as definite diagnostic entities. Following in method the ophthalmologist's evaluation of the findings of the fundus of the eye-ground, I intend to present an evaluation of some of the findings of the mesopharynx with their corresponding diagnostic implications. In order to emphasize the value of every day observation, the study will include only that portion of the oropharynx that can be seen with the head mirror.

The anatomy of the area will be considered in detail, as a thorough view of the anatomical relationships is essential for an evaluation of the clinical findings. Most textbook pictures give only a hazy view of the mesopharynx, a typical example being shown by figure X. Free use has been made in this paper of pertinent photographs and drawings from recognized sources. Grouping these with the original sketches and photographs presented in the paper, will, I hope, facilitate reference to this area. In this connection I was fortunate in securing the services of Dr. Lewis Waters, Professor of Medical Art and Visual Education at the Southwestern Medical College.

## HISTORY

With the exception of the inadequate anatomical descriptions referred to above, there seems to be no available material on the mesopharynx *per se*. Many authors refer to this area in a general description of the pharynx, and these remarks are in no way intended to exclude the many references made to the area as part of some excellent discussions of the pharynx. Dr. Oscar V. Batson, in his scholarly review of the veins of the pharynx, states that he could find only four original accounts and two textbook summaries which include a discussion of the true pharyngeal veins. In view of this dearth of material on a related subject and so little extant descriptive material on the area under consideration, further study of the mesopharynx seems in order.

## EMBRYOLOGY

In Mall's specimen with seven somites (fig. 1) the anterior end of the foregut comes in contact with the ectodermal pouch called the stomodeum or buccal sinus. There the ectoderm unites with the entoderm to form the pharyngeal or buccopharyngeal membrane. (See fig. 1 A and B for view of the early location of this membrane which is the analogue of the mesopharynx.) This small 2.15 mm. embryo has long since been considered pathological; it is given here as an easy view of the first signs of the mesopharynx. The pharynx is later separated from the mouth by the already perforated pharyngeal membrane (shown in fig. 1-A). Four pharyngeal pouches are present at this time. Another figure (2) is a graphic reconstruction of an embryo with twenty-three paired somites in a sagittal section of the digestive tract and shows the pharynx and the pharyngeal membrane mak-

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

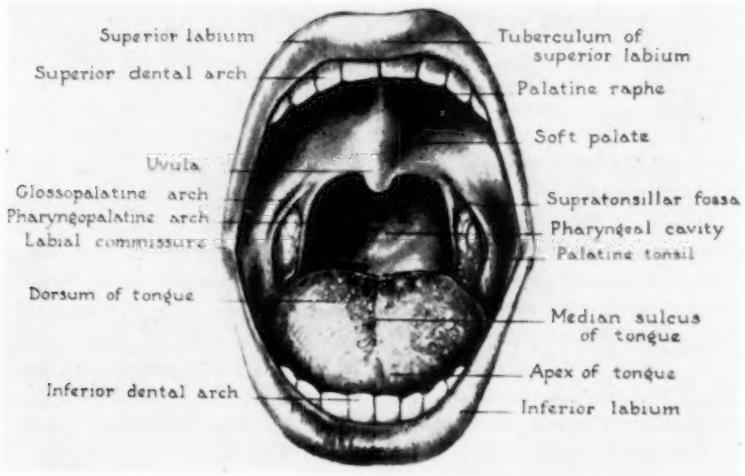



FIG. X. Example of usual textbook picture of mesopharynx.

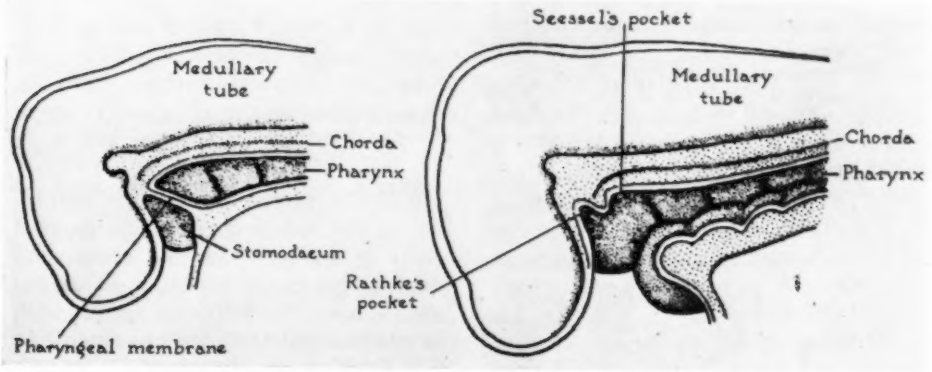



FIG. 1 (A and B) Mall's specimen with seven somites.

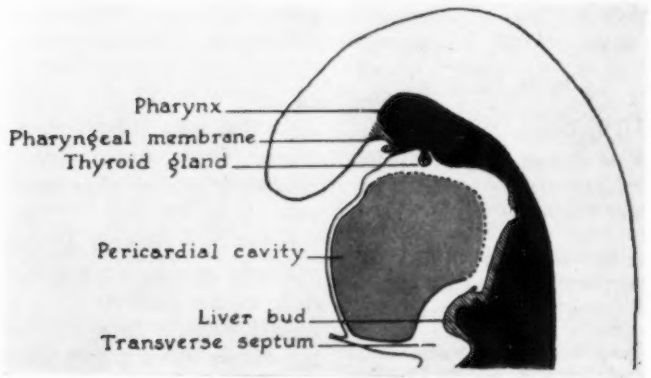



FIG. 2. Embryo with 23 paired somites in sagittal section (pharynx and pharyngeal membrane making their connection with the mouth).

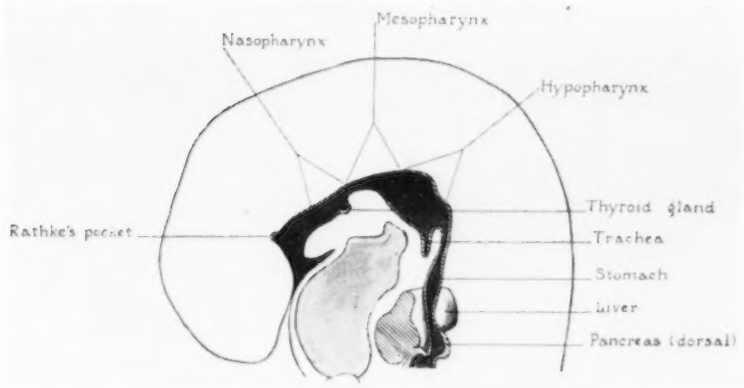



FIG. 3. Mesopharynx can be located

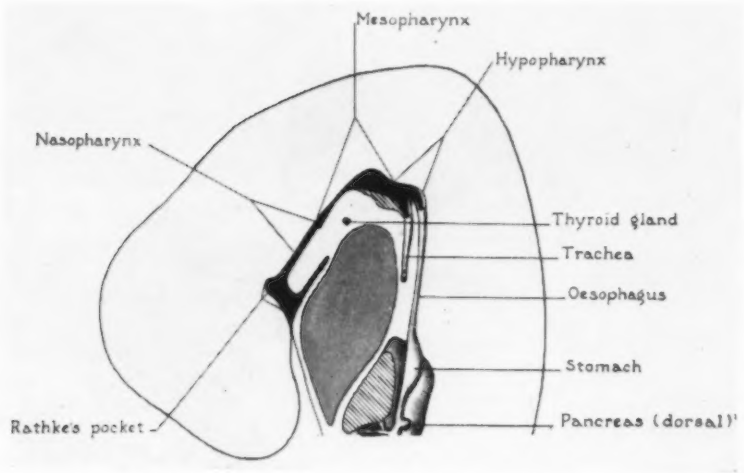



FIG. 4. Further differentiation in the mesopharynx.

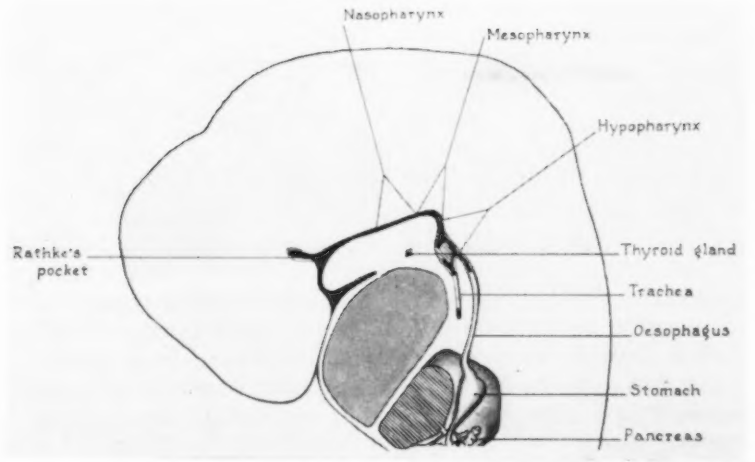



FIG. 5. Further differentiation in the mesopharynx.

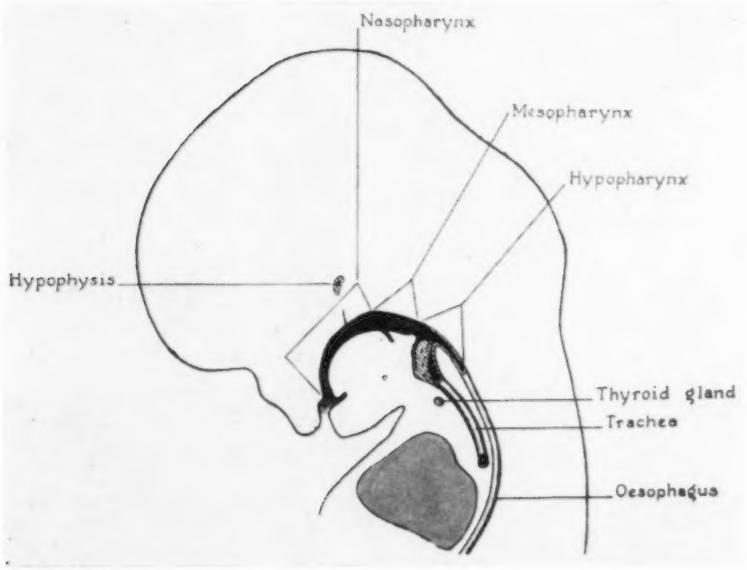



FIG. 6. Further differentiation in the mesopharynx.

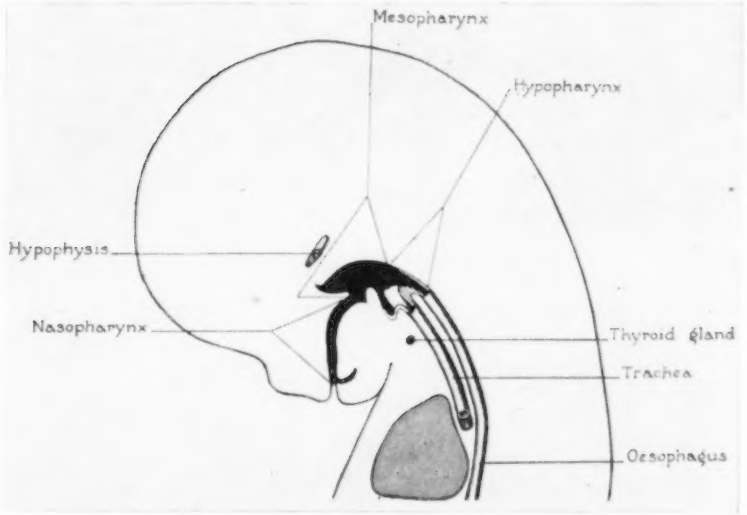



FIG. 7. Further differentiation in the mesopharynx.

ing their connection with the mouth. To carry this formation on, let us look at figures 3, 4, 5, 6, 7. Study of the youngest human embryos made by Professor Otto Grasser of Prague many years ago reveals that the analogue of the first pharyngeal pouch is apparent with the formation of the pharyngeal membrane. Thus we can see that we have the mesopharynx coming from mesoderm, entoderm, and ectoderm and assured of a rich vascular and nerve supply. Because of the almost simultaneous formation of the related parts, the five pharyngeal

FIG. 8. Submucous veins of the pharynx. (Taken from Batson's "Veins of the Pharynx".)

FIG. 9. Submucous veins of the pharynx, original, transparent preparation. The arrow indicates the locus of injection; E, the epiglottis turned back to show the vallecula, and P, the posterior plexus. (Also taken from Batson's "Veins of the Pharynx".)

pouches, the palate, the thymus, the thyroid gland, the thyroglossal duct, the liver and the rest of the digestive tract, it is surprising that we do not have more congenital anomalies in this area.

FIG. 10. More or less diagrammatic illustration to show the mucous membrane of the mesopharynx.

### VASCULAR AND LYMPHATIC SUPPLY

The blood vessels of the oropharynx form a virtual pool because this area seems to be at the "crossroads" of the descent from the nasopharynx to the hypopharynx and vice versa. The dense plexuses of veins have been ably demonstrated by Dr. Oscar V. Batson (figs. 8 and 9). The arterial supply is conceivably just as rich. The lymphatic plexus probably is more abundant because of the presence of so many large, obvious lymph structures, both in the immediate vicinity and scattered over the mucous membrane of the oropharynx. (See fig. 10.) The blood supply of the pharynx is of course derived from the external carotid through its pharyngeal branches

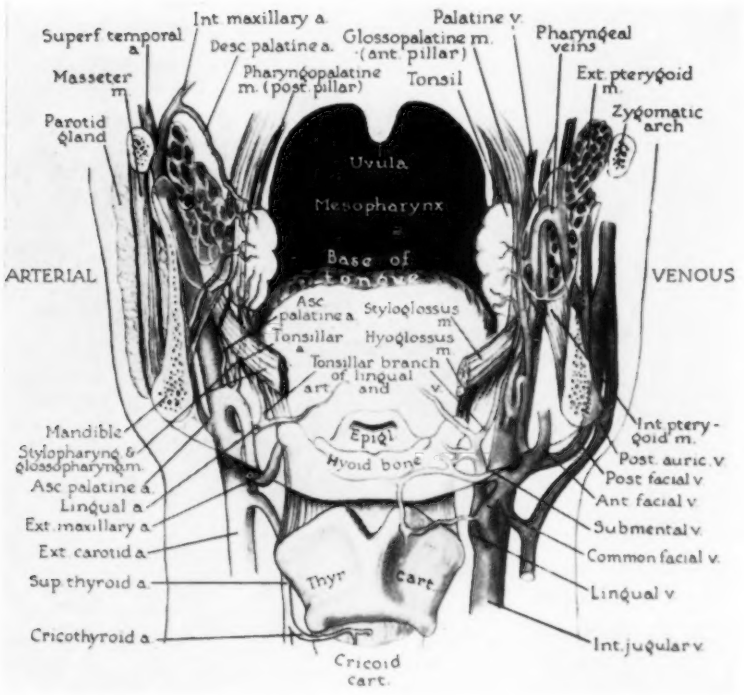



FIG. 11. Diagrammatic sketch of arterial and venous supply of the oropharynx.

(fig. 11). The veins of the pharyngeal plexus empty into the internal jugular by way of the pharyngeal vein, and into the external jugular by way of the posterior palatine to the pterygoid plexus. The rich plexus of lymphoid tissue is arranged anatomically to form a pharyngeal lymphoid ring consisting of the pharyngeal, tubal, faucial, and lingual tonsils as well as small masses through the pharynx. The pharyngeal mucosa together with lymphoid accumulations constitute the inner portion of Waldeyer's ring (fig. 12). The rich lymphatic plexus in the pharynx sends efferent vessels in two directions. Those which come from the tonsils and palate drain into the deep cervical nodes. The other group of lymphatics, including those from the nose, drain into the retropharyngeal nodes. These cervical lymph nodes represent the external loop of Waldeyer's ring. With this very rich vascular and lymphatic supply it is not surprising that Dr. Batson's injections revealed virtual "lakes" of veins, and we can logically assume that injections of the arteries or lymphatics, if possible, would yield similar results. Moreover, after considering the vascular supply of the mesopharynx we can see that the close proximity of the large vessels of the neck accounts for the very high temperature and chills which accompany infections in this area.

### NERVE SUPPLY

The nerve supply of the pharynx is likewise very rich. It is derived from branches of the glossopharyngeal and vagus, and fibers of the bulbar roots of the spinal accessary nerve, all of which form a sensory pharyngeal plexus in the mucous and submucous layers of the pharynx. A few fibers of the pala-

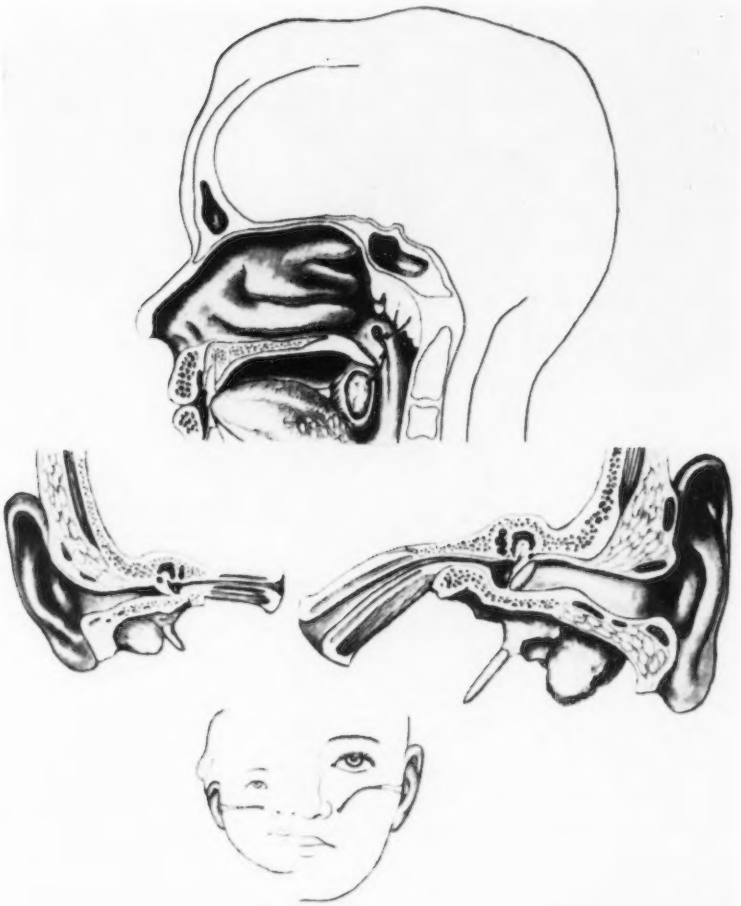


FIG. 12. Waldeyer's ring and diagrams to show easy path of infection into the middle ear (copied from ad sent out by Doho Co.)

tine nerve enter the sphenopalatine ganglion and are received by the maxillary branch of the trigeminal nerve. Some fibers pass through the lingual nerve to the mandibular branch of the trigeminal nerve. Sympathetic fibers in this dense network lose their anatomical identity in the mucous membrane. Motor innervation for the muscles of the pharynx, except for the tensor muscle of the palate which receives motor fibers from the otic ganglion from the motor root of the trigeminal, are controlled by fibers of the vagus, an accessory complex of the pharyngeal plexus (figs. 13 and 14).

HISTOLOGY OF MUCOUS MEMBRANE

The mucous membrane of the oropharynx is perforated by tubes of racemose, serous, and mucous glands, located in the stratum proprium or in the submucosa. A basement membrane lies between the epithelial and submucous layers. In the lower portion there is a nonciliated, stratified, squamous epithelium with no basement membrane but with a fine layer of elastic fibers instead. Many lymphocytes lie in a fibrous network which contains reticular tissue scattered throughout the entire mesopharynx (figs. 15 and 16).

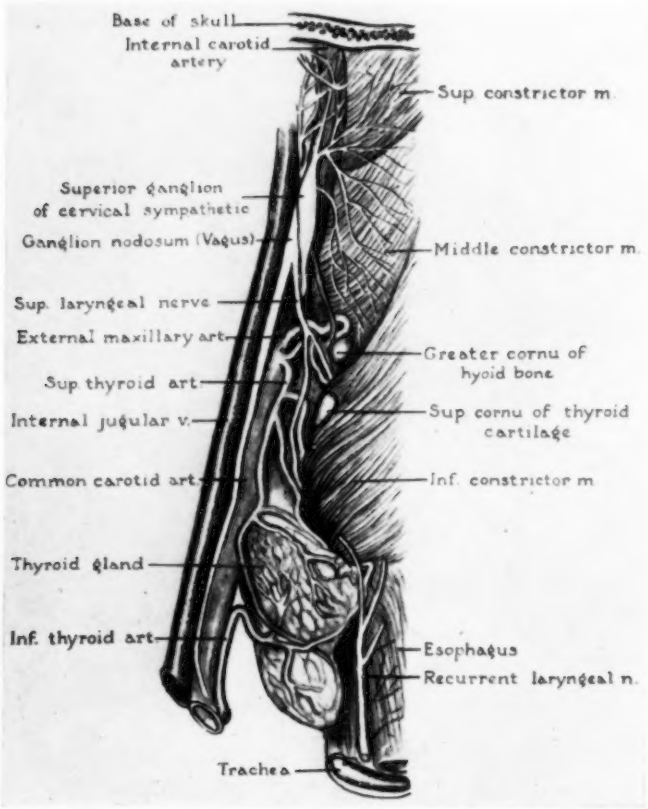



FIG. 13. Taken from Barnhill—illustrates the rich sensory sympathetic supply.

## FUNCTIONS

The chief functions of the oropharynx are respiration, swallowing, voice resonance, and articulation. Because of the presence of a few scattered taste buds, there is a minor gustatory function.

## GROSS ANATOMY

The mesopharynx or pars oralis is that portion, or clinical division, of the pharynx that has both alimentary and respiratory function. This portion of the pharynx opens into the oral cavity at the inferior border of the soft palate. The oropharynx or mesopharynx can be located at that portion of the pharynx below the soft palate and lying behind the mouth and going as far downward as the level of the hyoid bone. The mesopharynx is bound superiorly by an imaginary line drawn between the level of the base of the uvula and through the mid-portion of the first cervical vertebra (fig. 17). Laterally it is bound by the anterior and posterior pillars or the muscles glossopalatinus and pharyngopalatinus respectively and the palatine tonsils, lower portion of fossa of Rosenmuller, the lateral pharyngeal pillars or the salpingopharyngeal muscles. Inferiorly it is bound by an imaginary line between the level of the inferior border of the second cervical vertebra and the base of the tongue. Posteriorly the mesopharynx extends from a point at the

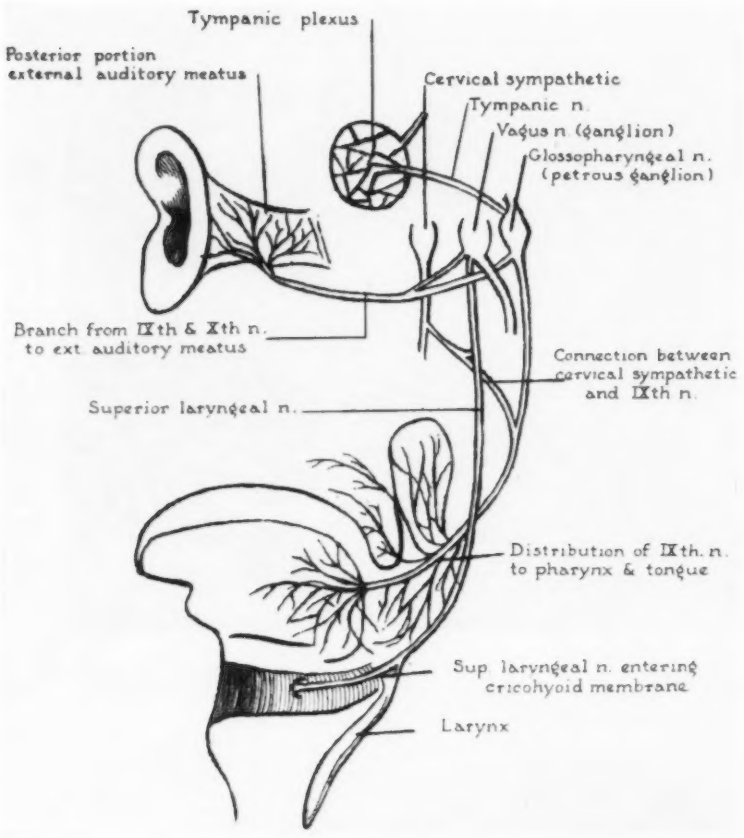

Tympanic plexus
Posterior portion external auditory meatus
Cervical sympathetic
Tympanic n.
Vagus n. (ganglion)
Glossopharyngeal n. (petrous ganglion)
Branch from IXth & Xth n. to ext. auditory meatus
Connection between cervical sympathetic and IXth n.
Superior laryngeal n.
Distribution of IXth. n. to pharynx & tongue
Sup. laryngeal n. entering cricohyoid membrane
Larynx

FIG. 14. Also taken from Barnhill—Diagram to show reason for secondary otolgia from cause in oropharynx, or to show direct connection between mesopharynx and the ear.

level of the junction of the upper and middle third of the first cervical vertebra to the level of the inferior border of the second cervical vertebra. This study aims to emphasize this portion of the pharynx. The anterior arch of the arcus glossopalatinus forms the lateral boundary of the anterior limits of the oropharynx. This faucial isthmus is formed anteriorly by the glossopalatinus muscles and posteriorly by the pharyngopalatinus (fig. 18) and the salpingopharyngeus muscles. A study of the following sketches reveals some of the anatomical details of the mesopharynx (figs. 17, 18, 19, 20, 21, 22, 23, 28, and 29).

If we should pass a probe through the mesopharynx at the level of the lower border of the first cervical vertebra from anterior to posterior, we would traverse the following structures named in order — mucous membrane, blood vessels, nerves and lymphatics, including lymph follicles, constrictor muscle, deep cervical fascia, retropharyngeal space, prevertebral fascia, longus colli muscles, periosteum, and bone of first cervical vertebra (figs. 22 and 23). Changes in these tissues are to be considered in this paper. A careful study of the foregoing figures will, I think, refresh our knowledge of the important anatomical details of the mesopharynx.

FIG. 16. High power to show lymph follicle taken from mucous membrane of mesopharynx or high power of characteristic section of fig. 15.

FIG. 15. Low power of lymph follicle taken from a boy 12 years of age whose mouth was crammed with lymphoid tissue.

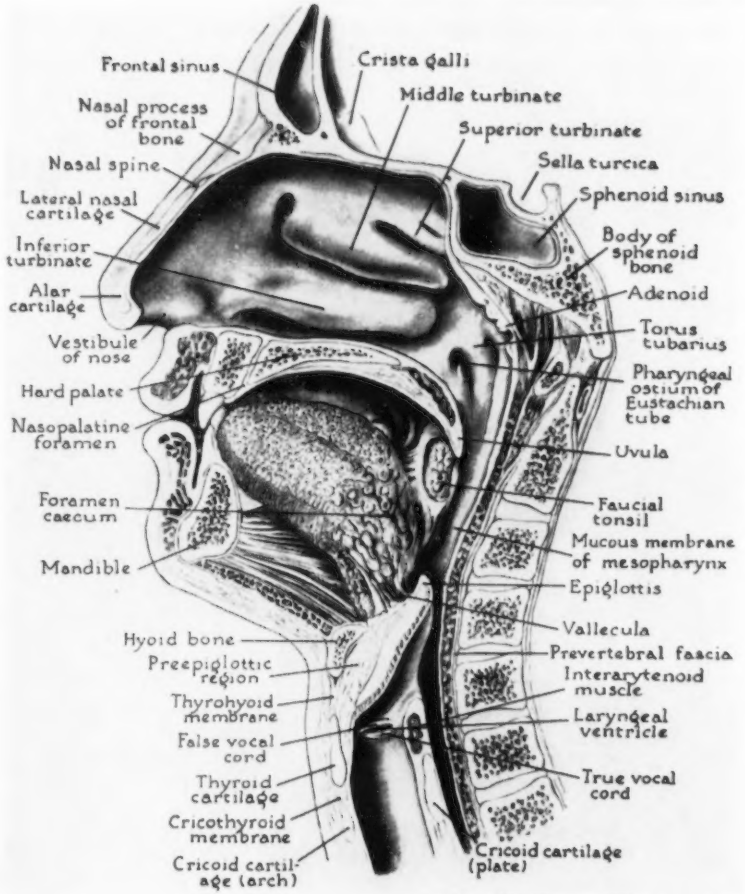



FIG. 17. Shows upper and lower boundaries of mesopharynx (see text of thesis).

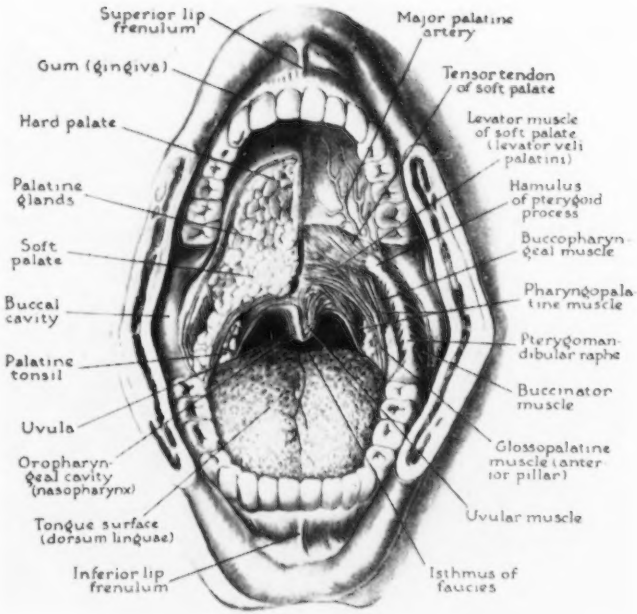



FIG. 18. Lateral boundary of mesopharynx.

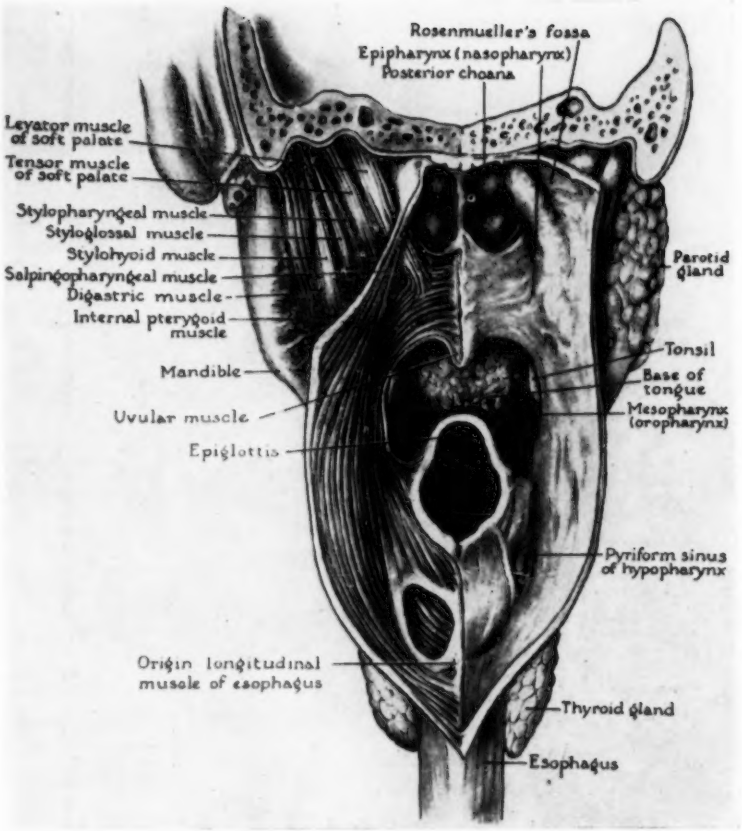



FIG. 19. Posterior view of muscles closely related to the mesopharynx.

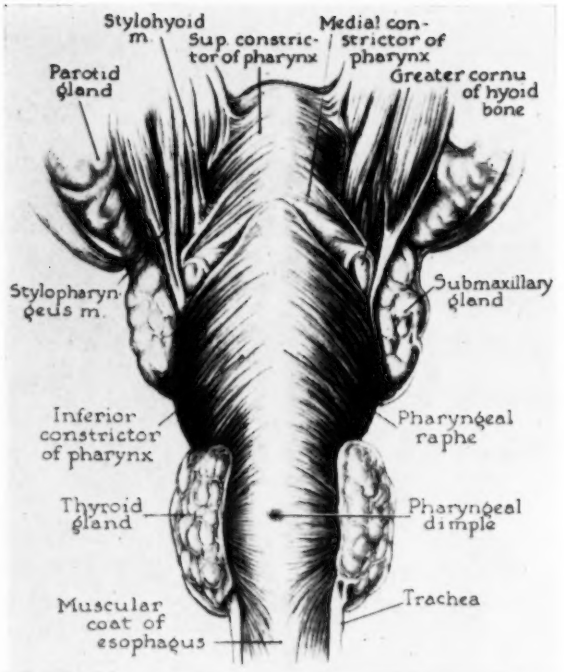



FIG. 20. Posterior view showing relation of the constrictor muscles to oropharynx.

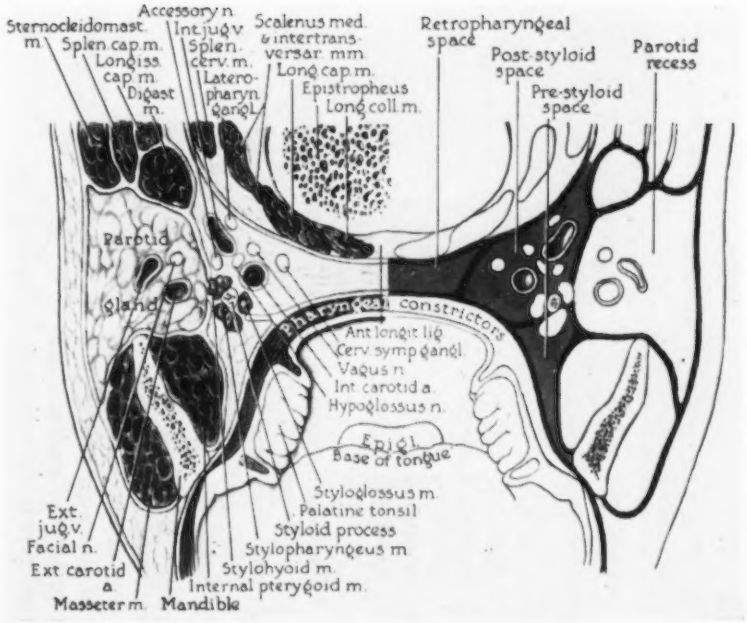



FIG. 21. Diagram to show relation of parapharyngeal spaces to mesopharynx.

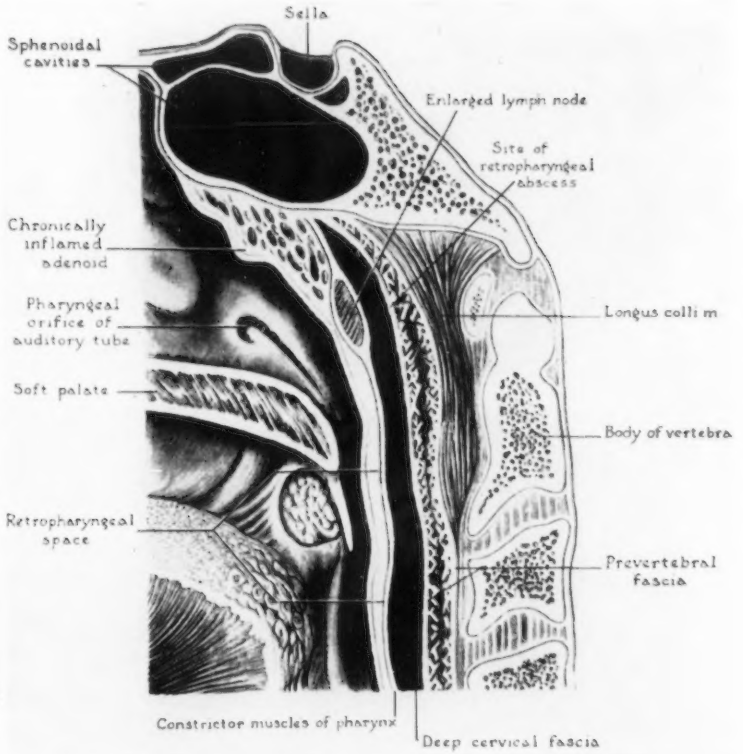



FIG. 22. Drawing taken from Barnhill to illustrate relation of mesopharynx to retropharyngeal space, deep cervical fascia and prevertebral fascia.

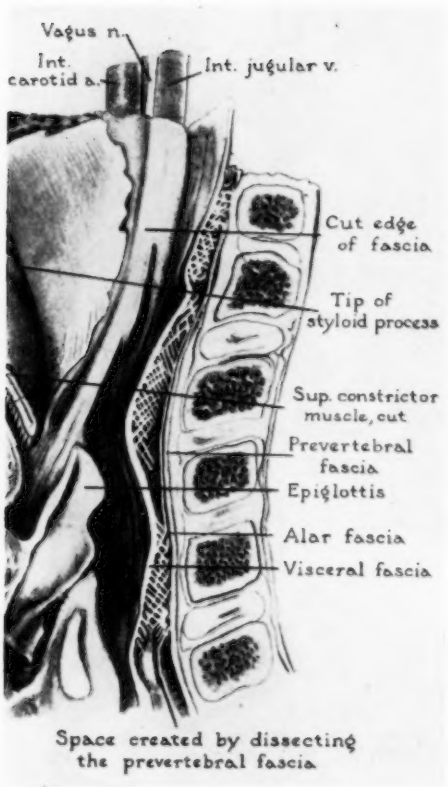



FIG. 23. Another dissection drawing taken from Barnhill to illustrate relation of the prevertebral fascia to the mesopharynx.

It is not surprising that the mesopharynx has such highly developed sensitive responses to stimuli (both sensory and motor) when we review the very rich nerve, lymphatic, venous and arterial supply of its abundant musculature.

TWO IMPORTANT ANOMALIES

1. *Prominent Mesopharynx:*

In many individuals one side of the posterior wall of the mesopharynx is much more prominent than the other. The area shown by figures 24 and 25 illustrates the appearance of this variation with no inflammation present. The visible portion of this area begins at a junction of the upper and middle third of the first cervical vertebra and extends

FIG. 24. Prominent mesopharynx without the presence of tonsils partly to obscure it, because of bony enlargement of anterior surface of half of a cervical vertebra.

FIG. 25. Same as Fig. 24 except in an individual with tonsils.

to the level of the inferior border of the second cervical vertebra. The enlargement ends abruptly at the mid-line, the soft parts making a gradual slope. The elevation of the anterior wall of the posterior pharynx is due to a congenital enlargement of the first and second vertebrae on their anterior surfaces or to a hyperplastic exostosis later in life. (See sagittal view in diagrams of this area, figs. 22 and 23, in order to visualize the position of this abnormality.) Consideration of this enlargement is helpful in both diagnosis and surgery. It is well to know that the unilateral enlargement on the anterior wall of the mesopharynx is due to the bony parts just posterior to or at different locations from anterior to posterior. Confronted in these cases with a peritonsillar abscess or a cervical lymphoid enlargement running a septic course, one can more accurately decide whether a complication is a retropharyngeal abscess, a cervical phlegmon or an extension of a peritonsillar abscess. Where a large cervical abscess is present, this knowledge is also of value in differentiating these three complications from a pharyngo-maxillary involvement. And, of course, knowing the position of these bony enlargements of one-half a cervical vertebra's anterior surface immediately posterior to the mesopharynx greatly facilitates surgery in this area.

2. *Abnormal Location of Arteries:*

The second anomaly is the presence of two very large curved bilateral pulsating vessels (internal carotids) apparently just under the mucous membrane located in the mid-portion of the usual position for the salpingopharyngeus muscles, these being the same pulsating vessels more frequently seen in the base of the tonsil fossae just after tonsillectomy. A drawing is shown here (fig. 26) to emphasize the importance of realizing this variation in opening a retropharyngeal or an extended peritonsillar abscess in this location. The guide in such cases is bilateral, symmetrical pulsations.

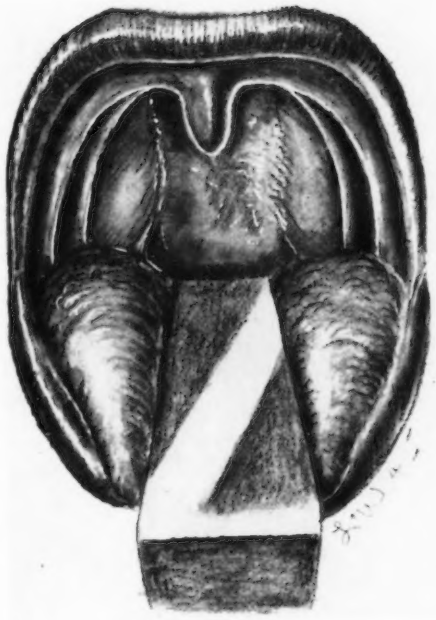


FIG. 26. Bilateral pulsating internal carotids deep only to mucous membrane.

DIAGNOSTIC ENTITIES

1. *Normal Mesopharynx:*

A. Elimination of Upper Respiratory Infection:

First, let us consider the appearance of the normal mucous membrane of the mesopharynx. It is very similar to the mucous membrane of the inner surface of the upper or lower lips. The rosy hue and texture appear almost identical although there is more transparency to the covering of the oropharynx and, of course, the action of the muscles just below the mucosa of the mesopharynx is absent from the lips. Until we have the results of infection and irritation the mesopharynx has a clear smooth glistening cover. In most cases when this normal mucous membrane is present we can assume that there is no chronic infection in the upper respiratory tract.

B. "Mesopharyngitis Symptomatica":

In every otolaryngologist's practice a very large group of time-consuming cases characteristically complain of very sore throats, difficulty in swallowing, sensation of foreign body, or even aphonia, when examination shows the ear, nose, throat and sinuses to be entirely normal. That is, no objective findings account for the symptoms. A careful history usually reveals the presence of domestic infelicity or menopausal or artificial menopausal findings. A psychiatrist can usually relieve the symptoms of the first group, and the proper estrogenic treatment will usually help the second. The predominant findings for diagnosis in these cases is a normal mesopharynx. We could logically call this type "mesopharyngitis symptomatica."

2. *Diseased Mesopharynx:*

A. Follicular Pharyngitis:

The so-called chronic follicular pharyngitis has been fully discussed in the literature and textbooks (fig. 10). This type of mesopharyngitis is more frequently seen in young individuals. Associated with excessive lymph formation in this area there is a preponderance of the same tissue in the lingual and pharyngeal tonsils and a marked hyperplasia of the adenoids. These follicles are true small lymph nodes of the mucous membrane. A microphotograph of one of these enlargements was taken from the mucous membrane of a fourteen-year-old boy's mesopharynx. His nasopharynx, mesopharynx, and hypopharynx were literally crammed with hypertrophied lymphoid tissue. His symptoms were those of recurring acute tonsillitis. Cultures taken from the throat were not significant. No pure culture or predominant organism could be found on five different occasions of acute attacks. These findings are essentially the same in all cases of this classification. As a rule these individuals are not relieved by removal of the tonsils and adenoids, but of course this procedure is necessary. In adults postnasal drip is frequently present. Careful study will usually reveal some past systemic pathology that has caused the usual lymphoid hypertrophy. Infection about the paranasal sinuses and teeth is the most common cause, but tuberculosis, syphilis, and malignancy should also be eliminated. When we consider the enormous vascular supply of the mesopharynx, as is so well shown by Dr. Batson's drawings, it is easy to see why we get these enlargements in such concentrated form, that is, at the same time assuming that there are corresponding pools of lymph channels in the mucous membrane of the mesopharynx. Note particularly the pool of vessels just below the uvula (figs. 8 and 9). I do not believe that we can help these individuals with local cauterization, chemical or actual, of these lymph follicles unless the cause underlying their formation is removed.

B. Ozena of the Mesopharynx:

When we see a parchment-dry, pale mesopharynx with crust-like exudative deposits superimposed we realize at once that we have a condition of the throat similar to the one of the nose which we term ozena. This is too familiar to need further description and is covered here to illustrate the fact that conditions fully described in the literature have characteristic signs appearing in the mesopharynx.

C. Large Lateral Pharyngeal Pillars:

One type of mesopharynx has very large lateral pharyngeal pillars (figs. 27, 28, 29). A typical case, adult, thirty-five years of age, apparently healthy except for her throat findings, came with temperature of 104 degrees, 20,000 total white cell count. The oropharynx showed marked edema and a raw beefy appearance. Enlargements two centi-

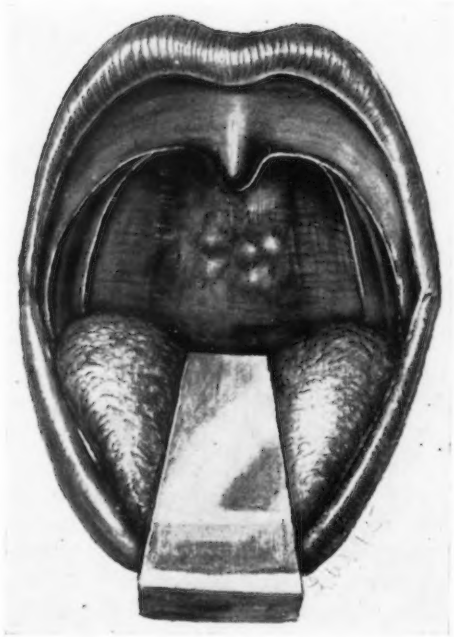


FIG. 27. Large lateral pharyngeal pillars.

meters in diameter were present on each side. They extended above the uvula and soft palate and below the base of the tongue. These enlargements took on an elongated, short pipe-like shape with the mucous membrane intact over the entire oropharynx. A greatly exaggerated, trough-like depression appeared in the midline when the patient swallowed or gagged. The mucous membrane was smooth with no lymph follicle-like enlargements. Pain was greatly increased with attempted muscular movements of the pharynx. The intense pain and board-like feeling is easy to understand when we keep in mind the manner in which the muscles of the pharynx slide by and on each other (figs. 18, 19, 20, 28, 29). Pain in the throat is also very intense in this area due to the rich sensory nerve supply. (See figs. 13 and 14 showing nerve supply.) Her tonsils had been removed and there was no congestion of the anterior pillars (glossopalatinus muscles). The vault of the nasopharynx revealed very little congestion and no abnormal secretions were present posteriorly in the middle or inferior meatuses. Transillumination and x-ray of the sinuses were normal. Her intranasal findings, except for marked congestion and redness, were normal. There was no abnormal secretion in either middle or inferior or superior meatus. She complained of no pain referable or suggestive of sinus involvement. There was no history of "postnasal drip." She did complain of similar sore throats every three to four months. These attacks usually occurred when fatigue was prominent and constipation present. Culture revealed pure pneumococci type III on the first occasion and during eleven similar attacks. Even after chemotherapy was available, whole blood transfusions and other energetic treatments were necessary before relief could be obtained from her more severe exacerbations. Complete relief with no recurrent attacks was obtained after a long regime of close attention to the gastrointestinal tract.

The important and significant finding here and in many other cases of this type is the recognition of the diagnostic lead for eliminating the cause of the illness. Significant and characteristic in all of these cases is the diagnostic suggestion of the presence of large lateral pharyngeal pillars. A constant vigil for the presence of large lateral pharyngeal pillars (figs. 27 and 29) is a material aid in early diagnosis and the instigation of proper treatment. Even in the quiescent period the large pharyngeal pillars are present. Therefore I present this type of throat with very large lateral pharyngeal pillars as a diagnostic entity for a fulminating infection just as the ophthalmologist would use bone-like, corpuscle-shaped pigment deposits in ring formation at the periphery of the fundus to diagnose retinitis pigmentosa.

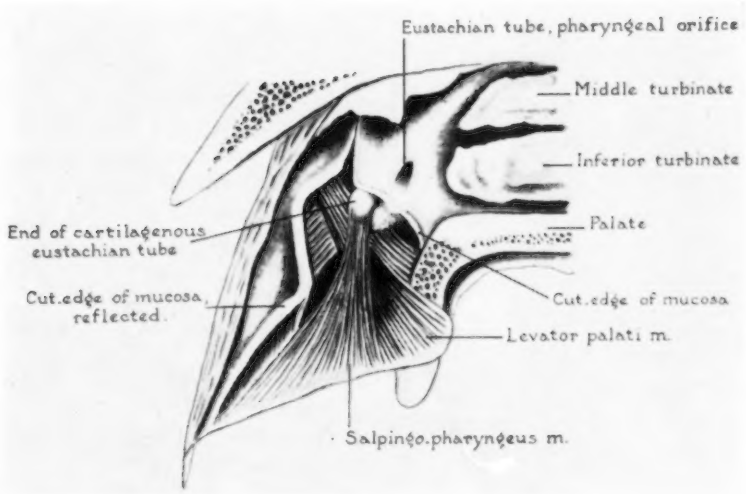



FIG. 28. Drawing from Barnhill showing position of salpingopharyngeus muscle.

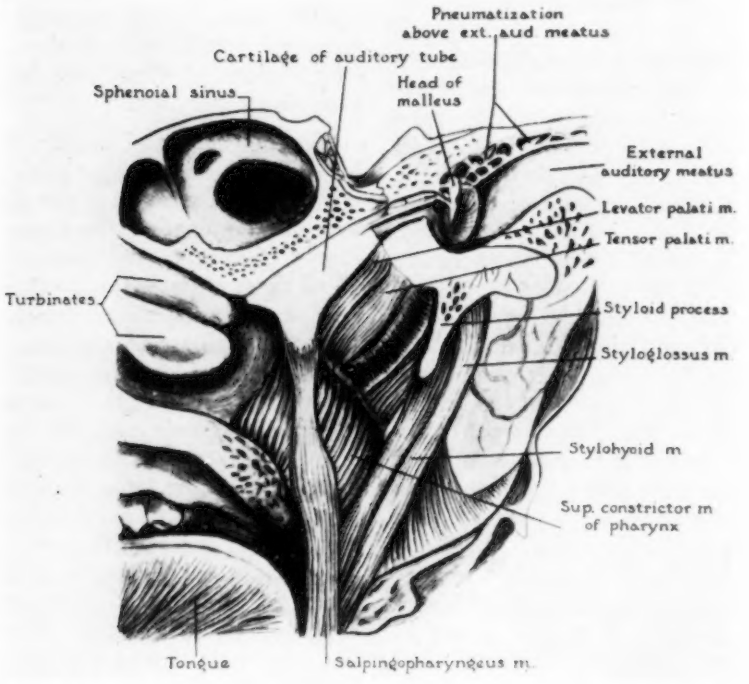



FIG. 29. Drawing from Barnhill showing position of salpingopharyngeus muscle—different view from that shown in fig. 28.

It would be presumptuous to say that all cases of the type shown in the sketch or alluded to could be diagnosed as acute pharyngitis of pneumococcal type III infection. We can presume, however, that in the presence of large lateral pharyngeal pillars the course of this disease will be very septic and will require extraordinary measures for alleviation. Another look at the sketch will give us a definite picture to keep in mind for purposes of diagnosis. This lady seemed to harbor pneumococci in her throat and with the slightest lowered resistance, the disease would recur. She is an expression teacher. Other similar cases have been lawyers, preachers, teachers, and auctioneers—all of whom use their voices incessantly. It is suggested that this excessive use of the voice has caused the hypertrophy of the lateral pharyngeal pillars (salpingopharyngeal) during the presence of the recurrent local infections of these parts. All of the muscles of the oropharynx are commonly thicker and larger, but this one is more prominent because of its location (figs. 27 and 29).

We know that very red anterior pillars of the pharynx indicate systemic infection and that very red acute pharyngitis without the large pharyngeal pillars indicates a mixed infection of the oropharynx that has not been persistently recurrent over a long period. Adding the type of case described with very large lateral pharyngeal pillars gives us a third diagnostic lead. We can not explain why certain areas at times have a predilection for becoming the host of certain infections, but this certainly seems to be so.

Although therapy is not the point of this discussion, a brief panoramic review of the treatment of sore throat would seem to be of interest and some diagnostic value. Seventy-five years ago my grandfather treated recurrent sore throats by the process of clearing the intestinal tract of stasis and infection. During my father's lifetime the importance of the elimination of foci of infection other than gastro-intestinal was stressed and the allergic theories began. More recently chemotherapy has made possible many wonderful recoveries, and the whole new field of vitamin deficiencies is being explored. In spite of the miracles wrought by chemotherapy, we have all seen recurrent infections become fast to known chemo-therapeutic agents. Therefore we should not be led into utter dependency upon the apparent panacea in spite of its value in acute infections. In special selected cases with excessive lymphoid tissue the use of radium is indicated. In all cases the known therapeutic procedures should be considered, and a combination of several may be needed for the proper alleviation of the malady.

D. "Mesopharyngitis Proliferans":

A large group of these cases complain of very severe sore throat, difficulty in swallowing, violent rapid toxemia and febrile reaction when a so-called lowered resistance or fatigue occurs. The infection may spread rapidly from the mesopharynx to become a manifest pneumonia or a complicating otitis media or pharyngomaxillary abscess. These individuals are suddenly sick and severe complications follow rapidly. The tonsils and adenoids have usually been removed. Often most of the paranasal sinuses have had surgical drainage and frequently there has been radical surgery in this area. Most of these people are in early middle life and are carrying on a very strenuous business or professional career. Frequently, loss of sleep, excessive stimulation, constipation, and poor dietary habits have added to the impairment of the normal physiological mechanism. They complain of severe throat illness at periodic intervals. They

learn to know what is to follow by the presence of a "peculiar type of feeling." In most cases they have chilly sensations soon followed by temperatures of 103 to 105 degrees, complicating lobar pneumonia with culture of streptococci of beta and alpha type. Next in frequency of complication is an influenzal type of illness in which the consulting physician can not determine definitely the causative organism. The acute condition is combatted with chemotherapy and, if necessary, whole blood transfusions. Often a blunt dissection of a pharyngomaxillary abscess will give rapid recovery. Recurrent attacks are eliminated by removing all possible foci of infection and restoring normal rest and dietary habits. Frequently it will take months to restore properly general health before completing the radical surgery necessary to remove the causative infection from a sphenoid or ethmoid group, frontal or maxillary sinus (fig. 30).

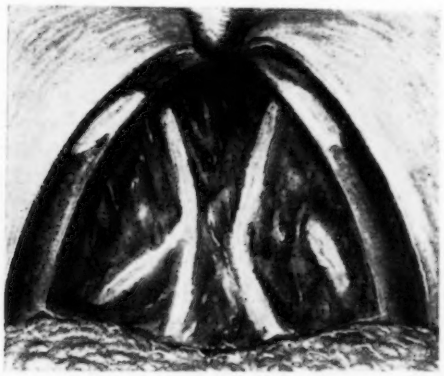


FIG. 30. Mesopharyngitis proliferans.

The mucosa of this type of mesopharynx has large white bands criss-crossing it. Where these slick dry white ribbon-like bands meet, there are enlarged white areas that have a gristle or tendon-like appearance. In between the white areas are dark red granulations of knob-like enlargements. All of the normal luster of the mucous membrane covering the mesopharynx is gone. The gag-reflex is usually greatly diminished. The general appearance during an acute attack is like the ulcerated surface of a granulating-appearing carcinoma. The white bands are due to fibroplasia resulting in scar tissue caused by many previous infections and to the effects of the use of cauterizing agents. Irrigations, appropriate powders, gargles, etc., are indicated in addition to the systemic treatment outlined. Cauterizing agents contribute nothing but trauma to the final picture, and should be avoided in spite of temporary symptomatic relief. The otolaryngologist could call this type of oropharynx a "mesopharyngitis proliferans" corresponding to the ophthalmologist's "retinitis proliferans." He would mean the presence of destroyed normal tissue due to previous infection and trauma. This predominance of scar tissue and red islands of mucosa presents a fixed picture that illustrates the purpose of this paper. Diagnosis is facilitated by having in mind various characteristic findings of the diseased mucosa of the oropharynx that represent the results of definite causative factors.

## SUMMARY

The anatomy and embryology of the mesopharynx have been abstracted. Two important anomalies have been described. The diagnostic value of large pharyngeal pillars has been indicated. Follicular pharyngitis has been reviewed. The possibility of the rapid elimination of chronic infection in the sinus and upper respiratory tract in the presence of normal mucosa of the mesopharynx has been shown. In the absence of other signs and symptoms certain of these cases have been grouped under the convenient term of "mesopharyngitis symptomatica." The term, "mesopharyngitis proliferans," has been suggested to cover certain other signs associated by me in a diagnostic group.

## CONCLUSION

If we can help the beginner to form definite concrete pictures of the mesopharynx for each of the conditions discussed and others as they occur, he will be able to diagnose many rather complicated syndromes with one careful look at the mesopharynx. It is hoped that the mesopharynx will receive more attention in the literature.

## REFERENCES


Barnhill, John Finch: Surgical anatomy of the head and neck, Baltimore, William Wood & Company, 1937, pp. 697, 698, 767, 768, 780.

Batson, Oscar V.: Veins of the pharynx, Arch. Otolaryng., 36:212-219 (Aug.) 1942.

Batson, Oscar V.: The anatomy of the head and neck as related to dental infections, Official Bull., Dental Soc. State of New York.

Bryan, Joseph Hammond: The history of laryngology and rhinology and the influence of America in the development of this specialty, Ann. M. Hist., 5:151-170 (March) 1933.

Burnam, Curtis F., Crowe, Samuel J., Fisher, Gilbert Eugene and Swanberg, Harold: A symposium on radium in benign nasopharyngeal pathology, Mississippi Va'ley M. J. & Radiologic Review, 67:109-119 (Oct.) 1945.

Costen, James B.: Acute pharyngeal infection: Management and factors involved, J. Missouri M. A., 29:525-529 (Nov.) 1932.

Dean, L. W.: The proper procedure for external drainage of retropharyngeal abscess secondary to caries of the vertebrae, Ann. Otol., Rhin. & Laryng., 28:566-569 (June) 1919.

Denison, W. C.: Milk injection for pharyngeal and laryngeal infections, Laryngoscope, 46: 643-644 (Aug.) 1936.

Dick, G. F. and Dick, G. H.: Scarlet fever, Edinburg M.J., 41:1-13 (Jan.) 1934.

Diphtheria: Its prevention and control, Supplement No. 156 to the Public Health Reports.

Edmunds, Charles W.: Circulatory collapse in in diphtheria, Am. J. Dis. Child., 54:1066-1079 (Nov.) 1937.

Emerson, Francis P.: Chronic infections of the upper respiratory tract and their relation to general disease, Tr. Am. Laryng. A., 45:119-131, 1923.

Frank, Ira: Retropharyngeal abscess, J.A.M.A., 77:517 (Aug.) 1921.

Getting, Vlado A.: Report on medical progress; some epidemiological considerations of diphtheria, New England J. Med., 223:717-721 (Oct.) 1940.

Gordon, John E.: Current epidemiological aspects of scarlet fever, New England J. Med., 221:1024-1029 (Dec.) 1939.

Grant, Samuel B., Mudd, Stuart and Goldman, Alfred: Experimental study on excitation of infections of the throat, J. Exper. Med., 32: 87-112 (July 1) 1920.

Grodinsky, Manuel: Retropharyngeal and lateral pharyngeal abscesses; an anatomic and clinical study, Ann. Surg., 110:177-199 (Aug.) 1939.

Havens, Fred Z.: Severe hemorrhage secondary to retropharyngeal abscess, Am. J. Dis. Child., 58:1256-1258 (Dec.) 1939.

Hochfilzer, John J.: Fatal asphixia in a case of retropharyngeal abscess, Minnesota Med., 19:461 (July) 1936.

Hume, E. E.: Francis Home, M.D., Scottish military surgeon who first discovered diphtheria, Mil. Surg., 91:218-228 (Aug.) 1942.

Keibel, Franz and Mall, Franklin P.: Manual of human embryology, Philadelphia and London, J. B. Lippincott Company, 1912, pp. 309, 311, 314, 316, 320, 321, 327.

King, John T.: On the occurrence of pharyngeal infections in exophthalmic goiter, Bull. Johns Hopkins Hosp., 62:593-609 (June) 1938.

Lees, H. D.: Scarlet fever, Lancet, 51:211-214 (March) 1931.

Levin, M. B.: Nose and throat infections in general medicine, Laryngoscope, 43:450-548 (July) 1933.

Levine, H. D., Hoerr, S. O. and Allanson, J. C.: Vesicular pharyngitis and stomatitis; an unusual epidemic of possible herpetic origin, J.A.M.A., 112:2020-2022 (May 20) 1939.

Lederer, Francis L.: Diseases of the ear, nose and throat, Ed. 4, Philadelphia, F. A. Davis Company, 1944.

Liang, P. K., Lei, C. H. and Liang, P. T.: Retropharyngeal abscess; secondary haemorrhage; ligature of common carotid; recovery, Brit. M. J., 1:967 (May) 1925.

Lillie, Harold I.: The clinical significance of compensatory granular pharyngitis, Arch. Otolaryng., 24:319-324 (Sept.) 1936.

Lukens, J. A.: Fatal type of hemorrhage following throat infections, Ohio State M. J., 25: 879-882 (Nov.) 1929.

Mason, H. H.: Preventive pediatrics, J. M. Soc. New Jersey, 24:473-476 (Aug.) 1927.

Nelson, R. F.: Parapharyngeal abscess with complications; case report, California & West. Med., 27:519-520, 1927.

Orton, Henry Boylan: Lateral transthyroid pharyngotomy, Ann. Otol., Rhin. & Laryng., 51:774-779 (Sept.) 1942.

Pomales, Americo: Bacteriologic study of normal throats, pathological throats and tonsils removed at operation in Porto Rico, Porto Rico

J. Pub. Health & Trop. Med., 5:196-210 (Dec.) 1929.

Porter, Charles T.: Unrecognized complications secondary to peritonsillar and lateral pharyngeal abscess; with case reports, Arch. Otolaryng., 26:127-131 (Aug.) 1937.

Rhodes, George K.: Retropharyngeal infections with associated hemorrhages; a rationale of procedure for a standardized operation, Surg., Gynec. & Obst., 70:834-840 (April) 1940.

Richards, Lyman: The surgical aspects of otitis media, peritonsillar abscess and retropharyngeal abscess, Am. J. Surg., 46:505-518 (Dec.) 1939.

Robison, J. Mathews: Lymphangitis of the retropharyngeal lymphatic system, Reprinted with additions from Arch. Otolaryng., 40:385-395 (Nov.) 1944.

Robison, J. Mathews: Lymphangitis of the mucosa of the paranasal sinuses, Reprinted from Texas State J. Med., 40:193-199 (July) 1944.

Roberts, Edward Russell: Fatal idiopathic pharyngeal hemorrhage. Case report, Laryngoscope, 40:807-808 (Nov.) 1930.

Salinger, Samuel and Pearlman, S. J.: Hemorrhage from pharyngeal and peritonsillar abscesses; report of a case, resume of the literature and discussion of ligation of the carotid artery, J.A.M.A., 18:464-509 (Oct.) 1938.

Savage, C. P.: Scarlet fever and its complications, J.M.A. Georgia, 23:336-341 (Sept.) 1934.

Schwentker, Francis F. and Noel, W. W.: The circulatory failure of diphtheria, Bull. Johns Hopkins Hosp., 46:359-364 (June) 1930.

Schmiegelow, E.: Acute infectious phlegmon of pharynx with sepsis, Hospitalstid, Copenhagen, 68:11-22 (Jan.) 1925. Abs., J.A.M.A., 84: 864, 1925.

Scholes, F. V.: Streptococcal pharyngitis and its complications, M. J. Australia, 2:1065-1077 (Dec. 24) 1938.

Shackleton, P.: Two cases of pharyngeal hemorrhage, Brit. M. J., 2:1167, (Dec.) 1933.

Shallow, T. A.: Pharyngeal diverticulum; one stage resection, J. Internat. Coll. Surgeons, 2: 48-52 (Jan. & April) 1939.

Shambaugh, George E.: The recognition of chronically infected faucial tonsils, Illinois M. J., 1941.

Snow, W. J.: Diagnostic signs in acute suppurative frontal sinusitis, Medical Record and Annals, 38:797-798 (May) 1944.

Stevenson, W.: Acute infections of the pharynx and larynx, Illinois M. J., 72:153-158 (Aug.) 1937.

Teed, R. Wallace: Bilateral encapsulated peritonsillar abscess in a child; review of the literature and report of a case, Arch. Otolaryng., 22:90-95 (July) 1935.

Todd, Thomas Wingate and Fowler, Robert Henry: The muscular relations of the tonsil, Am. J. Anat., 40:355-371 (Nov. 15) 1927.

Touroff, Arthur S. W.: Intraperitoneal streptococcic abscess complicating acute pharyngitis; acute ileus, operation, recovery, Ann. Surg., 107:469-472 (March) 1938.

Veeder, Borden S.: The systemic manifestations of upper respiratory infections, M. Clin. North America, 11:415-423 (Sept.) 1927.

Veldee, M. V.: Scarlet fever; its prevention and control, Reprint No. 1202, Public Health Reports, 1937.

Victor, K. N.: Diagnosis; complications of common infectious diseases in the throat and carriers of these infections as related to the nose and throat, Kentucky M. J., 32:544-546 (Oct.) 1934.

Wasson, W. W.: Infection of the respiratory tract in the infant and child, Texas State M. J., 22:623-626 (Feb.) 1927.

Watson-Williams, E.: Parapharyngeal abscess, the quinsy that isn't, Lancet, 1:792-794 (April 12) 1930.

Wells, J. Ralph: The origin of immunity to diphtheria in central and polar Eskimos. A study of the throat flora, Am. J. Hyg., 18: 629-655 (Nov.) 1933.

Wesselhoeft, Conrad: The treatment of diphtheria and scarlet fever, M. Clin. North America, (Sept.) 1941, pp. 1273-1285.

Williams, Horace James: Otitis media in scarlet fever. A report of the aural complications occurring in the scarlet fever patients at the Philadelphia Hospital for Contagious Diseases during the years 1922 to 1929, inclusive, Tr. Am. Laryng., Rhin., & Otol. Soc., 247-255 1932.

Willett, Thomas: A clinical review of an epidemic of contagious nasopharyngitis, Wisconsin M. J., 27:360-362 (Aug.) 1928.

Wilson, T. G.: Pharyngeal hemorrhage—revealed and concealed, Irish J. M., 307-308 (July) 1934.

Woodson, W. B.: Benign mixed tumor of the pharyngomaxillary space and related area, report of case, Texas State J. Med., 37:785-788 (April) 1942.

## DISCUSSION

ARTHUR W. PROETZ, M.D., St. Louis, Mo.: I shall attempt to discuss Dr. Woodson's paper in two sections as it was presented.

The first part devoted to anatomy, physiology and pathology, unfortunately was read pretty quickly and did not do justice to the great amount of work and careful reading that Dr.

Woodson devoted to it. Since these are hardly controversial subjects they do not lend themselves to much discussion. I should like to add just two things: first I should like to have more emphasis on aberrations of the internal carotid artery as they are sometimes seen on the face of the pharynx. Every year I call the attention of my students to a classical article on that subject by J. Parson Shaffer, published in the Journal of the American Medical Association, volume 44, page 14, 1921, which is worth anybody's time to look up and read.

The other is the importance which should be given to the differentiation between the retropharyngeal space and the prevertebral space. Most dissections are made in the mid-line, as they were in Dr. Woodson's picture and the prevertebral space does not appear. Off to one side, however, the spaces behind the pharynx are two. There is a thin, tough, aponeurosis between them. Offhand this would seem to be of little importance except for the fact that the retropharyngeal space extends laterally only to the carotid sheath and stops there. However if infection breaks through the thin membrane into the prevertebral space, then it is not limited laterally and can spread to the sides of the bodies of the vertebrae to the cranial nerves and cause pain.

So much for the anatomical considerations.

When it comes to the outlining of a system of diagnosis based upon minor changes in the mesopharynx, such as the ophthalmologist finds in the retina, I am afraid I cannot go along with Dr. Woodson,—at least I cannot go all the way. When one tries to elaborate such a system all at once there is always the temptation to make things fit more or less into a pattern which rarely exists in the human being, anatomy being what it is. For example, Dr. Woodson says, "Even in the quiescent period the large pharyngeal pillars are present." Therefore the author presents this type of throat with very large lateral pharyngeal pillars as a diagnostic entity for a fulminating infection just as the ophthalmologist would use bone-like corpuscle-shaped pigment deposits in ring formation at the periphery of the fundus to diagnose retinitis pigmentosa. I would not agree that the large lateral bands indicate a fulminating infection. I would say, rather, they indicate a latent infection, and, as in the case of any easily infected lymph tissue, are the point of low resistance.

To go further, "We can presume, however, that in the presence of large lateral pharyngeal pillars the course of this disease will be very septic and will require extraordinary measures for alleviation." I do not believe that.

Further, "We know that very red anterior pillars of the pharynx indicate systemic infection." I cannot agree to that, either. If I am not mistaken, it was Jackson who showed us some time ago that the sharply delineated band of congestion on the edge of the anterior pillars suggested, if it didn't indicate, low grade chronic infection in the tonsil.

I don't want to belabor these points but there are many others of similar nature.

I believe it is dangerous to put into print at one shot any complete system of diagnosis, depending upon minor changes in an area like the pharynx, which is subjected to so many different kinds of irritations and injuries that it may show almost anything. I don't want to be too critical, but if we begin to think in such terms, it will lead us into error. I suggest that you read Dr. Woodson's observations very critically and form your own conclusions.

W. BURBANK WOODSON, M.D., Temple, Texas: I wish to thank Dr. Proetz for his discussion.

These remarks are greatly appreciated, because anything that is controversial is worth while. My suggestions were, as indicated, intended as diagnostic leads and not as pathognomonic findings.

# A STUDY OF THE ANATOMICAL VARIATIONS IN THE ATTACHMENT OF THE OBLIQUE MUSCLES OF THE EYEBALL

WALTER H. FINK, M.D.
MINNEAPOLIS, MINN.

THE purpose of this investigation is to acquire a more complete conception of the variations found in the oblique bulbar (extraocular) muscles, with special emphasis on the clinical aspects.

The study was prompted by the desire to find an explanation for some of the unusual clinical findings associated with the ocular muscles, which seemed to point to the oblique muscles as a causative factor. As it is possible that anatomical variations may account for some of the abnormal action of these muscles, it seemed worthwhile to conduct an investigation to obtain more concrete information. Such information concerning the anatomical variation may explain some of the unusual and complicated clinical findings. These can thereby be diagnosed more accurately, instead of explaining the findings on the basis of paresis, secondary contractions, spasm, and other indefinite terms.

In addition to making a more accurate diagnosis, a more complete knowledge of the anatomy of the oblique muscles will permit the surgeon to approach an operative problem with better judgment. It is generally recognized that of all ocular muscle surgery, the surgery of the obliques is least understood and the least standardized.

An operation upon an oblique muscle is a rarity, in spite of the fact that it is frequently the site of trouble. Such a situation exists because most surgeons are not sufficiently familiar with the anatomy of this region. Surgical procedure involving the oblique muscles necessitates an intimate knowledge of their anatomy and a full appreciation of the possible variations.

It seems apparent, therefore, that an anatomical study of the oblique muscles is justified, because a better knowledge of this anatomy may aid in explaining some of the obscure findings and lead to more accurate diagnosis. In addition, a better surgical approach to these problems may be developed.

## METHOD OF PROCEDURE

Because of the wide scope of this problem, only one phase of it will be considered, namely, the insertion of these muscles on the bulb (globe). From the clinical standpoint, it appears that this is the phase which is most important. Variation in the anatomy of the origins of these muscles would not exert so much influence as the insertions. The study considers only the measurements which seem to be of special clinical value, namely, the relation of the oblique muscles to the anterior segment of the eye, and especially the relation of each to the adjacent rectus muscle.

The investigation comprises a study of one hundred specimens. The eyeballs were dissected free from the surrounding tissue and then distended with paraffin. Various measurements were taken with a millimeter rule and recorded in diagrams drawn to exact measurements, so that an accurate conception of the relation of the muscles could be obtained. The superior oblique was considered in relation to the superior rectus; the infe-

Material obtained through the courtesy of the Department of Anatomy, University of Minnesota.

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

rior oblique in relation to the lateral rectus and optic nerve.

Allowance should be made for the fact that the material was not fresh and the measurements were necessarily influenced to a certain degree.

THE SUPERIOR OBLIQUE MUSCLE

According to Whitnall,[1] the superior oblique, after it leaves the trochlea, "bends abruptly backwards, slightly down, and lateralwards at a 'trochlear angle' of about 54°, and the round tendon broadens out, pierces the fascia bulbi, and passes beneath the superior rectus, to be inserted into the back and lateral side of the eyeball. The first part of the muscle, from its origin to the trochlea, is referred to as the direct portion, and the tendinous part, between the trochlea and the globe, as the reflected portion. The tendon measures 20 mm. or ¾ inch in length from pulley to globe, and is enveloped by a prolongation of the fascia bulbi as far as the pulley.

"In most mammals, this reflected portion is also fleshy, and in lower vertebrates the whole muscle is represented by the reflected portion only, which arises from the medial angle of the orbit near the inferior oblique. The insertions of the tendons of the oblique muscles differ from these of the recti in being placed on the posterior instead of the anterior hemisphere of the eyeball; they also show much more variation in position.

"The line of insertion of the superior oblique tendon crosses the vertical meridian obliquely, and is curved with a backwardly directed convexity. The breadth as measured by Fuchs on 31 emmetropic globes was an average 10.7 mm., with extremes of from 7.5 to 12.7 mm., but was found narrower in myopic globes. In 20 of which it averaged 9.6 mm., the extremes were of from 6.8 to 14 mm. The distance from the corneal margin to where the line crosses the meridian is from 16 to 17.9 mm. (Fuchs), but Weiss in 5 adult emmetropic eyes found the anterior edge of the obliquely placed line to lie 13.85 mm. from the cornea and 7.56 mm. from the nerve; the anterior end also lies on the same meridian as the lateral end of the line of attachment of the superior rectus, but distant 4.6 mm. (with extremes of from 0.5 to 8 mm.) from it, though the two tendons are often connected by connective tissue strands. The line of the superior oblique is similarly connected with that of the inferior oblique, but is separated from it by 15 mm. at the anterior end, and 11.9 mm. at the posterior end. Fuchs described two types of insertion; one in which the line is broad with a strongly marked anterior concavity, which lies in an equatorial direction across the vertical meridian with the greater part on its lateral side, as more commonly found in emmetropic and hypermetropic eyes; the other, narrower and flattened, and lying entirely in the lateral quadrant, almost parallel to the vertical meridian, as found in myopic, though occasionally in normal globes.

"The trochlear or fourth nerve crosses over the muscle from the medial side to enter its substance at about 12 mm. from its origin; the blood supply is derived from the superior muscular branch of the ophthalmic artery."

*Analysis of the Data Pertaining to the Superior Oblique Muscle Which Was Obtained from the Examination of the Specimens*

From a clinical standpoint, certain data pertaining to the superior oblique muscle are of special importance. These data should not only include information concerning its insertion, but also its relationship to the superior rectus insertion. For more complete information, it is considered necessary to include measurements showing the relationship of

the superior rectus insertion to the limbus of the cornea.

The following are the data obtained pertaining to the superior oblique attachment, based upon an analysis of the 100 specimens.

First to be considered are the measurements of the superior rectus insertion and the relation of this insertion to the limbus of the cornea.

Measurement of the length of superior rectus insertions are as follows: of 100 specimens, the average length of insertion was 10.23 mm.; of 98 specimens, the average length of insertion was 10.16 mm., the measurements varying between 9 mm. and 12 mm.; of the remaining specimens, 1 measured 13 mm. and another 14 mm.

Measurements of the distance between the nasal end of the insertion of the superior rectus tendon and the limbus of the cornea are as follows: of 71 specimens, the average distance was 6.82 mm.; of 65 cases the average distance was 6.98 mm., the measurements varying between 6 mm. and 8 mm. Six cases measured 5 mm.

Measurements of the distance between the temporal end of the insertion of the superior rectus tendon and the limbus of the cornea showed that of 71 specimens the average distance was 9.23 mm. Of 70 specimens the average distance was 9.26 mm., the measurements varying between 8 mm. and 10 mm. One case measured 7 mm.

The relationship between the superior rectus and the superior oblique insertions is as follows:

Analysis of the measurements dealing with the length of insertion of the superior oblique tendons are as follows: of 100 specimens, the average length of insertion was 11.01 mm.; of 73 specimens the average length was 10.80 mm., the measurements varying between 9 mm. and 13 mm.; of the remaining cases, 3 were 7 mm., 10 were 8 mm., 6 were 14 mm., 5 were 15 mm., 2 were 17 mm., and 1 was 18 mm.

The measurements of the distance between the temporal ends of the insertions of the superior oblique and the superior rectus showed that in 100 specimens the average distance was 4.57 mm. Of 90 specimens the average distance was 4.47 mm., the measurements varying between 3 mm. and 6 mm. Of the remaining specimens, 4 were 2 mm., 3 were 7 mm., and 3 were 8 mm.

The measurement of the distance between the nasal ends of the two insertions showed that in 100 specimens the average distance was 13.33 mm. Of 83 specimens the average was 13.59 mm., the measurements varying between 12 mm. and 15 mm. Of the remaining cases, 8 were 10 mm., 4 were 11 mm., 4 were 16 mm., and 1 was 17 mm.

The comparison between the outer limits of these insertions is as follows: (The reading indicates the number of millimeters the temporal end of the insertion of the superior oblique lies medial to (+) or lateral to (-) the temporal end of the insertion of the superior rectus.) Of 100 specimens the average reading was -.72 mm.; of 90 cases the average was -.59 mm., the measurements varying between -2 mm. and +2 mm.; of the remaining specimens, 1 was -4 mm., 7 were -3 mm., and 2 were +3 mm.

The comparison between the inner limits of these insertions is as follows: (The reading indicates the number of millimeters the nasal end of the insertion of the superior oblique lies lateral to (-) or medial to (+) the nasal end of the superior rectus.) Of 100 specimens the average reading was +.02 mm.; of 85 cases the average was +.329 mm., the measurements varying between -3 mm. and +3 mm.; of the remaining specimens, 5 were -4 mm., 3 were +4 mm., 2 were -5 mm., 2 were -6 mm., and 1 was -8 mm.

In summarizing the above data, it is evident that in the relationship of the superior oblique insertion to the superior rectus insertion, certain data are fairly constant, whereas other are more variable. The data may be divided, therefore, into two groups: first, the group showing average measurements which may be designated as normal limits; and, second, the group below or above the average measurements which may be designated as the abnormal.

In the first group, which may be considered average measurements, a fairly high percentage of consistent measurements was found. The material is arranged according to the percentage in consistency of measurements. (Table I.)

It is therefore evident that the relationships are fairly consistent, with the exception of the last measurement.

The following diagram illustrates the relationships as based upon the average measurements:

Average Measurements of Superior Oblique Insertion

Measurements in millimeters

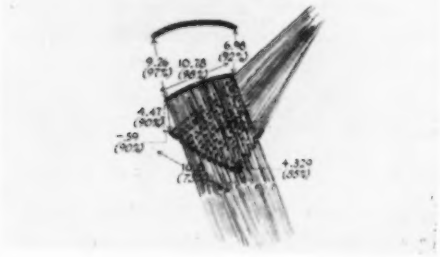



FIG. 1

In the second group the measurements may be considered abnormal. They are so designated because they are either greater or less than what may be considered average measurements.

The measurements, which are arranged in the order of the degree of variation, are presented in table II.

In analyzing the above data, it seems evident that the variability in measurements is dependent chiefly upon two factors, namely, the variable length of the superior oblique insertion, and the variable angle of this insertion.

The following diagram is constructed to three times the scale, and consists of 15 of the more pronounced variations superimposed.

Variable Position of Superior Oblique Insertion

FIG. 2

Although wide variations existed in the insertion of the superior oblique and its relationship to the superior rectus, no true anomalies of the superior oblique tendon were encountered in this study. Whitnall,[1] however, mentions one. "The superior oblique may be closely accompanied by an offshoot from the levator palpebrae superioris, sometimes called the 'comes oblique superioris.' In the preparation shown, there were present two long muscle bundles, arising in common with the levator and ending, the one upon the fascia bulbi between the superior oblique and the globe, the other on the orbital margin beneath the pulley; the nerve supply came from the fourth nerve; the superior oblique was broader than usual. Ledouble has found supernumerary fasciculi accompanying the reflected tendon; and has further recorded a case where the direct or normal fleshy part of this muscle was absent, and the reflected or usually tendinous part of this muscle was from the site of the pulley, recalling the type normally found in nonmammalian vertebrates."

TABLE I

| | Average measurements of specimens considered within normal limits | Percentage of specimens within normal limits | Designated limits of variation in measurement |
|---|---|---|---|
| A. Length of attachment of the superior rectus tendon | 10.16 mm. | 98% | 9 - 12 mm. |
| B. Distance between the temporal end of the insertion of the superior rectus and the limbus of the cornea | 9.26 mm. | 97% | 8 - 10 mm. |
| C. Distance between the nasal end of the insertion of the superior rectus tendon and the limbus of the cornea | 6.98 mm. | 92% | 6 - 8 mm. |
| D. Distance between the temporal ends of the two insertions, the superior rectus and superior oblique tendons | 4.47 mm. | 90% | 3 - 6 mm. |
| E. Comparison between the outer limits of the two insertions | -.59 mm. | 90% | -2 - +2 mm. |
| F. Comparison between the inner limits of the two insertions | +.329 mm. | 85% | -3 - +3 mm. |
| G. Distance between the nasal ends of the two insertions | 13.59 mm. | 83% | 12 - 15 mm. |
| H. Length of the attachment of the superior oblique tendon | 10.8 mm. | 73% | 9 - 13 mm. |

TABLE II

| | Percentage of abnormal measurements | Width of variation in measurement |
|---|---|---|
| A. Length of the attachment of the superior oblique tendon | 27% | 7 - 18 mm. |
| B. Distance between the nasal ends of the two insertions of the superior oblique and superior rectus | 17% | 10 - 17 mm. |
| C. Inner limits of the two insertions of the superior oblique and superior rectus tendons, showing the number of mm. the nasal end of the superior oblique tendon lies lateral to (+) or medial to (-) the nasal end of the superior rectus | 15% | -8 - +4 mm. |
| D. Comparison of the outer limits of the two insertions showing the number of mm. the temporal end of the superior oblique lies medial to (+) or lateral to (-) the temporal end of the superior rectus tendon | 10% | +3 - -4 mm. |
| E. Distance between the temporal ends of the two insertions | 10% | 2 - 8 mm. |
| F. Distance between the nasal end of the insertion of the superior rectus and the limbus of the cornea | 8% | 5 mm. |
| G. Distance between the temporal ends of the insertion of the superior rectus tendon and the limbus of the cornea | 3% | 13 - 14 mm. |
| H. Length of attachment of the superior rectus tendon | 2% | 7 mm. |

### THE INFERIOR OBLIQUE

Whitnall[1] states: "The inferior oblique muscle is the only one of the ocular muscles which does not arise from the apex of the orbit. The origin is from a small shallow depression on the orbital floor, just within the inferior orbital margin lateral, and usually, close to the opening of the nasolacrimal sac. The muscle passes laterally and backwards as a flat band beneath the inferior rectus, to which it is closely connected by the fusion of their fascial sheaths, is separated from the floor of the orbit by a compact mass of fat, and is inserted, like the superior oblique, on to the posterior hemisphere of the eyeball; its course intersects the axis of the orbit at an angle of 75°; it is fleshy in nearly its whole extent, the tendon being only 2.6 mm. long or indeed, often nonexistent, as that the muscle fibres pierce the sclera. The line of insertion lies obliquely in the lateral and inferior quadrant of the posterior hemisphere, farther back than that of the superior oblique,

with which it is not parallel but makes an angle of 25°, and nearer the optic nerve, from which it is distant about 5.2 mm. in normal but 7.1 mm. in myopic globes; it lies 9.5 mm. away from the insertion of the lateral rectus and almost on the same meridian, though at right angles to the line of the latter, and is only 2.2 mm. from a spot corresponding to the fovea of the retina. The distance from the corneal margin is from 17.3 mm. to 19.1 mm. (Fuchs). Salzmann (1912) notes that the line of its insertion often shows gross irregularities, angular serrations, or even dehiscences. The branch from the inferior division of the oculomotor nerve enters the muscle belly about the middle of its hinder border, and its blood supply is derived from the infra-orbital artery as well as the inferior muscular branch of the ophthalmic artery."

*Analysis of the Data Pertaining to the Inferior Oblique Muscle Which Was Obtained from the Examination of the Specimens*

The data pertaining to the inferior oblique are limited to the relation of the inferior oblique tendon to the two major adjacent structures, namely, the lateral rectus insertion and the optic nerve. These measurements would seem to be of both diagnostic and surgical importance.

The measurements of the length of insertion of the external rectus muscle are as follows: of 100 specimens, the average length of insertion was 9.98 mm.; of 90 specimens the average length was 10.78 mm., the measurements varying between 9 and 12 mm. Of the remaining cases, 8 were 8 mm., 1 was 13 mm., and 1 was 14 mm.

Measurements of the length of insertion of the inferior oblique muscle showed that of 100 specimens the average length was 9.67 mm. Of 93 cases the average was 9.58 mm., the measurements varying between 8 and 10 mm. Of the remaining cases 1 was 5 mm., 1 was 7 mm., 3 were 12 mm., and 2 were 14 mm.

The measurement of the distance of the anterior end of the insertion of the inferior oblique muscle from the lower end of the insertion of the lateral rectus tendon showed that in 100 specimens the average distance was 9.46 mm. Of 88 specimens the average was 9.6 mm., the measurements varying between 8 mm. and 11 mm. Of the remaining specimens, 1 was 6 mm., 7 were 7 mm., 3 were 12 mm., and 1 was 13 mm.

Measurements of the distance between the lower end of the external rectus insertions and the horizontal level of the insertion of the inferior oblique are as follows: of 100 specimens the average distance was 2.39 mm.; of 85 cases the average was 2.09 mm., the measurements varying between 0 and 3 mm.; of the remaining cases, 14 were 4 mm., and 1 was 5 mm.

The measurement of the distance between the posterior end of the inferior oblique insertion and the optic nerve showed that of 100 specimens the average distance was 4.26 mm. Of 94 specimens, the average was 4.15 mm., the measurements varying between 3 mm. and 6 mm. Of the remaining cases, 2 were 2 mm., 2 were 7 mm., 1 was 8 mm., and 1 was 10 mm.

Measurements of the horizontal level of the inferior oblique attachment as compared with the optic nerve level are as follows: (This indicates in millimeters the level of the oblique attachment above (+) or below (-) the lower level of the optic nerve.) Of 100 specimens the average reading was +.84 mm.; of 93 specimens the average was +.99 mm., the measurements varying between -1 mm. and +3 mm.; of the remaining specimens, 4 were -3 mm., 1 was -4 mm., 2 were +4 mm.

In summarizing the above data, it is evident that the insertion of the inferior oblique shows greater uniformity than that of the superior oblique. As in the case of the superior oblique, the data can be divided into two groups: first, the group showing average measurements which may be designated as within normal limits; and, second, the group below or above the average measurements which may be designated as the abnormal.

In the first group, which may be considered average measurements, a high percentage of consistent measurements was found.

The material is arranged according to the percentage of consistency of measurements. (Table III.)

In analyzing this group, it is evident that the various measurements show a high percentage of uniformity.

The following diagram illustrates the relationships as based upon the average measurements:

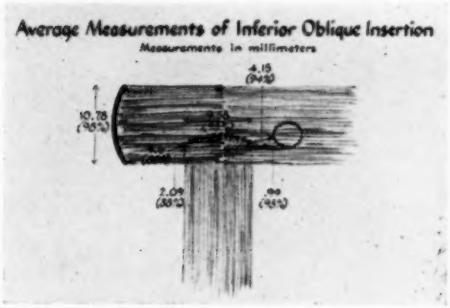



FIG. 3

In the second group, as in the superior oblique, the measurements may be considered abnormal, and are either greater or less than the average given above. These measurements showing these abnormalities are as follows:

The arrangement of data is based upon the percentage of variation. (Table IV.)

From the above, it is evident that the various relationships of the inferior oblique insertion are more consistent than those of the superior oblique insertion. The greatest variation occurs in the relationship between the lower end of the external rectus insertion and the anterior end of the inferior oblique insertion. In spite of this variation, the general average is not appreciably altered.

The following diagram is constructed to three times the scale and consists of 15 of the more pronounced variations superimposed.

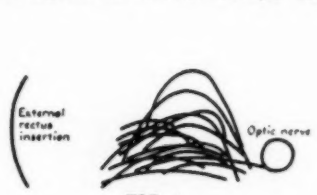



FIG. 4

Aside from the variation in the insertion of the tendon, one true anomaly was noted in the case of the inferior oblique, and it is important enough to be of clinical significance.

In specimen eight, the insertion of the muscle was unusually long and surrounded the optic nerve. It is clear how important this anomaly would be to a surgeon who wishes to cut the insertion of the muscle.

In addition to the above, it seems advisable to mention that on two occasions during operation, a fusion of the inferior oblique and the lateral rectus was noted. This anomaly apparently attributed to the unusual type of strabismus noted in the patient pre-operatively. Whitnall did not note any anomalies in the attachment of this muscle.

The following quotation from Whitnall[1] seems to be in agreement with the data recorded in this series of specimens examined.

TABLE III

| | Average measurement of specimens considered within normal limits | Percentage of specimens within normal limits | Designated limit of variation in measurement |
|---|---|---|---|
| A. Distance between the posterior end of the inferior oblique insertion and the optic nerve | 4.15 mm. | 94% | 3 - 6 mm. |
| B. Length of the insertion of the inferior oblique | 9.58 mm. | 93% | 8 - 10 mm. |
| C Horizontal level of the inferior oblique attachment as compared with the optic nerve level. (This indicates in mm. the level of the oblique attachment above (+) or below (-) the lower level of the optic nerve.) | +.99 mm. | 93% | -1 - +3 mm. |
| D. Length of the insertion of the external rectus muscle | 10.78 mm. | 90% | 9 - 12 mm. |
| E. Distance of the anterior end of insertion of the inferior oblique from the lower end of insertion of the lateral rectus tendon | 9.56 mm. | 87% | 8 - 11 mm. |
| F. Distance between the lower end of external rectus insertion and the horizontal level of the insertion of the inferior oblique | 2.09 mm. | 85% | 0 - 3 mm. |

TABLE IV

| | Percentage of abnormal measurements | Width of variation in measurements |
|---|---|---|
| A. The distance between the lower end of the external rectus insertion and the horizontal level of the insertion of the inferior oblique | 14% | 4 - 5 mm. |
| B. The distance of the anterior end of the insertion of the inferior oblique from the lower end of the insertion of the lateral rectus tendon | 12% | 6 - 13 mm. |
| C. Length of the insertion of the external rectus muscle | 10% | 8 - 14 mm. |
| D. The measurement of the horizontal level of the inferior oblique attachment as compared with the optic nerve level | 7% | +4 - -4 mm. |
| E. Distance between the posterior end of the inferior oblique insertion and the optic nerve | 6% | 2 - 10 mm. |

"It is probable to judge from the writer's individual experience in finding quite a number of gross abnormalities of the ocular muscles in a series of dissections, that such are by no means as excessively rare as would appear from the number recorded in the literature; dissecting-room conditions do not favor their identification, and in life some may be unrecognizable through compensatory action of the other muscles. They are explained by errors in development by cleavage from the common pre-muscular mesoblastic mass."

### CLINICAL APPLICATIONS OF THE DATA

From a diagnostic standpoint, the study emphasizes certain points of importance. It is not unusual in muscle diagnosis to encounter clinical findings which point to an oblique muscle as a causative factor. Frequently these findings are explained on the basis of paresis, secondary contractions, spasms, and other indefinite terms. An analysis of the anatomical variations, as seen in this series, suggests the possibility that some of the above-mentioned abnormal actions of the oblique may be explained on the basis of an abnormal anatomical condition. The study reveals that there are numerous variations in the anatomy of the oblique muscles, and serves to emphasize the possibility that many so-called paretic muscles may be normally innervated muscles acting in an abnormal manner because of the abnormal

insertions. It seems, therefore, likely that some of the abnormal action of the oblique muscles may be explained on this basis.

From a surgical standpoint, the work emphasizes several factors. In surgery of the superior rectus, it is important to remember that the nasal end of the insertion is normally 2 to 3 mm. closer to the limbus of the cornea than the temporal end. It is therefore important in an operation requiring cutting of the tendon, that in reattaching the tendon to the eyeball, it be placed in the same relationship to the cornea as was the original insertion.

In considering surgery of the superior oblique muscle, this work suggests that a favorable and feasible surgical approach could be made near its attachment. It is possible for the surgeon to orient himself by using the attachment of the superior rectus as a landmark. By retracting the superior rectus muscle laterally, the tendon of the superior oblique muscle can be isolated. Surgical procedures could be carried out from this point rather than at the trochlea. It seems that surgical manipulations at this point would produce the more effective results and the least degree of disturbance to the structures in this region.

It is also important to emphasize the comparatively intimate relation of the superior oblique tendon to the superior rectus tendon. As shown in various specimens, the tendon of the superior oblique tendon passes within a few mm. in some cases from the nasal edge of the superior rectus tendon. When working on the superior rectus, because of this relationship, the danger of severing or injuring the tendons of the superior oblique should be considered.

In the case of the inferior oblique, this study suggests the possibilities of a more satisfactory surgical management. Surgery should be done on the insertion of the inferior oblique rather than the origin because of its easy approach and the nature of anatomy. Fusion of the fascial sheaths of the inferior oblique to the inferior rectus at the "ligament of Lockwood," even though not intimate, may nullify to a variable degree the operative results if the operation is done at the bony origin.

The degree of effect would seem to be much more readily controlled when approached at the insertion of the inferior oblique. The recent trend to operate on the insertion rather than the bony origin seems to be justified.

The study also serves to show the possible intimate relation of the inferior oblique muscle to the optic nerve. In this connection, attention should be called to the close approximation of the posterior edge of the insertion of the muscle to the optic nerve. Several specimens showed the posterior insertion of the muscle to be 2 mm. from the optic nerve, and one specimen had fibers encircling the nerve. In the process of dissection, practically all specimens showed an extension of the sheaths which surround the muscle to the optic nerve sheaths. In addition, the posterior ciliary nerve and vessels which enter the globe in the region of the optic nerve are frequently found to be surrounded by the fascial extensions from the muscle. In doing a recession of this muscle, it would seem desirable to avoid these fascial extensions of the muscle sheath because of the structures mentioned. Attempt should therefore be made to free the muscle from the inner portion of the sheath. This intimate relation to the optic nerve sheaths, ciliary nerves, and vessels should be borne in mind when operating on the muscle. It is possible that some of our poor results in recessing the muscle may be due to an incomplete severing of the innermost attachments. One should also remember that the fovea is immediately beneath

the posterior tip of the insertion. The vortex vein also lies 10-12 mm. below the posterior portion of the attachment.

The inferior oblique line of attachment is usually muscular, although a very short tendon may be found. In recessions of this muscle, it seems logical to sever the muscle as close to its insertion as possible. This would eliminate the possibility of disturbing its innervation, which enters the muscle on its posterior edge just after it passes the inferior rectus muscle at the "ligament of Lockwood." Detaching the muscle near its insertion, permits a more accurate estimation of the degree of effect, because it permits the entire muscle to remain; a reattachment of a shorter muscle may result in an altered action.

The study suggests two points in the method of reattachment of the severed muscle. First, two sutures should be used to create a broad line of contact at the new point of insertion. One suture causes the muscle to attach at one limited area instead of a line of reattachment, which should be the width of the muscle. The second point is the method of determining the position of reattachment. Based on a study of the specimens, it seems feasible that the new line of attachment should begin at a point which is an equal distance from the limbus as was the original attachment. By so doing, the direction of muscle action is not altered. The anterior point of the new position may be determined by using a caliper which is set so that the two points of the caliper are the same distance apart as the distance between the limbus and the anterior tip of the original inferior oblique attachment. This distance is determined by adding the distance from the limbus to the external rectus attachment to 10 mm. (the average distance of the external rectus tendon to the anterior tip of the inferior oblique attachment). When one point of the adjusted caliper is placed on the limbus, the other point will determine the point where the new line of attachment should begin. If you wish to recess the muscle 6 mm., the measurement with the caliper is taken 4 mm. below the level of the lower end of the lateral rectus tendon. It will be recalled that the average attachment of the inferior oblique is 2 mm. higher than the lower end of the insertion of the lateral rectus.

When the anterior point of reattachment of the muscle is determined, it can be marked with a small spot of gentian violet. With this technic, the direction of muscle action will remain approximately the same as it was before it was detached from its insertion. A certain amount of abnormal action should result if the reattachment is not in the line of muscle traction.

The effect of high myopia on the position of the inferior oblique insertion should be kept in mind. In high myopia, the insertion is more posterior and the muscles assume a greater forward angle. In reattaching the muscle, the new insertion should be 1 to 2 mm. more posterior.

## CONCLUSION

An anatomical study is made of the superior and inferior oblique muscles. The problem is confined to the insertion of these muscles and the relationship to the adjacent rectus muscle. The work is approached from the clinical standpoint and with the hope that, with a broader knowledge of this anatomy, our ability to diagnose and treat some of the obscure muscle defects will be enhanced.

Although the series of specimens is small and the data is restricted to only the insertion of these muscles, certain facts seem evident.

A. Data obtained indicates that the anatomical variations are more numerous in the insertion of the superior oblique than any other rectus muscle. Variations in the insertion of

the inferior oblique are next in frequency.

B. Because of the wide variations in the position of the insertion of these muscles, the study suggests the possibility that a so-called "paretic" muscle may be abnormal in action because of an unusual insertion. It seems likely that many of the obscure actions of these muscles may be explained by variation in insertion to the eyeball.

C. In considering the superior oblique muscle, the work indicates that a favorable and feasible surgical approach could be made at its insertion.

D. In the case of the inferior oblique, the study suggests the possibilities of a more satisfactory surgical management. Surgery can and should be done on the insertion rather than the origin. This is due to the easier approach and the nature of the anatomy.

E. The study also serves to show the possible intimate relation of the inferior oblique muscle to the optic nerve and other important structures. This should be kept in mind in operation upon the muscle.

Eye muscle surgery would take a step forward if a more accurate technic could be applied to the surgery of the oblique muscles. As matters stand now, we have limited possibilities for correcting an oblique muscle defect. This state of affairs is due apparently to our lack of knowledge concerning the anatomy of the obliques.

It is surprising how little data there is available concerning the variations in these muscles. The inquiring student is faced with only generalities. If the need for more exact data on this subject could be appreciated, more work would be forthcoming, and it would open up greater possibilities for doing more scientific work on these obscure and poorly understood muscles.

## REFERENCE

1. Whitnall, Samuel Ernest: Anatomy of the human orbit and accessory organs of vision, H. Frowde, Hodder & Staughton, London, 1921. An Oxford Medical Publication.

## DISCUSSION

F. BRUCE FRALICK, M.D., Ann Arbor, Mich.: Dr. Fink has started on a very commendable research project, which when completed will add materially to the general knowledge of the anatomy of the extraocular muscles. The statistical analysis of the results of the investigation can be completely appreciated only after careful study of his beautifully exhibited display in the scientific exhibit. Only then are you able to comprehend the thorough and meticulous care needed to supply the information given us today.

A fearful approach to muscle surgery is engendered by uncertainty of diagnosis, physiology or anatomy of the extraocular muscles. Acquisition of the anatomical relationships portrayed by Dr. Fink will lead to a better brand of muscle surgery than is generally being done today.

The various measurements of the eye muscles need not necessarily be remembered in their entirety, since all are not of too much clinical value. Some of the measurements given aid the ophthalmic surgeon in locating the muscles at operation. For instance, if the surgeon did not know that the global insertion of the inferior oblique was about 10 millimeters posterior to the insertion of the external rectus, difficulty might be experienced in finding the insertion when recession or resection of this muscle is attempted. If we remember that the anterior edge of the superior oblique tendon is only about 2.5 millimeters behind the nasal edge of the superior rectus insertion, no trouble should be experienced in finding this tendon for resection or recession. All these data are not available in any of the standard texts of ophthalmology or of ophthalmic anatomy.

For many years we have been teaching our residents the necessity of retaining normal anatomical relationships of eye muscles when doing muscle surgery. We must have great respect for the fibrous sheath of the muscle and leave it intact. Stripping of the sheath from the muscle and tendon leads to adhesions of the muscle to the sclera.

Dr. Fink has emphasized that in recessions the tendon should not only be spread out to the normal breadth of insertion found in this eye, but the recession should be measured equally from both ends of the scleral insertion. The

only exception to this rule is in those rare eyes in which the line of insertion is definitely abnormal. A muscle cannot be adequately reattached to the globe with a single central suture and expect that attachment to have a spread of approximately 10 millimeters. Very likely a narrow point of attachment would have little effect on the efficiency of the lateral and mesial recti when the eye is moved in the horizontal plane. This narrow point of insertion could conceivably have a marked effect on the efficiency of these same muscles when they are used in the oblique or vertical positions of gaze.

I can see no excuse for the rather common practice of reinserting a mesial or lateral rectus muscle higher or lower in an attempt to correct a hyperphoria or hypertropia. Such a practice is an admission of inadequate diagnosis of the anomaly at hand.

I agree perfectly with Dr. Fink on the advisability of spreading the scleral insertion of the obliques to their normal insertion width when recessed or resected. This principle should apply just as much to the obliques as to the rectus muscles. Reattaching the oblique is far more difficult than the recti, and considerably more manual dexterity is required to insert two sutures to obtain the normal width of the attachment. This is especially true when the inferior oblique is alone recessed without taking off the external rectus. More exposure is afforded when recession of the inferior oblique is combined with resection of the lateral rectus, but even then I have found difficulty in placing the posterior suture far enough back on the globe.

My poorest results from recession of the inferior oblique have resulted from incomplete severence of the scleral attachment posteriorly. Since this attachment extends within 5 millimeters of the optic nerve, we are working blindly with scissors in making this section. It is, therefore, not difficult to leave some of the posterior fibers intact.

I cannot agree with Dr. Fink's interpretation of the line of action of the inferior oblique and his method of suturing the inferior oblique to the sclera along this line. If he reattaches the anterior end of the tendon the same distance from the cornea as it was originally, he is placing the new insertion too far posteriorly and his new line of action will be more oblique than formerly. It is my belief that the new insertion should be along the normal line of action of the muscle. This line of action is obliquely forward and down around the globe.

Dr. Fink has pointed out the relationship of the superior rectus to the superior oblique. I feel that an even more important relationship is that of the superior rectus and the levator. The union of the superior rectus with the levator is so strong that if the superior rectus is recessed too much the upper lid will be elevated, giving an apparent exophthalmos on this side. Conversely, if the superior rectus is resected too much a relative ptosis will result. These same relationships are present in the lower lid, although here we have no levator tendon. There are fascial bands from the inferior rectus going to the lower tarsus which act in the same manner as the levator of the upper lid. Too generous recession of the inferior rectus widens the palpebral fissure by the lower lid being pulled down. Too extensive resection of the inferior rectus causes elevation of the lower lid with a resultant narrowing of the palpebral fissure.

I have enjoyed reading Dr. Fink's research material and feel that much good will come out of a re-evaluation of muscle relationships and the part they play in diagnosis and surgery.

RAYNOLD N. BERKE, M.D., Hackensack, N. J.: Dr. Fink's presentation this afternoon is in my opinion very timely and to the point. It is timely because never before have so many operations been proposed for the treatment of hypertropia associated with over-action or under-action of the oblique muscles. Briefly, these operations are as follows:

To decrease the action of the inferior oblique muscle, Dr. J. H. Dunnington, a number of years ago, advocated myectomy, tenotomy, or myotomy of the over-active muscle. Later Dr. White described an operation for recession of the inferior oblique muscle to decrease its action.

To increase the action of the inferior oblique muscle, Dr. J. M. Wheeler designed an operation for advancing the tendon of the inferior oblique muscle over the lower margin of the orbit. Later Dr. J. W. White suggested tucking the inferior oblique muscle to increase its action.

To increase the lifting power of the superior oblique muscle, Dr. J. M. Wheeler; a number of years ago, designed an operation for tucking the reflected tendon of this muscle. Recently Dr. J. M. McGuire has described an operation, which I understand is now in print, for resecting the tendon of the superior oblique muscle to increase its action.

To decrease the action of the superior oblique muscle, Dr. W. L. Hughes advocated recession of its pulley. Today Dr. Fink suggested that it may be feasible to tenotomize the superior

oblique muscle lateral to or under the superior rectus muscle. Three months ago I described an operation for tenotomy of the superior oblique tendon on the nasal side of the superior rectus.

This summary gives a total of eight different operations which we have available for the treatment of hypertropia associated with dysfunction of one or more of the oblique muscles.

Those surgeons who do these operations frequently say that they are easy and simple to do. Those who do them rarely or not at all believe they are difficult. This difference of opinion I believe is due to the fact that those who do them frequently are familiar with the anatomy. Those who do them rarely are not so familiar with it; therefore, I say that Dr. Fink's paper today is very much to the point because, as we all know, the better we know our anatomy, the better surgeons we will be.

Dr. Fink's paper brings up a number of interesting and practical points. I should like to enlarge briefly on two or three of them.

(Slide) Dr. Fink has shown that the insertion of the inferior oblique muscle begins about 10 millimeters posterior to the inferior edge of the insertion of the external rectus and from this point recedes backward in the average case for a distance of about 10 millimeters. The entire insertion of the inferior oblique usually lies under the inferior half of the belly of the external rectus. Obviously, in doing a recession of the inferior oblique it is necessary to keep this relationship in mind. It is also important to keep this relationship in mind in every operation on the external rectus, because it is possible to accidently tenotomize this muscle during a resection, a recession, a tucking, or an advancement procedure. I know of one such accident in which the inferior oblique muscle was accidently tenotomized during a routine resection of the external rectus muscle.

This drawing is intended to explain how this accident may develop. After the conjunctiva has been incised and an opening made in Tenon's capsule, the muscle hook is passed under the external rectus muscle, and during this maneuver the belly of the inferior oblique is accidently picked up either because the insertion is farther forward than normal or because the hook is passed too deeply into Tenon's space or for some other reason the inferior oblique is brought forward to a position directly under the insertion of the external rectus muscle. One can readily see how easy it would be for the surgeon, when freeing the external rectus muscle from Tenon's capsule, to accidently sever the inferior oblique muscle. In the case I referred to, a second operation was done at which time the inferior oblique was found attached to the sclera at the insertion of the external rectus muscle, having been completely severed from its normal attachment. It is only necessary to keep this possibility in mind in doing operations on the external rectus to avoid this complication.

(Slide) Dr. Fink has shown that the reflected tendon of the superior oblique muscle passes only 2 or 3 millimeters posterior to the insertion of the superior rectus muscle. This is important, as already brought out by him and Dr. Fralick, because in doing an operation on the superior rectus the tendon of the superior oblique may accidently be severed. I know of one such accident.

Dr. Fink has suggested that it may be possible to tenotomize the superior oblique muscle lateral to the superior rectus muscle. In this he is perfectly right, but unfortunately tenotomy of the superior oblique at this place is difficult and uncertain. It is difficult because the insertion of the superior oblique extends so far posterior to the equator. It is uncertain in its effect, because one cannot be sure that all the fibers have been severed from the globe. Unless they are all severed, little or no effect will result. Out of four tenotomies of the superior oblique done lateral to the superior rectus at the Eye Institute, Presbyterian Hospital, N. Y. C., only one was entirely successful. The three unsuccessful cases were attributed to incomplete tenotomy.

A better place to tenotomize the tendon of the superior oblique muscle is on the nasal side of the superior rectus muscle. This is the site of choice for tenotomy of the superior oblique muscle for the following reasons:

First, the reflected tendon of the superior oblique muscle at this point is well forward being only 2 or 3 mm. from the insertion of the superior rectus muscle.

Second, the width of the tendon of the superior oblique muscle at this point is only 5 or 6 mm., the tendon being quite compact. These two facts make it easy to pick up the reflected tendon of the superior oblique muscle.

Third, if the operation is done at this point, it is not necessary to remove the superior rectus or to disturb its insertion, as would be the case if the operation were done lateral to the superior rectus muscle.

Fourth, there are at this point no important nerves, arteries, veins, or other periocular structures which might accidently be injured during the operation.

Finally, if the operation is done here, it will be done under Tenon's capsule and within the sheath of the superior oblique muscle. If the operation is done here comp'ete paralysis of the superior oblique muscle will not follow the operation.

(Slide) This drawing is an attempt to illustrate why tenotomy of the superior oblique muscle on the nasal side of the superior rectus muscle does not result in complete paralysis. This is the right eye; this is the cornea; this is the superior rectus; this is Tenon's capsule; this is the reflected tendon of the superior oblique muscle, and here lies the trochlea of the superior ob'ique muscle. You will note that there are numerous adhesions or fine bands running from the tendon of the superior oblique muscle to its sheath. You will note also that the sheath blends with the sheath of the superior rectus and with Tenon's capsule. This explains why tenotomy or tenectomy of the superior oblique muscle does not result in complete paralysis, because of the many adhesions between the tendon of the superior oblique muscle and its sheath on the one hand and the several attachments of this sheath to the globe on the other hand.

Dr. Fink's paper brings up a number of interesting points. I should like to ask him just one question. Given a patient in whom he has been able to diagnose clinically a dysfunction of one or more of the oblique muscles, can he tell us from his clinical study what the anatomical condition is.

I think Dr. Fink has done a fine piece of work, and I want to thank him for bringing to us the benefit of his study.

Thank you very much.

WALTER H. FINK, M.D., Minneapolis, Minn.: I appreciate very much the discussion of Dr. Fralick and Dr. Berke.

In answer to the question as to the interpretation of the anatomical variations, I cannot with certainty diagnose a case of abnormal insertion. There is the possibility that a certain percentage have abnormal muscle action because of an abnormal insertion.

I believe that tenotomy of the superior oblique should be done on the tendon and not on the insertion. I think Dr. Berke is correct, and I am in full agreement with his approach because to me it seems the most logical procedure.

I thank you.

# RECONSTRUCTION OF THE EYELID

ARTHUR E. SHERMAN, M.D.
EAST ORANGE, N. J.

FROM the summer of 1944 to the spring of 1946, nine of the Army General Hospitals in this country were designated as plastic and ophthalmic centers. Of the large volume of casualties evacuated to these centers for reconstructive surgery during that period, each center handled from 300 to 400 patients requiring ophthalmic reconstructive surgery. In some of these centers this type of surgery was the function of the Eye Service, in others of the General Plastic Service.

Certainly all of us who did much of this work are greatly indebted to the late Dr. John M. Wheeler for the methods and principles developed and advocated by him. Wendell Hughes' method of total eyelid reconstruction has also been largely used. Both of these men have been strong advocates of the principle that whenever possible eyelid tissues should be replaced by eyelid tissues.

When there has been a loss or contracture of eyelid skin alone from burns or lacerations producing ectropion or lagophthalmos, we can expect practically perfect results if we carefully follow in detail the method used by Wheeler[6] for replacing eyelid skin. There is some tendency on the part of general plastic surgeons to take short cuts by using an epidermic inlay or epithelial outlay type graft without lid adhesions but rarely does the end result equal that obtained by Wheeler's method, and too often there is an additional deformity produced, especially a noticeable cicatricial epicanthus type deformity. I know of no other method that gives comparable results to the technic perfected by Wheeler utilizing a full thickness or Wolfe graft carefully sutured in a bed prepared by thorough excision of scar tissue plus the use of intermarginal lid adhesions which are left in place from two to three months. As pointed out by Wheeler[7] the graft should be taken from the upper eyelid if possible, skin from the postauricular angle being second choice. As pointed out by J. B. Brown[1] and others, skin from the supraclavicular or lower neck area gives a fairly good color match although slightly thicker than desirable. I cannot agree with Brown in his statement that, "where pressure alone cannot be relied on—as on the lids—the fixation is obtained and the proper opening of the area and the proper stretch of the graft obtained by the long sutures being tied over cotton mechanics waste carefully packed into place." As with most plastic procedures of the eyelids a good pressure dressing is extremely important, such as that described by Pfeiffer.[4] This should not be changed for five or six days, at which time it is reapplied after removing the lid adhesion mattress sutures.

For small vertical full thickness loss of an eyelid and for vertical scars with notching of the lid margin, Wheeler's[8] "halving" type of repair is the procedure of choice. Larger defects or traumatic colobomata of the eyelids up to as much as one-third loss can be satisfactorily corrected in similar manner by releasing the lid laterally by means of an extended lateral canthotomy and cutting the tarsal attachment to the lateral ligament. When a half of the lid is lost the operation is enlarged to a sliding flap of the Celsus-Knapp type from the temporal

From the Surgical Service, O'Reilly General Hospital, Springfield, Mo.

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

side as described by Wheeler[9] for correction of congenital coloboma. These procedures are very well described by our secretary, Dr. A. B. Reese,[5] in the September, 1944, *Archives of Ophthalmology*. His description of the "halving" procedure is of the method used by Wheeler in his later years, and is simpler than the procedure described in Wheeler's earlier papers and in several textbooks.

Except in older people, I believe that Wendell Hughes'[2] method of eyelid reconstruction is to be preferred to Wheeler's sliding flap correction of loss of one-half of the eyelid. The lateral linear scars produced by the latter procedure may be quite noticeable in the smooth skin of younger individuals. Hughes[3] has recently described another ingenious method of reconstructing a loss of the nasal (or temporal) half of the upper eyelid by use of a sliding flap of tarsus from the same lid which is temporarily fixed to the opposing lid margin and upon which is placed a free skin graft from the opposite upper eyelid.

Almost without exception one should avoid the use of pedicle flaps from the forehead or cheek for eyelid reconstruction. This not only produces a lid that is too thick and of poor color but also causes additional scarring to the forehead or cheek.

Hughes' method of total reconstruction of the lower eyelid gives excellent results when little more than the tarsal portion of the eyelid is missing, but I believe it is also the method of choice when there has been a more extensive loss down to the lower orbital margin. For these more extensive losses it has at times been found advantageous to elevate the skin and subcutaneous tissue below by means of sliding flaps, the skin and remains of conjunctiva being joined to the upper tarsus by the Hughes' procedure at a second operation. Following that, a free skin graft from the upper eyelid to the future lower eyelid has been helpful to elevate the future eye fissure. This type of case often has an accompanying loss of lower orbital margin for which a bone graft from the crest of the ilium has given the most satisfactory results. The cancellous bone from the ilium is easily shaped and will be firmly attached to the adjacent bone in a few weeks. In our experience a bone graft gives far superior results to those obtained with cartilage or inorganic substances used to replace lower or lateral orbital margin.

In my experience the most satisfactory eyelash grafts have been those taken from the mid-portion of the nasal half of the eyebrow. The strip of hair-bearing skin should be 3 mm. wide and slightly wider on its under surface than on the skin surface so that the hair follicles close to the edge are not damaged. For the upper eyelid the graft is taken from the eyebrow on the same side; for the lower eyelid it is taken from the opposite eyebrow and reversed in direction when suturing in place. In preparing the bed for the graft only one horizontal skin incision is required, placed about 2 mm. from the future lid margin. Small triangles of skin are excised at either end of the incision to form a narrow rectangle. Scar tissue is excised down to the tarsus.

Total reconstruction of the upper eyelid does not give as satisfactory results as for the lower lid, but I believe the Hughes' procedure is to be preferred for this. After joining the remains of the conjunctiva from above to the elevated tarsus from the lower eyelid, a thin Thiersch graft is more likely to take than a full thickness graft. However, if the bed for the graft appears good, and sufficient skin from the opposite upper eyelid is available, a full thickness skin graft of eyelid skin is preferred.

The following 3 cases illustrate the use of various types of full thickness skin grafts to replace loss of eyelid skin.

FIG. 1 — Case 1 — Ectropion of right upper eyelid and loss of lateral half of eyebrow from burns.

FIG. 2 — Case 1 — Final result following skin graft to eyelid and eyebrow graft.

Case 1. (Figs. 1 and 2) S/Sgt. D. Second and third degree burns of forehead and eyelids when plane was shot down over Italy, July 16, 1943. Taken prisoner of war. Admitted to O'Reilly General Hospital on July 7, 1945. Cicatricial ectropion of right upper eyelid with some loss of lateral half of eyebrow. Wolfe graft from left upper lid to right upper eyelid with two lid adhesions, October 18, 1945. Eyebrow graft January 8, 1946. Lids cut apart in January, 1946. Good functional and cosmetic final result.

FIG. 3 — Case 2 — Severe ectropion of right eyelids and lagophthalmos of left, one year after burns of face.

FIG. 4 — Case 2 — Ectropion and lagophthalmos corrected by skin grafts from postauricular angle. Still needs eyebrow grafts and plastic surgery for deformity of nose and lips.

Case 2. (Figs. 3 and 4) S/Sgt. H. Severe ectropion of both right eyelids with cicatricial lagophthalmos of left; result of burns incurred when his B-24 was shot down April 26, 1944.

FIG. 5 — Case 3 — Three weeks after skin graft.

FIG. 6 — Case 3 — One month after lid adhesions cut, and five months after skin grafting.

Was prisoner of war for nine months. Admitted to O'Reilly General Hospital March 1, 1945. Right eyelids corrected by Wolfe grafts of post-auricular skin with 3 lid adhesions on March 20, 1945. Left corrected by similar grafts on November 21, 1945. Lid adhesions cut four months after surgery. Good final result.

Case 3. (Figs. 5 and 6) Sgt. A. Developed cicatricial ectropion of both lower eyelids following burns of face when his tank destroyer was struck by enemy shell Oct. 14, 1944. Admitted to O'Reilly General Hospital three months later and on February 5, 1945 the ectropion was corrected by full thickness skin grafts from the supraclavicular area. Lid adhesions cut in June, 1945. Good final results.

The use of Wheeler's halving repair is illustrated by the following 2 cases.

Case 4. (Figs. 7 and 8) Lt. O. Lacerated wound of right eye, eyelids and cheek from shell fragment on September 10, 1944, with fracture of zygoma and maxilla. Admitted to O'Reilly General Hospital on October 20, 1944. Dense vertical linear scars of nasal third of both right eyelids with notch of margins and some ectropion of lower eyelid. Excision of scar of upper eyelid and halving type repair in December, 1944. Similar procedure of lower

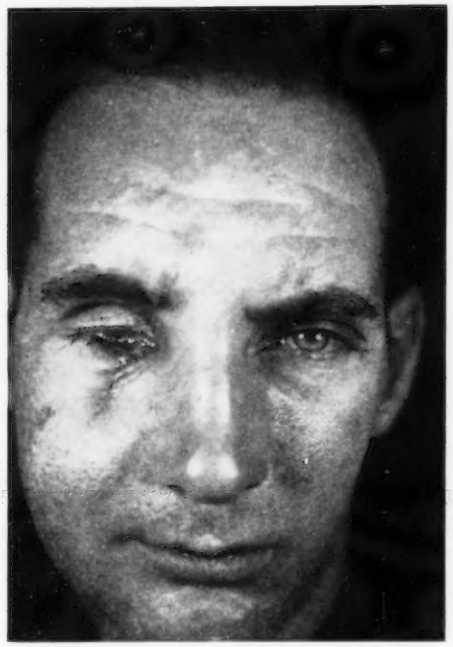


FIG. 7 — Case 4 — Vertical scar and notch deformity of both right eyelids with loss of right eye from shell fragment wound.

FIG. 8 — Case 4 — Final result after "halving" type repair of both eyelids and skin graft to lower eyelid.

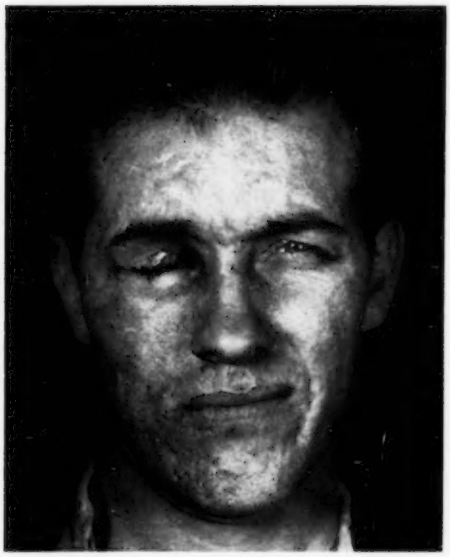


FIG. 9 — Case 5 — Loss of right eye and middle third of upper eyelid.

FIG. 10 — Case 5 — Lid defect repaired by sliding temporal flap with halving type closure.

eyelid one month later. Excision of additional scar tissue of lower eyelid, skin graft from upper eyelid, and lid adhesion in February, 1945. This procedure repeated in May, 1945, because lid adhesion did not hold. Lid adhesion cut in September, 1945 and eye prosthesis fitted. Satisfactory result.

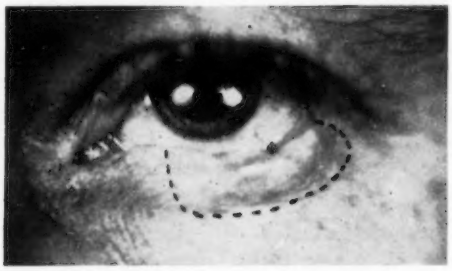


FIG. 11 — Case 6 — Showing portion of lid excised.

FIG. 12 — Case 6 — Final result.

Case 5. (Figs. 9 and 10) Pvt. P. Multiple penetrating wounds including right eye and upper eyelid by grenade fragments on July 4, 1944. Eye enucleated that day. Admitted to O'Reilly General Hospital on August 30, 1944. Loss of middle third of upper eyelid with mass of granulomatous tissue projecting through defect and adherent to it. Excision of granulomatous tissue of upper cul-de-sac in November, 1944. Closure of defect of upper lid by halving type closure with extended canthotomy and freeing of lateral end of upper tarsus from ligament in January, 1945. Excision of scar tissue of upper socket and mucous membrane graft from inner surface of lower lip in July, 1945. Fascia lata graft as filling below brow in September, 1945. Final result fairly satisfactory. Eye fissure is too low.

The total reconstruction of the tarsal portion of the upper or lower eyelid by Hughes method is illustrated by the following 2 cases.

Case 6. (Figs. 11 and 12) Pfc. S. Admitted to O'Reilly General Hospital on November 24, 1944. Diagnosis of basal-cell epithelioma of lateral two-thirds of left lower eyelid, involving chiefly the tarsus. On December 2, 1944 the lateral three-fourths of the eyelid was entirely excised and the skin and conjunctiva from below joined to the upper tarsus according to the Hughes procedure. Eyelash graft from cen-

FIG. 13 — Case 7 — Loss of left eye and middle half of upper eyelid from ricochetted bullet.

FIG. 14 — Case 7 — Final result after Hughes' type repair.

FIG. 15 — Case 8 — Loss of right eye, entire lower eyelid, and lateral two-thirds of upper eyelid margin.

FIG. 16 — Case 8 — Final result.

tral nasal portion of right eyebrow to lateral three-fourths of future left lower lid three months later. New palpebral fissure cut one month later. Final result two months later very satisfactory.

Case 7. (Figs 13 and 14) T/Sgt. W. Anophthalmos and loss of middle two-thirds of tarsal portion of left upper eyelid from richochetted bullet August 9, 1944. Eye enucleated and small split skin graft to upper eyelid at a Polish hospital in Italy. Admitted to O'Reilly General Hospital on November 12, 1944. Eyelids joined by first stage Hughes' procedure on December 27, 1944. Eyelash graft two months later. Split skin graft replaced by Wolfe graft from right upper eyelid on May 2, 1945. Lids separated two months later and fitted with eye prosthesis. Very satisfactory result.

The combination of Hughes' method of reconstruction combined with other procedures was used in the following cases having more extensive total eyelid loss.

Case 8. (Figs. 15 and 16) Cpl. B. Anophthalmos with loss of entire right lower eyelid and lateral two-thirds of upper eyelid margin result of bullet wound June 18, 1944. Admitted to O'Reilly General Hospital on September 27, 1944. Had had debridement and enucleation at an evacuation hospital the day he was wounded. On November 28, 1944 the remains of the upper eyelid were joined to the skin and conjunctiva from below. Eyelash grafts from midportion of both eyebrows to lateral two-thirds of both eyelids on February 1, 1945. One month later a skin graft from upper lid to lower lid. Three months later the lids were separated. In June, 1945, the lower cul-de-sac was enlarged with a mucous membrane graft. One month later the fissure was shortened by a lateral canthoplasty. In September, 1945, the Kuhnt-Szymanowski procedure was used to correct the relaxed lower eyelid. One month later a 16 mm. grooved glass sphere was implanted in the muscle cone for retracted socket. Final result only fair because fissure is too low and lateral canthal angle too rounded.

Case 9. (Figs. 17 and 18) Capt. R. Incurred severe bullet wound entering right preauricular area and exiting at right side of nose, which resulted in destruction of zygomatic arch, lateral orbital margin, eyeball and entire upper eyelid, on June 9, 1944. Admitted to O'Reilly General Hospital one month later. Broad, depressed granulating area from preauricular area to orbit with loss of entire upper eyelid, drawn down eyebrow and deformity of medial canthal region. Shrunken, retracted remains of eyeball

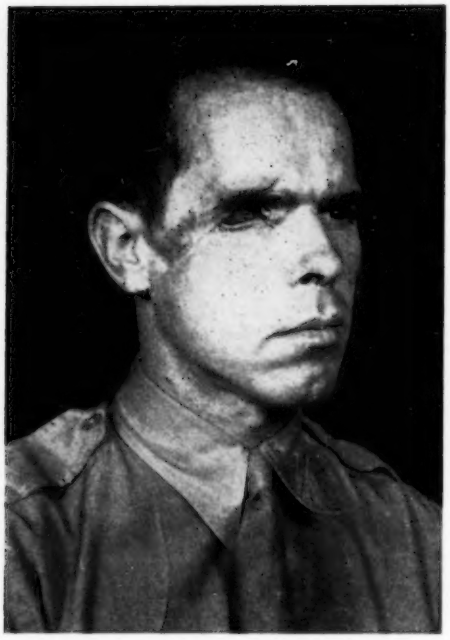


FIG. 17 — Case 9 — Loss of right eye, entire upper eyelid, and lateral orbital area from bullet wound.

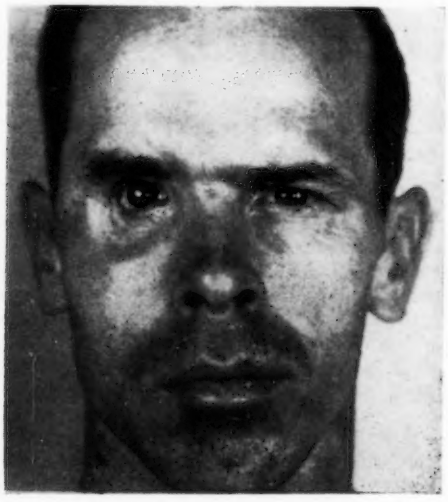


FIG. 18 — Case 9 — Final result.

present and a chronic dacryocystitis. The granulating area was allowed to heal and the lacrimal sac removed. The remains of the eye was enucleated in September, 1944. From January, 1945, to May, 1946, eight operations were performed to reconstruct the upper eyelid. These include a Hughes' type procedure with epidermal graft, skin graft from opposite upper eyelid, Z-plasty of eyebrow, eyelash graft, Z-plasty to elevate medial canthal area, cutting the lids apart, and two buccal mucous membrane grafts to enlarge the eye socket. An iliac bone graft to the lateral orbital area was performed by Major Douglas Macomber of the General Plastic Service before the eyelids were cut apart. The final result is only fairly satisfactory in that the fissure is short horizontally and the upper eyelid and eye prosthesis have little movement.

FIG. 19 — Case 10 — Loss of right eye and entire lower eyelid area including lower orbital margin from shell fragment wound.

Case 10. (Figs 19, 20 and 21) Cpl. F. Shell fragment wound of right eye with avulsion of lower eyelid on November 24, 1944. Eye eviscerated a week later. Admitted to O'Reilly General Hospital on January 15, 1945. Loss of entire lower eyelid with conjunctiva and skin adherent to remains of lower orbital margin. Broad irregular scar temporal from missing lateral canthus. Elevation of skin and conjunctiva below by sliding skin flaps, large flap with base temporal, small flap with base nasal, February, 1945. First stage Hughes, to join skin and conjunctiva from below to upper lid, May, 1945. Wolfe graft from upper lid to lower lid, August, 1945. Eyelash graft to lower eyelid, October, 1945. Iliac bone graft to lower orbital area in January, 1946. Lids cut apart three months later and mucous membrane graft to lower fornix in May, 1946. Fascia lata filling below brow in June, 1946. Final result fairly satisfactory.

FIG. 20 — Case 10 — Has had first four operations for reconstruction of lower eyelid. Bone graft to lower orbital margin will be next operation before lids are cut apart.

FIG. 21 — Case 10 — Final result.

## REFERENCES

1. Brown, Lt. Col. J. B. and Cannon, Major B.: Full-thickness skin grafts from the neck for function and color in eyelid and face repair, Ann. Surg., 121:639-643 (May) 1945.
2. Hughes, W. L.: A new method for rebuilding a lower lid, Arch. Ophth., 17:1008-1017 (June) 1937.
3. Hughes, W. L.: Reconstruction of the lids, Am. J. Ophth., 28:1203-1211 (Nov.) 1945.
4. Pfeiffer, R. L.: Pressure dressing, Am. J. Ophth., 23:1156-1158 (Oct.) 1940.
5. Reese, A. B.: Partial resection of lid and plastic repair for epithelioma and other lesions involving margin of lid, Arch. Ophth., 32:173-178 (Sept.) 1944.
6. Wheeler, J. M.: Free dermic grafts for the correction of cicatricial ectropion, Am. J. Ophth., 3:251-255 (April) 1920.
7. Wheeler, J. M.: Ectropion, South. M. J., 29:377-382 (April) 1936.
8. Wheeler, J. M.: War injuries of the eyelids, Arch. Ophth., 49:35-42 (Jan.) 1920.
9. Wheeler, J. M.: Treatment of tumors of the eyelids by surgery, New York State J. Med., 39:870-874 (May) 1939.

## DISCUSSION

EDMUND B. SPAETH, M.D., Philadelphia, Pa.: Dr. Sherman has had a tremendous experience recently in ophthalmic plastic surgery. His experience is a clinical one. We have seen some of his results and it behooves us to profit by these experiences of his. Differences of opinion are natural—perhaps even healthy. My discussion, while it does express a difference of opinion on some of his points, in the final analysis simply supplements and emphasizes some of his points and statements.

First, as to his dislike for the use of pedicle flaps, I see no way out of it but that they must be used in certain cases. Underlying disease of the bone structures and the presence of deep cicatrices makes it almost impossible to utilize, with full satisfaction, free skin grafts, either razor cut or full thickness. Dr. Sherman calls attention to his satisfaction with the use of supraclavicular skin—almost any amount of this can be easily and safely transplanted by means of a delayed transfer pedicle flap from that region to the region of the eyelids. Even more, however, is his dislike for pedicle flaps from the immediate neighborhood of the eyelids. Given a case with an intact eyeball and with a marked deficiency in the conjunctiva, there is no nicer way of transplanting both simultaneously than that of lining a pedicle flap first with the mucous membrane and then secondarily transplanting both to the new position. (Slide 1).

Second—slide 2 answers the problem of bone disease now quiescent as the result of osteomyelitis, and sequestrectomy. Also it shows my preference for the use of cartilage, in this instance an autogenous graft,—though cadaver cartilage might have been as satisfactory, as compared to Dr. Sherman's desire to use cancel-

lous bone from the ilium by preference. Frankly, I have discontinued the use of autogenous bone grafts wholly and for at least five years. This may be an error on my part, but it seems that the cartilage grafts have done as well.

Third—I agree wholly and without equivocation relative to Dr. Sherman's statement in regard to pressure dressings and his disagreement with Brown in reference to this. The sutures that Brown has used are not only unnecessary but really unwise. They must be introduced; they must be removed; they are a wick for additional low grade secondary infection of the graft, cause additional scarring, limit the size of the graft possible, and give a pressure dressing—to be true—but one less satisfactory than that obtained by a mold of dental stent or dental wax fitted to the site for the graft or grafts immediately prior to their placement in position.

One other point—Dr. Sherman emphasizes the necessity for intermarginal adhesions with free skin grafts to the eyelids. I agree to this without argument—when both upper and lower lids are simultaneously corrected. When the upper lid, however, needs correction alone and the conjunctiva is adequate, intermarginal adhesions are not necessary.

Slide 3 is such a case. It illustrates the ectropion residual following a severe herpes ophthalmicus necroticans. The patient was blind in her opposite eye, and intermarginal adhesions would have limited her vision markedly during the process of recovery. The same opinion applies to an isolated correction for ectropion in the lower lid if similar circumstances are present. Temporary intermarginal sutures must be used during the postoperative dressing to protect the cornea from damage, but as soon as the dressings are permanently removed, then also can the sutures be removed.

Dr. Sherman gives credit to Dr. Wendell Hughes for a principle in total eyelid reconstruction which I also want to mention. A tremendous amount of credit belongs to Dr. Hughes properly for the work he has done in reconstructing these difficult cases. It in no way detracts from this credit to call attention to the fact that the underlying principles connected with this procedure were first presented by Landolt and subsequently augmented considerably by Axenfeld. This criticism may seem trite but it is proper. It was a pleasure to discuss this paper and I am complimenting Dr. Sherman on his beautiful work.

Thank you.

JAMES N. GREEAR, JR., M.D., Washington, D. C.: The Army Medical Corps has, indeed, been fortunate in having available men with such thorough understanding of ophthalmic plastic surgery, as Major Sherman's work so ably demonstrates. I believe that we are all agreed as to our indebtedness to the late Dr. John M. Wheeler for the principles of this branch of surgery as developed and laid down by him. Dr. Wendell Hughes has made a valuable contribution to reconstructive surgery of the lids and the procedures advocated by him have proven extremely useful, especially where there has been extensive damage to one of the eyelids.

It has been my experience that there is more redundant skin on the eyelids of old people, thus making the procedures advocated by Hughes much more feasible than in the case with younger individuals.

It is essential that uniform pressure be maintained over the grafted area in any plastic procedure. This is particularly true where free grafts are used. It is especially difficult to maintain such pressure after grafting an eyelid, if enucleation has been previously performed.

The method of applying pressure as advocated by Dr. Barrett Brown yields excellent results in such cases. Dr. Brown's procedure is to leave the sutures long around the margin of the free graft, tying these over mechanics' waste so as to produce uniform pressure.

There is certainly no more adequate operative measure for correction of notching of the lid margin than Wheeler's "halving" operation.

I should like to emphasize the importance of early treatment and care of severe injuries to the eyelids. Due to the marked increased activity of our Air Forces, and motorized units of troops in the recent war, there were large numbers of patients with severe burns involving the face. These patients were given emergency treatment in evacuation or station hospitals, and very soon were returned to general hospitals where they got definite care. It is generally recognized that probably the most serious sequelae of such an injury is damage to the eye from pressure. This was inevitable unless preventive measures were instituted at a very early stage. In order to forestall cicatricial ectropion with resultant exposure keratitis, one of two procedures was generally carried out in the general hospitals in the European theater of operation.

As soon as severely burned lids began to granulate, a split thickness skin graft was placed over the raw surfaces, which served as a dressing graft and prevented extensive contracture of the lids. A dressing graft may be taken from another individual, in which case it serves for only a limited period of time. This was necessary in exceptional cases where it was felt that further loss of skin would be detrimental to the patient. This procedure was helpful and no doubt it was responsible for preventing serious damage to the eyes of many patients. Even if the patient was most severely burned about the face, there was seldom serious damage to the lid margins; therefore, intermarginal adhesions were quite feasible in these cases. The formation of these adhesions at an early stage prevented the marked cicatricial ectropion which was certain to develop. This procedure could be followed by early grafting, if extensive damage to the skin and subcutaneous tissue predicted extensive cicatricial contracture as an end result. These temporary grafts could be replaced by more suitable skin at a later date if need be. This method proved to be eminently satisfactory and provided adequate protection to the eye.

In most instances, cicatrization could take place and suitable skin graft to the lids be made at a later period, the eye having been well protected in the meantime.

In many of the patients receiving such severe burns of the head, there was no available skin on the eyelids or postauricular area for covering the eyelids as a final operative measure. In these patients, free grafts from the supraclavicular area proved to be very satisfactory. It was found that early repair of damage to eyelids would in many instances save the patient weeks of hospitalization. Even where there was loss of more than one-third of the eyelid the defect could be corrected by utilizing the sliding flap from the temporal side. This could be carried out as a primary measure or after debridement and secondary closure, even though a period of from three to five days had elapsed since the original injury to the lid. This procedure was particularly applicable to injury to the lower lid.

If such complete repair of injury to the eyelid was not feasible, a marginal suture was placed wherever possible in order to preserve the lid margin. This was a very distinct aid in subsequent reconstruction of the lid.

In reviewing the recent literature dealing with surgery of the eyelid one is impressed with such expressions as "marked tendency to tissue separation and frequent recurrences of initial defect." Such an occurrence should be rare if one of the most important principles of plastic surgery is observed, that is, that the suture lines should never be closed under tension.

ARTHUR E. SHERMAN, M.D., East Orange, N. J.: I wish to thank both Dr. Spaeth and Dr. Greear for their discussions. I appreciate that there would be some difference of opinion as to some of these procedures. I did say in my paper that as far as pedicle flaps are concerned, one should avoid them if at all possible. I did not mean that there are not, at times, cases when we must use them.

With regard to bone grafts and cartilage grafts, again there is a great deal of difference of opinion. We used cartilage grafts, both autogenous and pickled cartilage, and we were not as satisfied with the cartilage as we were with cancellous bone from the ilium. I feel that the advantage of cancellous bone is that it is easily shaped so as to fit the defect. Within two or three weeks it is very firmly attached, that is, it has grown fast to the adjacent bone, and within another month or two the cancellous bone has been covered with a pseudocortex. The absorption of the bone graft is negligible.

I have never felt that dental stents should be used when using a full thickness graft on the eyelid but there is some difference of opinion on that.

With regard to lid adhesions, I still feel that they are extremely important if one wishes to obtain the most uniformly good result or excellent result. They serve a very important purpose in that during the period from about the second to the eighth week after operation, these full thickness grafts or Thiersch grafts will try to contract, but with the constant pull of the opposite lid, through the lid adhesions, the grafts will become very smooth and very fine textured, so that at the end of three months when the lid adhesions are cut, the graft is usually at least the size it was originally and there should ordinarily be no residual slight ectropion. I realize that Dr. Hughes' operation is a modification of older procedures, but it is a definite modification and I believe that it is more useful than some of the older procedures although there are times when some of the older procedures do have to be used.

With regard to Dr. Brown's method of using pressure on the eyelid, that again I think is simply a modification of the Gillies' form of dressing, you might say, when he used an epithelial outlay, as he called it. In the hands of

such surgeons as Dr. Brown, they usually obtain fairly good results, but I do not think that they are likely to obtain as uniformly good results; certainly in the hands of the average man doing ophthalmic plastic surgery one does obtain much more uniformly good results by following Wheeler's perfected procedure.

I am glad that Dr. Greear brought out the use of lid adhesions and the use of the Thiersch type graft as immediate care in these burned cases overseas. I can recall several that were evacuated to us and nothing other than one or two lid adhesions on each side had been done, and after a period of three months from the time they were burned, they required no additional skin and all that one had to do was to cut the lid adhesions and their lids were perfectly normal.

# THE USE OF X-RAY THERAPY FOR RETINAL DISEASES CHARACTERIZED BY NEW-FORMED BLOOD VESSELS

## (Eales' disease, retinitis proliferans): A preliminary report

JACK S. GUYTON, M.D.
BALTIMORE, MD.
*and*
ALGERNON B. REESE, M.D.
NEW YORK, N. Y.

DURING the past 18 months we have given intensive x-ray therapy to the posterior ocular segment in a series of patients with ocular diseases characterized by retinal and vitreous hemorrhages, with secondary fibrous tissue formation and with new-formed blood vessels extending into the vitreous. A total of 22 eyes in 14 patients have been so treated. The ocular disease was classified as typical Eales' disease in 8 of these patients, as atypical Eales' disease in 4, and as diabetic retinitis proliferans in 2. The x-ray therapy has given sufficiently encouraging immediate results to warrant a preliminary report.

### TECHNIC OF X-RAY THERAPY

The irradiation technic devised by Martin and Reese[1] for the treatment of retinoblastoma was utilized to give large doses of x-ray to the posterior ocular segment with minimal effects on the vulnerable anterior segment. The cones and portals employed were the same as those used in the retinoblastoma cases (cylinders 2.5 cm. in diameter shaped to fit the lateral orbital margin and the base of the nose). The following factors were employed: 250 kilovolts, 1.5 mm. Cu filter, half value layer 1.9 mm. Cu, 60 cm. target skin distance, 30 milliamperes, 2.5 cm. portals. The dosage was varied considerably inasmuch as the exact nature of the diseases and the response to deep x-ray therapy were not known. The first patients treated received 400 r (in air) three times a week, utilizing the temporal portal more frequently than the nasal. The nasal portal was later eliminated because the presence of the fellow eye hinders its proper application, whereas in the retinoblastoma cases the fellow eye has always been enucleated. Also, if the nasal portals are used for treatment of both eyes, the cross-fire through the bridge of the nose gives a double dose to this area which might produce necrosis. The original cases received 8,000 to 10,000 r through the temporal portal and 5,000 to 6,000 r through the nasal one, but in the later cases this was reduced to a total of 3,500-6,000 r given only through the temporal portal. Erythema of the skin appeared after approximately 1,000 to 1,500 r had been given to a field and became more brisk as the dosage went higher. No ulceration or other serious complications occurred. The skin healed rapidly after treatment was discontinued but late radiation changes (some atrophy and pigmentation of the skin) occurred in some patients. If the total dosage did not exceed 6,000 r, the patients could tolerate 500 r three times a week through the temporal portal alone, thus shortening the treatment time. The

From the Wilmer Ophthalmological Institute of the Johns Hopkins Medical School, Baltimore, Md., and from the Institute of Ophthalmology of the Presbyterian Hospital and the Memorial Cancer Center, New York, N. Y.

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

eyes of all patients treated were examined before and at frequent intervals during and following the x-ray therapy.

## REPORT OF CASES

The 14 cases in this series are summarized in Table 1. Certain aspects of these cases are also described briefly.

### A. Typical Eales' disease.

Case I. W. G., a 37 year old white male, had a number of small recurrent hemorrhages with rapid proliferation of blood vessels in the vitreous of each eye. There was no apparent uveitis. During four months observation without x-ray therapy, there were additional fresh hemorrhages in each eye with extension of new-formed vessels into the anterior vitreous, and at least one of the fresh hemorrhages in the right vitreous appeared to come directly from one of these new vessels. There was a questionable relationship of several of these recurrent hemorrhages to tuberculin injections. The right eye was then treated with x-ray (total dosage 13,000 r). There were no further vitreous hemorrhages in this eye and the new-formed vessels melted away with amazing rapidity. (Figs 1-4). In the untreated left eye there were four additional recurrent hemorrhages and considerable progression of new-formed vessels during the succeeding two months. We no longer felt justified in withholding x-ray therapy to the left eye, and the effect of therapy (total dosage 14,000 r) was similar to that in the right eye. (Figs. 5-7). Subsequently, a patch of healed choroiditis could be seen in the far lower periphery of the left eye, but there was no definite choroiditis in the right eye. There have been no recurrences of either hemorrhages or new-formed vessels in the 18 months since the eyes were treated. The patient was sensitive to 1:1000 mgm. OT intracutaneously.

Case II. B. deE., a 40 year old white male, developed an anterior uveitis in both eyes in March, 1944. The uveitis subsided within several weeks, but the patient developed massive vitreous hemorrhages in the right eye in August, 1944, and in the left eye in February, 1945. On July 3, 1945, he was found to have in the right eye extensive retinitis proliferans with an abundance of new-formed vessels and partial retinal detachment. In the left eye there was only a faint red reflex. X-ray therapy was given both eyes (total dosages were right 12,000 r, left 10,000 r). The new-formed vessels in the right vitreous thereupon disappeared completely and there was considerable regression of the fibrous bands extending into the vitreous and of the retinal detachment. The left fundus still could not be seen when last examined (September 15, 1946). He was sensitive to 1:1000 mgm. OT intracutaneously.

Case III. S. M., a 24 year old white female, had recurrent retinal and vitreous hemorrhages for two years before she was first examined January 6, 1944. She had developed glaucoma in the right eye ten days previously, with new-formed vessels on the anterior surface of the iris. This eye was enucleated, and showed, in the posterior segment, retinal and vitreous hemorrhages with fibrous tissue and new-formed vessels extending from the retina into the vitreous (Fig. 8). The vitreous contained a moderate amount of partially absorbed hemorrhage. There was an inflammatory occlusion of the retinal veins and arteries and a peculiar thickening of the internal limiting membrane, but the inflammatory response was nonspecific in type. (Fig. 9). The left eye showed considerable fibrous tissue and new vessel formation extending into the vitreous, and extreme retinal perivascular sheathing. Her left vision gradually failed until June, 1945, when she was given x-ray therapy in a total dosage of 15,000 r to the left eye. The new vessels disappeared and there have been no fresh hemorrhages during the 14 months she has been followed since the x-ray therapy, but there are still many fine dilatations and tortuosities of the retinal venules and the perivascular streaking is unchanged. There was never any apparent uveitis in either eye. A patch test for OT 1:10,000, was negative. She, at one time, had an eosinophilia of from 6 to 8 per cent and a 2+ cephalin flocculation, but all other examinations (including sedimentation rate, complete blood studies and muscle biopsy) were negative.

Case IV. P. M., a 30 year old white male, had a history of at least two distinct vitreous hemorrhages in each eye during the preceding 5 years. Examination, August 10, 1945, showed in the right eye a little blood scattered through the lower vitreous with new-formed vessels extending far into the vitreous in three areas, and small aneurysmal dilatations in those new-formed vessels in the lower temporal part of the vitreous (Fig. 10). There was a small, apparently fresh vitreous hemorrhage which appeared to come from one of the new-formed vessels in the anterior vitreous (not shown in the illustration). The left eye had poor light projection and no red reflex. The right eye was given x-ray therapy (total dosage 15,000 r). The new-formed vessels disappeared completely but a few

## COLOR PLATE I

FIG. 1—Case I—Right eye four months preceding x-ray therapy. (November 28, 1944.)

FIG. 2—Case I—Right eye shortly before x-ray therapy, showing extension of vessels into anterior vitreous. (March 20, 1945.)

FIG. 3—Case I—Right eye after receiving 8,000 r, showing marked regression of vessels. (May 25, 1945.)

FIG. 4—Case I—Right eye after receiving the entire course of 13,000 r, showing complete disappearance of new-formed vessels. (July 13, 1945.)

FIG. 5—Case I—Left eye two months preceding x-ray therapy. (March 20, 1945.)

FIG. 6—Case I—Left eye immediately preceding x-ray therapy, showing extension of vessels into anterior vitreous. (May 25, 1945.)

## COLOR PLATE II

FIG. 7—Case I—Left eye after receiving 7,200 r, showing marked regression of vessels. (July 13, 1945.)

FIG. 10—Case IV—Right eye immediately preceding x-ray therapy. (August 10, 1945.)

FIG. 11—Case IV—Right eye after completion of therapy (15,000 r), showing complete collapse of new-formed vessels but persistence of tiny aneurysmal dilatations in lower temporal retinal venules. (December 7, 1945.)

FIG. 14—Case V—Left eye after end of first course of therapy (4,000 r), showing area of probable healed chorioretinitis and occlusion of terminal retinal vein, with two small fresh hemorrhages. (July 10, 1946.)

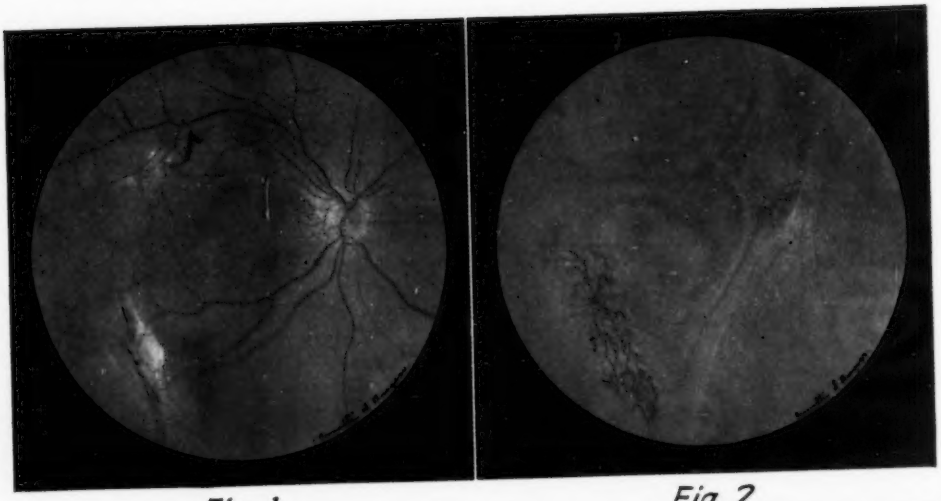


Fig. 1 Fig. 2

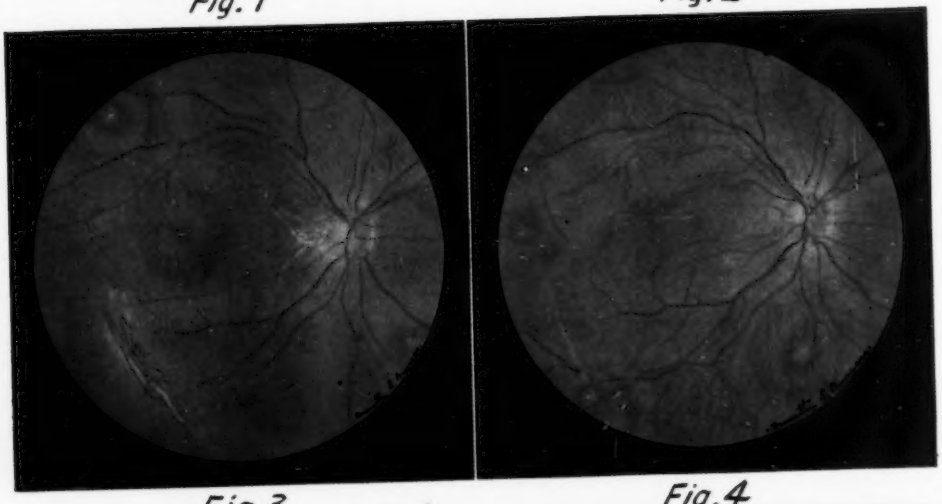


Fig. 3 Fig. 4

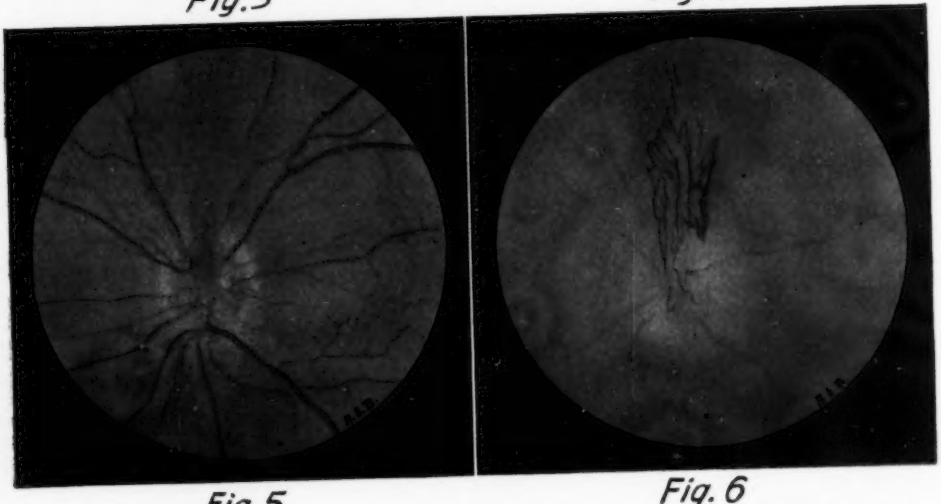


Fig. 5 Fig. 6

COLOR PLATE I

GUYTON AND REESE

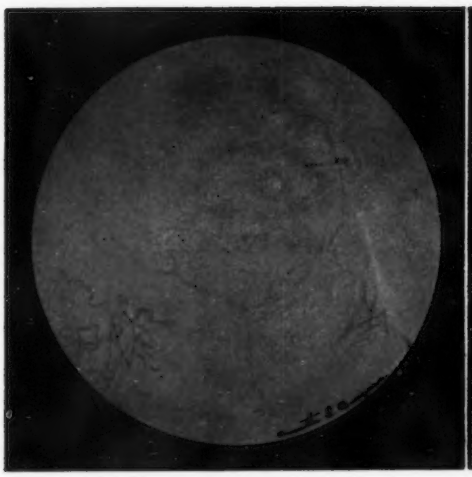


Fig. 10

Fig. 11

Fig. 7

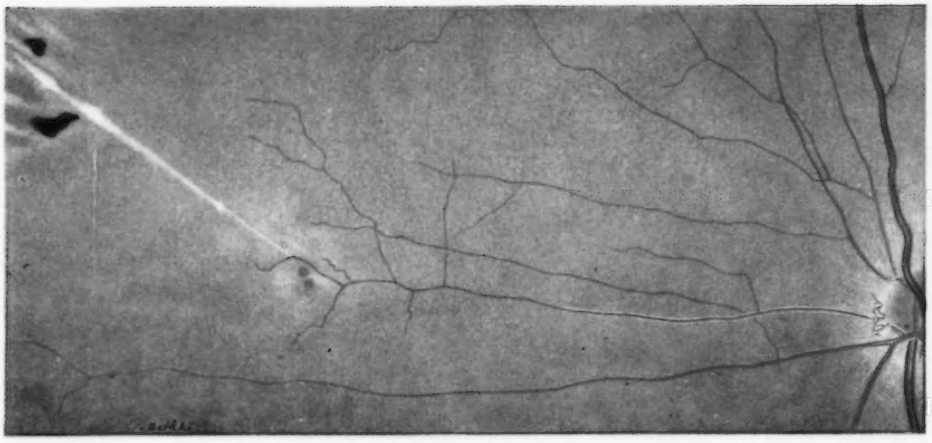


Fig. 14

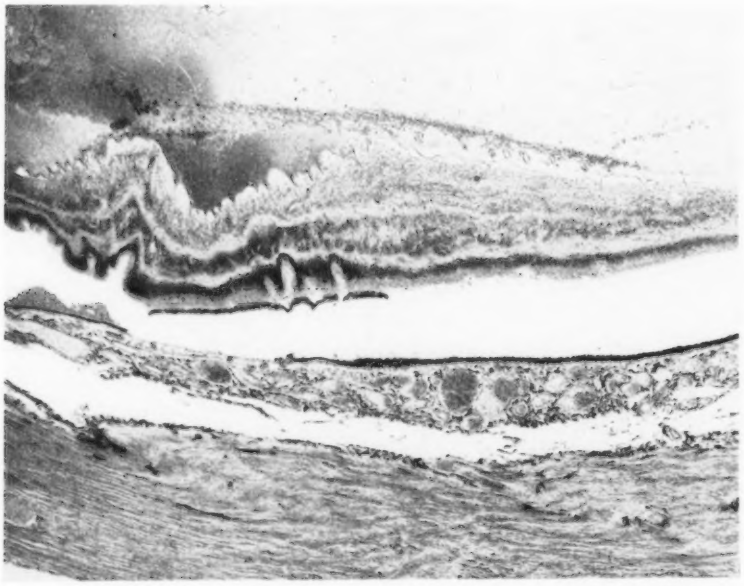


FIG. 8—Case III—Microphotograph from posterior segment of right eye, showing new-formed vessels and fibrous tissue extending from retina into vitreous hemorrhage.

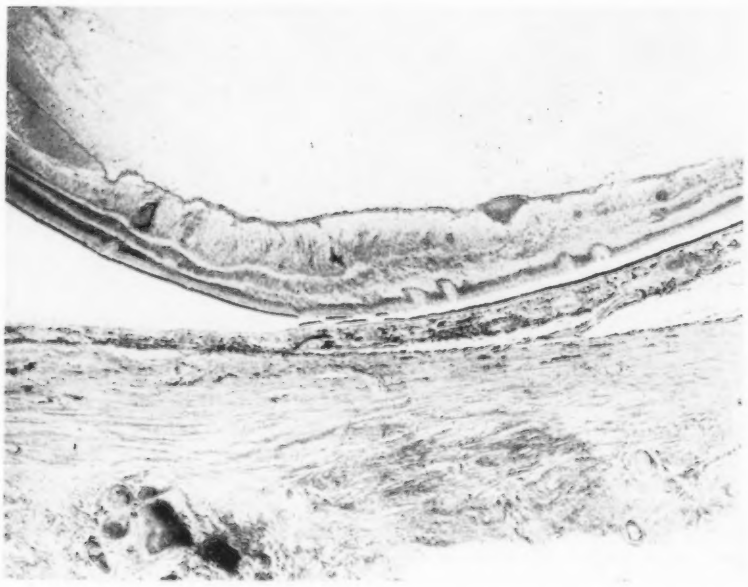


FIG. 9—Case III—Microphotograph from posterior segment of right eye, showing retinal periva-scular inflammation, inflammatory occlusion of retinal veins and peculiar thickening of internal limiting membrane.

tiny aneurysmal dilatations of retinal venules remained in the lower temporal quadrant of the retina (Fig. 11). There have been no recurrences of hemorrhages or of new-formed vessels during the year since he was treated. There was never any apparent uveitis in either

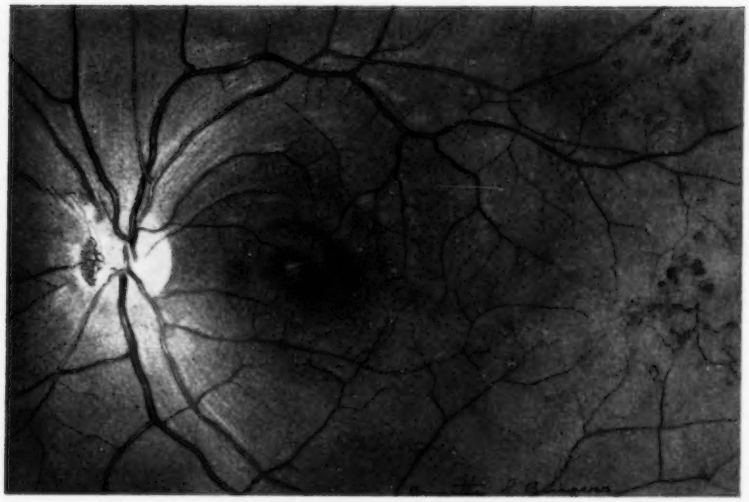


FIG. 12—Case V—Left eye immediately preceding x-ray therapy. (February 13, 1946.)

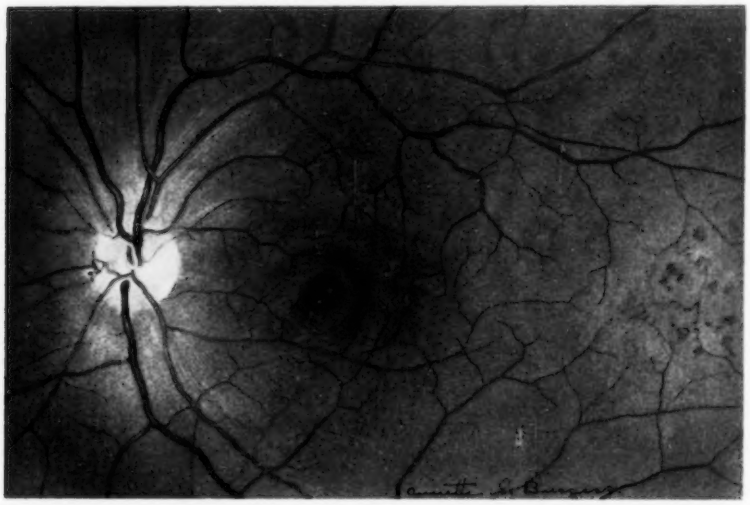


FIG. 13—Case V—Left eye after conclusion of first course therapy (4,000 r), showing partial collapse of new-formed vessels and persistence of aneurysmal dilatations of retinal venules in temporal periphery. (June 5, 1946.)

eye. He was sensitive to 1:1000 mgm. OT intracutaneously and biopsy of a cervical lymph node showed actual tubercle bacilli.

Case V. R. B., a 29 year old white male, began having many small recurrent retinal and vitreous hemorrhages in the right eye during July, 1945, and in the left eye two months later. Examination February 13, 1946, showed extensive fibrosis and new vessel formation in the right vitreous with partial retinal detachment. In the left eye there were scattered hemorrhages in the temporal retina and vitreous, with aneurysmal dilations of some retinal venules in this area, an area of retinal hemorrhage with obliteration of a terminal arteriole in the nasal periphery, and a sheet of new-formed vessels extending into the vitreous from the nasal side of the disc (Fig. 12). There was no evidence whatever of any vitreous or retinal hemorrhage anywhere close to the retinitis proliferans, which extended forward from the disc. X-ray therapy

FIG. 15—Case VII—Right eye immediately preceding x-ray therapy. (March 26, 1946.)

FIG. 16—Case VII—Right eye three months after completion of therapy (4,000 r), showing partial collapse of new-formed vessels. (July 31, 1946.)

was given in a total dosage of 3,500 r to the right eye and 4,000 r to the left eye. There was partial collapse of the new-formed vessels in each eye, only the larger trunks remaining patent, but litt'e or no decrease in fibrosis (Fig. 13). On July 1, 1946, the patient had symptoms of another hemorrhage in the left eye and was found to have two small retinal hemorrhages with a faint diffuse haze in the vitreous. (Fig. 14). He was given additional 2,000 r to the left eye, and has not been re-examined since that course was completed. The indistinct scar in the nasal periphery of the left eye was the only evidence of probable uveitis in either eye. He was sensitive to 1:1000 mgm. OT intracutaneously.

Case VI. R. R., a 26 year old white male, began having recurrent retinal and vitreous hemorrhages in both eyes 18 months before he was examined, and 6 months before examination he was said to have shown "organization" of hemorrhages with an area of chorioretinitis in the right eye but no uveitis in the left eye.

He was said to have shown a positive tuberculin reaction (dilution not known). On March 1, 1946, the vitreous of each eye contained so much blood nothing could be determined ophthalmoscopically except that there was retinitis proliferans with partial retinal detachment in each eye. X-ray therapy (4,000 r to each eye) was followed by gradual clearing of each vitreous. Three months later the left vision had improved from 20/200 to 20/25 and the left fundus could be seen fairly well, showing partial retinal detachment still present with moderate fibrosis on the surface of the retina but no new-formed vessels. The right fundus still could not be seen plainly.

Case VII. O. StJ., a 37 year old white female, began having many small recurrent vitreous hemorrhages in her left eye during November, 1945, and in her right eye on February 1, 1946. Examination March 15, 1946, showed small clumps of blood in the right vitreous with extensive retinitis proliferans, including many new-formed vessels and partial retinal detachment. (Fig. 15). The left eye showed a similar but more advanced picture, with massive retinitis proliferans and complete retinal detachment. X-ray therapy was given in a total dosage of 4,000 r to each eye. The smaller new-formed vessels in both eyes completely disappeared, but the larger ones only shrunk in size, remaining patent. (Fig. 16). There was some decrease in fibrosis of the vitreous and some flattening of the right retina, but the left retina remained completely detached. There have been no recurrences of hemorrhages or of new vessel formation during 5 months observation since the x-ray therapy. There was never any apparent uveitis. The patient was negative to 1 mgm. OT intracutaneously but showed hilar nodes in chest x-rays.

Case VIII. G. P., a 46 year old white male, had at least 20 vitreous hemorrhages in his left eye during the preceding 6 years and at least 10 in his right eye during the preceding 2 years. Examination July 10, 1946, showed considerable unabsorbed blood in each vitreous. Fundus details could not be seen except that massive retinitis proliferans was dimly visible in each eye. X-ray therapy was then given, 4,000 r to each eye. The vitreous is slowly clearing in each eye but fundus details are still not clearly visible. There has been no apparent uveitis. He has shown a positive reaction to 1:1000 mgm. OT intracutaneously and has a healed tuberculous lesion in the apex of his left lung.

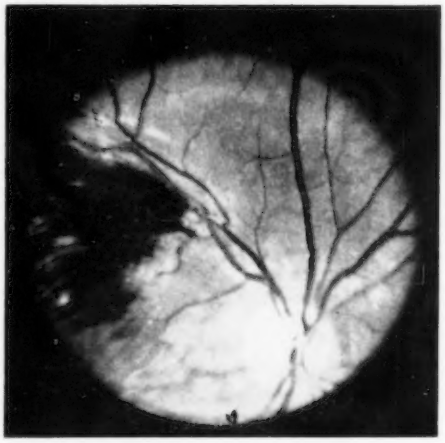


FIG. 17—Case X—Right eye one month after first hemorrhage, showing cork-screw anomaly in lower branch of superior temporal vein. (February, 1941.)

## B. Atypical Eales' disease.

Case IX. B. G., a 25 year old white female, developed numerous retinal and questionable vitreous hemorrhages in her right eye in January, 1946. Examination February 19, 1946, showed marked fibrosis with new-vessel formation on the right retina and extending slightly into the vitreous. There was apparent partial occlusion of several of the retinal veins. The left eye was entirely normal except for unusually large veins. She received 4,000 r to the right eye beginning April 13, 1946. The new-formed vessels partially disappeared and there have been no additional hemorrhages in the five months since the eye was treated. She had diabetes for eighteen months preceding therapy but no diabetic retinopathy. She had no apparent uveitis and tuberculin sensitivity was not determined.

Case X. A. H., a 40 year old white male, had developed a massive retinal and vitreous hemorrhage in his right eye while lifting a heavy weight 5 years previously. The vitreous hemorrhage had cleared quickly and a fundus photograph taken one month later (Fig. 17) showed an extensive retinal hemorrhage along the lower branch of the superior temporal vein, occurring distal to a point where this vein coiled in corkscrew fashion around the superior temporal artery. The retinal hemorrhage had gradually cleared, with some fibrosis, and he had had no further symptoms until a second large hemorrhage occurred December 1, 1945—this time spontaneously. Examination December 18, 1945, showed the left eye normal except for

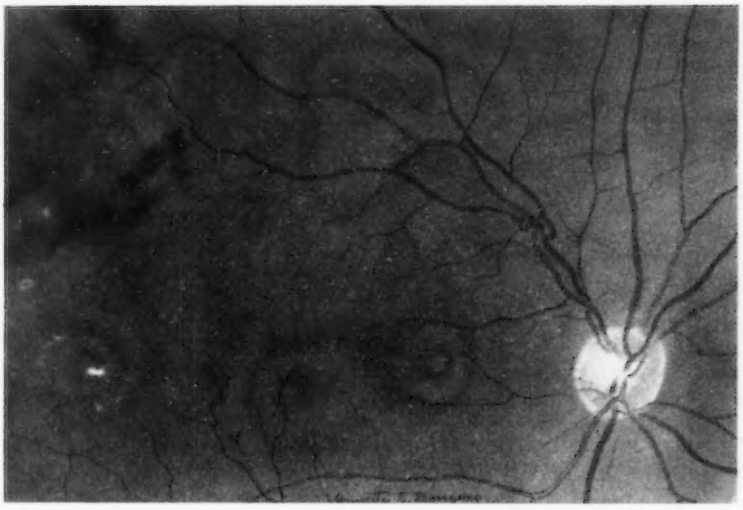


FIG. 18—Case X—Right eye immediately preceding x-ray therapy. Note the varicosities and tiny aneurysmal dilatations in terminal venules of lower temporal vein as well as of upper vein. (December 18, 1945.)

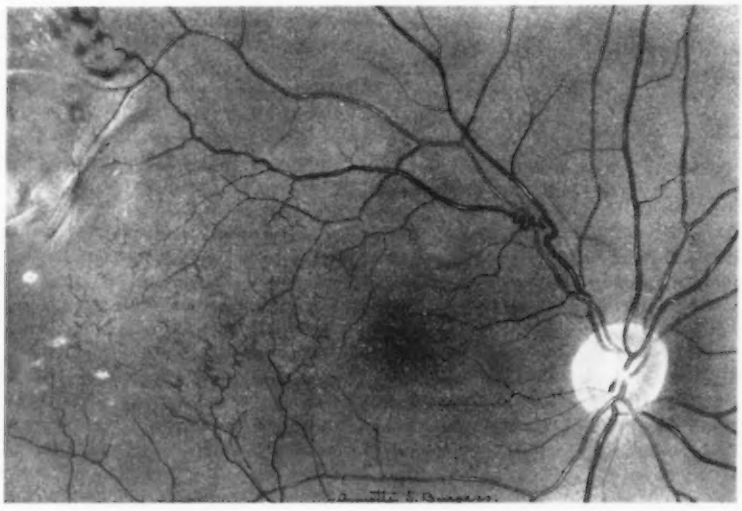


FIG. 19—Case X—Right eye after completion of therapy (6,000 r), showing regression of new-formed vessels but some persistence of varicosities in retinal venules. (February 12, 1946.)

amblyopia exanopsia. In the right eye there was a massive retinal and slight vitreous hemorrhage along the distribution of the lower branch of the superior temporal vein, a small branch of which was obliterated, with some organization of the hemorrhage (including new-formed vessels) in some places. There was also some white serous exudate in the lower vitreous. Throughout the involved area of retina but extending also into areas supplied by the lower temporal vein, there were cork-screw irregularities and tiny aneurysmal dilations of the fine retinal venules (Fig. 18). These small varicosities were still present after x-ray therapy (6,000 r) but were less pronounced than before treatment (Fig. 19). There was slight decrease in fibrosis following x-ray therapy and the new-formed vessels disappeared. He had

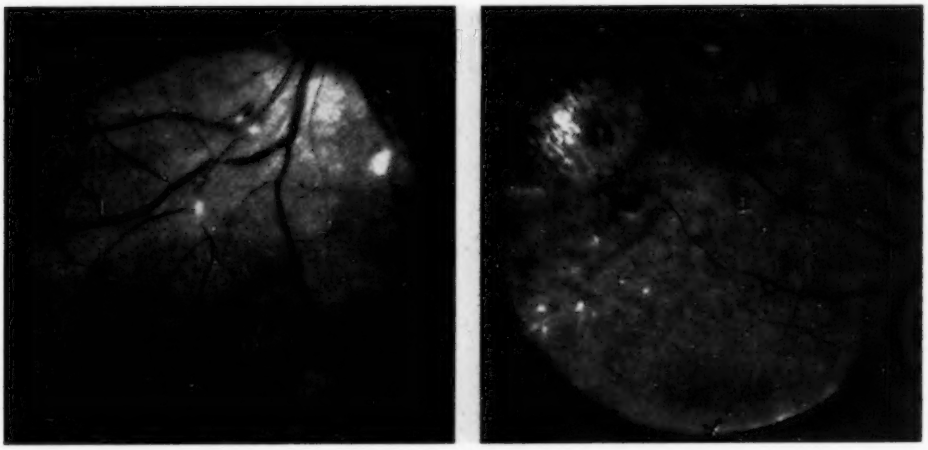


FIG. 20 (a and b)—Case X—Right eye 7 months later, showing new retinal hemorrhages and cotton wool exudates, and small plexus of dilated capillaries.

symptoms of two small hemorrhages during June, 1946, and examination October 1, 1946, showed several tiny retinal hemorrhages and cotton-wool exudates scattered about the posterior fundus, partial absorption of the serous exudate in the lower vitreous, and a peculiar localized plexus of dilated capillaries at one point along the remaining branch of the originally involved vein. (Fig. 20). He is now receiving 3,000 r additional x-ray therapy. There was never any evidence of uveitis. He was sensitive to 1:1000 mgm. OT intracutaneously.

Case XI. L.P., a 42 year old white male, had had numerous retinal and small vitreous hemorrhages in the left eye during the preceding 14 months and in the right eye during the preceding six months. He had had subjective exacerbations in both eyes approximately 48 hours after each of three intracutaneous tuberculin injections. Examination July 19, 1946, showed in the right eye slight vitreous hemorrhage and a peripheral medium-sized retinal hemorrhage with early organization, with dilation of a retinal vein just at the site of its emergence from the area of hemorrhage. In the left eye there were more vitreous hemorrhages and clumps of white serous exudate in the lower vitreous, with streaky retinal hemorrhages undergoing absorption but without organization in the temporal periphery. A retinal vein supplying this latter area had two short stretches of doubling of the channel at one place, with a trace of adjacent absorbing retinal hemorrhage suggesting occlusion and recanalization of the vein at this point. There was also a small aneurysmal dilatation at one point in a branch of the superior nasal vein, but no adjacent hemorrhage. A total of 4,000 r was given each eye and he has not been examined since the conclusion of this therapy (August 10, 1946).

Case XII. S. A., a 34 year old white female with a history of many vitreous hemorrhages in both eyes during the preceding 6 years, was first examined March 5, 1943. The right eye was blind, with evidence of healed anterior uveitis and a dense complicated cataract. The left eye showed a normal anterior segment, some vitreous hemorrhage, and scattered retinal hemorrhages with some fibrosis. Left vision at that time was 20/70. During the next three years she had several recurrent vitreous hemorrhages in the left eye and on June 9, 1946, she developed an acute anterior uveitis with a large hypopyon (containing some hemorrhage) and temporary loss of light perception. The anterior uveitis cleared within a few days and she was found to have moderate vitreous hemorrhage. X-ray therapy (3,500 r) was given the left eye. The vitreous hemorrhage gradually cleared within a month and she was then seen to have indistinct white areas (apparently a mixture of atrophy and of fibrosis) scattered throughout the retina, with a pale disc and vision still limited to light perception. She had aphthous lesions of the mucous membranes, a deficiency of plasma ascorbic acid, a slightly increased capillary fragility, a probable tuberculous infection of a kidney, and sensitivity to 1:1000 mgm. OT intracutaneously.

## C. Diabetic retinitis proliferans

Case XIII. M. B., a 53 year old colored female, was found to have diabetes in 1943. At that time, she had many small hemorrhages and

FIG. 21—Case XIII—Right eye immediately preceding x-ray therapy. (April 1, 1946.)

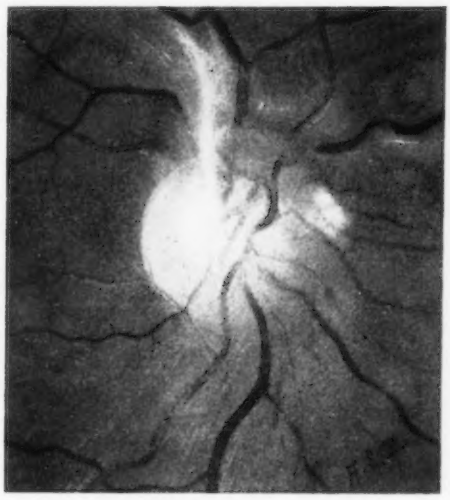


FIG. 22—Case XIII—Right eye after completion of therapy (4,000 r), showing disappearance of new-formed vessels. (June 4, 1946.)

irregular waxy exudates in each retina. In May, 1945, new-formed vessels and fibrous tissue began growing into the vitreous of the right eye from the upper temporal retina and from the disc. This retinitis proliferans progressed and on April 1, 1946, (Fig. 21) x-ray therapy (4,000 r) was begun to the right eye. The new-formed vessels collapsed completely within 7 weeks and the fibrosis became slightly less. (Fig. 22). There have since been new retinal hemorrhages from time to time, but there has been no recurrence of retinitis proliferans during the five months since x-ray therapy.

Case XIV. I. W., a 42 year old white male, had a history of diabetes of 3 years duration and of blurred vision for 9 months. Examination March 27, 1946, showed multiple retinal hemorrhages and hard white exudates and considerable retinitis proliferans, including many new-formed vessels, in each eye. X-ray therapy (4,000 r to each eye) resulted in marked diminution but not complete collapse of the new-formed vessels, with temporary improvement of vision. However, he returned 5 months later with fresh vitreous hemorrhages and greatly reduced vision in each eye.

## SUMMARY OF MATERIAL

The ocular diseases in these 14 patients did not constitute a single entity, but all the 22 eyes treated had in common retinal and vitreous hemorrhages with progressive formation of new vessels and fibrous tissue on the surface of the disc or retina and extending more or less into the vitreous. In every case the new-formed vessels and fibrosis could be called "retinitis proliferans," provided this term be used in a purely descriptive sense.

The ocular disease was classified as "typical" Eales' disease in 8 patients. This group consisted of 6 male and 2 female young adults with bilateral recurrent retinal and vitreous hemorrhages of obscure origin and with retinitis proliferans. One patient had evidence of old healed bilateral anterior uveitis, 3 others had a probable unilateral choroiditis at some time before they were examined and, in a fifth patient, an eye enucleated without having had x-ray therapy showed a nonspecific inflammatory occlusion of the retinal vessels. Tuberculin reactions were positive in 6 of these 8 patients, 5 of them giving a positive reaction to the small dose of 1/1000 mgm. OT intracutaneously and the sixth having shown a positive reaction to an unstated quantity elsewhere. One of the patients in this group was insensitive to 1 mgm. OT

intracutaneously but x-ray of the chest showed enlarged hilar nodes. The final patient did not react to a patch test with 1:10,000 dilution OT and the test was not carried further. The occupations of these patients were varied, consisting of business man, lawyer, seaman, clerk, housewife, and army officer.

The ocular disease was classified as "atypical" Eales' disease in 4 patients. In one of these patients the only affected eye showed probable partial occlusion of several retinal veins, which may have initiated the process. A second patient had hemorrhages recurring at first only in a localized area of the only involved eye and showed an anomalous, sharply localized narrowing of the retinal vein supplying this area. A third patient with bilateral involvement showed two localized dilatations of retinal veins and a probable localized occlusion and recanalization of another small vessel. The fourth patient had classical aphthous uveitis.

The ocular disease was classified as diabetic retinitis proliferans in 2 patients. Both patients had long standing diabetes with bilateral diabetic retinopathy, including retinal hemorrhages and exudates but little evidence of vitreous hemorrhages except for faint vitreous haze. Typical retinitis proliferans had developed in the three eyes which were treated.

## SUMMARY OF RESULTS

The first and most striking effect of x-ray therapy was a diminution in the new-formed vessels. The smaller vessels began shrinking in size within two to three weeks after therapy was begun and invariably disappeared from ophthalmoscopic visibility within 4 to 8 weeks, even if the total dosage was only 3,500 r. If a dosage of not more than 4,000 r was given, the larger new-formed vessels usually showed some shrinkage in calibre, but remained patent indefinitely. However, in those 6 eyes where the total dosage was 10,000 r or more, even the largest new vessels completely disappeared from ophthalmoscopic visibility. There has as yet been no definite recurrence of new-formed vessels in any of the 22 eyes treated.

Regression of fibrosis was less striking than the regression of vessels. If the fibrosis was scanty, almost translucent, before therapy began and if 10,000 r or more was given, the regression of fibrous tissue was marked. However, dense fibrosis of long duration was reduced only slightly or moderately even by heavy dosage, and dosages of only 4,000 r had little effect even on scanty fibrosis.

The tiny dilatations of retinal venules in 2 cases appeared to be less prominent but still present after large dosages of x-ray, and the varicosities of the tiny retinal venules showed no perceptible change.

In the group of 8 patients with "typical" Eales' disease there has been only one recurrent hemorrhage. It consisted of two small retinal hemorrhages and faint vitreous haze in an eye which had received only 4,000 r and did not show complete collapse of the larger new-formed vessels. This cessation of recurrent hemorrhages appeared to be more than a coincidence in 3 of these patients, in whom many small vitreous hemorrhages had appeared in each eye at intervals of 1 to 4 weeks right up to the beginning of therapy. However, this cessation of hemorrhages may well have been the result of breaking up a "vicious cycle" (see COMMENT) rather than an effect on factors initially responsible for the ocular diseases. It must be emphasized that these results are only immediate, short term results, since only 4 patients have been followed longer than 10 months. As we do not know the primary cause of the retinal and vitreous hemorrhages we cannot report at pres

TABLE I

| Diagnostic Classification | Case Number | Age | Sex | Eye | CONDITION PRECEDING X-RAY THERAPY | | | | | | | X-RAY THERAPY (roentgen units in air) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Vision with Correction | Duration of Symptoms | History of Vitreous Hemorrhages | New-formed Vessels in Vitreous | Fibrosis Extending into Vitreous | Retinal Detachment | Other Comment | Temporal Portal | Nasal Portal | Tot |
| TYPICAL EALES DISEASE | I. | 37 | M | R | 20/15 −1 | 10 mos. | 6 small | ++++ | + — | 0 | No evident uveitis. | 8000 | 5000 | 130 |
| | | | | L | 20/15 −2 | 12 mos. | 13 small | ++++ | + — | 0 | No evident uveitis. | 8000 | 6000 | 140 |
| | II. | 40 | M | R | 20/70 | 11 mos. | 1 massive ? other | +++ | +++ | ++ | Anterior uveitis 5 mos. before vitreous hemorrhages. | 7000 | 5000 | 120 |
| | | | | L | 1/200 | 6 mos. | 1 massive ? others | Faint | red | reflex | Anterior uveitis 11 mos. before vitreous hemorrhages. | 5000 | 5000 | 100 |
| | III | 24 | F | R | NLP. | 2 yrs. | several small | ++++ | +++ | + | Enucleated because of absolute glaucoma with new-formed vessels on iris. | | | |
| | | | | L | 20/100 +1 | 2 yrs. | several small | ++++ | +++ | + | Extreme periarterial streaking throughout fundus: irregular dilation and beading of retinal veins. | 10000 | 5000 | 150 |
| | IV. | 30 | M | R | 20/20 −3 | 5 yrs. | 2 moderate | +++ | ++ | 0 | No evident uveitis. | 10000 | 5000 | 150 |
| | | | | L | L.P. | 5 yrs. | 1 small and 1 massive | no | red | reflex | Poor light projection; X-ray therapy not believed justifiable. | | | |
| | V. | 29 | M | R | 3/200 | 7 mos. | many small | +++ | ++++ | + | No evident uveitis. | 2500 | 1000 | 35 |
| | | | | L | 20/15 | 5 mos. | many small | ++ | + | 0 | Marked streaking along one artery leading to area of possible chorioretinitis. | 2500 | 1500 | 40 |
| | VI | 26 | M | R | 20/400 | 17 mos. | many moderate | ? | ++ | ++ | Definite chorioretinitis noted in army while hemorrhages were developing. | 4000 | | 40 |
| | | | | L | 20/200 | 20 mos. | many moderate | ? | ++ | ++ | No evident uveitis. | 4000 | | 40 |
| | VII. | 37 | F | R | 20/70 | 1½ mos. | many small | +++ | +++ | ++ | No evident uveitis. | 2000 | 2000 | 40 |
| | | | | L | 1/200 | 5 mos. | many small | +++ | ++++ | ++++ | No evident uveitis. | 2000 | 2000 | 40 |
| | VIII. | 46 | M | R | H.M. | 2 yrs. | 10+ small | ? | ++++ | ? | Too much vitreous hemorrhage to see anything except marked fibrosis in vitreous. | 4000 | | 40 |
| | | | | L | H.M. | 6 yrs. | 20+ small | ? | ++++ | ? | Too much vitreous hemorrhage to see anything except marked fibrosis in vitreous. | 4000 | | 40 |
| ATYPICAL EALES DISEASE | IX. | 25 | F | R | 20/100 | 1 mo. | Questionable | +++ | +++ | 0 | Several retinal veins partially occluded } Diabetes for 18 mos. but no Diabetic Retinopathy | 4000 | | 40 |
| | | | | L | 20/15 | | 0 | 0 | 0 | 0 | Normal except veins appear large } | | | |
| | X. | 40 | M | R | 20/15 | 5 yrs. | 2 medium | + | + | 0 | Dilatation of venules in temporal periphery. Retinal hemorrhages only in region supplied by a branch of superior temporal vein. White serous exudate in lower vitreous. | 3000 | 3000 | 60 |
| | | | | L | 5/200 | — | 0 | 0 | 0 | 0 | Amblyopia exanopsia from anisometropia Eye otherwise normal | | | |
| | XI. | 42 | M | R | 20/70 +1 | 6 mos. | 10 small | 0 | + | 0 | Strabismus with partial amblyopia in right eye. Both eyes showed some irregular dilatation in retinal venules; organization of retinal hemorrhages and in the left vitreous was some white serous exudate. | 4000 | | 40 |
| | | | | L | 20/20 −1 | 14 mos. | 20 small | 0 | 0 | 0 | | 4000 | | 40 |
| | XII. | 34 | F | R | NLP. | 9 yrs. | many | no | red | reflex | Aphthous uveitis with recurrent attacks. | | | |
| | | | | L | LP. | 9 yrs. | many | + — | + | + — | Aphthous uveitis with recurrent attacks. | 1800 | 1700 | 35 |
| DIABETIC RETINITIS PROLIFERANS | XIII. | 53 | F | R | 20/40 | 3 yrs. | Questionable | ++ | ++ | 0 | Severe diabetic retinopathy. | 2000 | 2000 | 40 |
| | | | | L | 20/40 | 3 yrs. | 0 | 0 | 0 | 0 | Diabetic retinopathy only—no new formed vessels. | | | |
| | XIV. | 42 | M | R | 20/200 | 1 yr. | Questionable | ++ | + — | 0 | Severe diabetic retinopathy. | 4000 | | 40 |
| | | | | L | 20/200 | 1 yr. | Questionable | +++ | +++ | 0 | Severe diabetic retinopathy. | 4000 | | 40 |

TABLE I

| RECEDING X-RAY THERAPY | | | | X-RAY THERAPY (roentgen units in air) | | | Follo up |
|---|---|---|---|---|---|---|---|
| med in us | Fibrosis Extending into Vitreous | Retinal Detachment | Other Comment | Temporal Portal | Nasal Portal | Total | Peri |
| + | + — | 0 | No evident uveitis. | 8000 | 5000 | 13000 | 18 mos |
| + | + — | 0 | No evident uveitis. | 8000 | 6000 | 14000 | 16 mos |
| + | + + + | + + | Anterior uveitis 5 mos. before vitreous hemorrhages. | 7000 | 5000 | 12000 | 14 mos |
| t | red | reflex | Anterior uveitis 11 mos. before vitreous hemorrhages. | 5000 | 5000 | 10000 | 14 mos |
| + | + + + | + | Enucleated because of absolute glaucoma with new-formed vessels on iris. | | | | Sections o occlusion |
| + | + + + | + | Extreme periarterial streaking throughout fundus: irregular dilation and beading of retinal veins. | 10000 | 5000 | 15000 | 14 mos |
| + | + + | 0 | No evident uveitis. | 10000 | 5000 | 15000 | 12 mos |
| | red | reflex | Poor light projection; X-ray therapy not believed justifiable. | | | | |
| + | + + + + | + | No evident uveitis. | 2500 | 1000 | 3500 | 4 mos |
| | + | 0 | Marked streaking along one artery leading to area of possible chorioretinitis. | 2500 | 1500 | 4000 | 4 mos |
| | + + | + + | Definite chorioretinitis noted in army while hemorrhages were developing. | 4000 | | 4000 | 3 mos |
| | + + | + + | No evident uveitis. | 4000 | | 4000 | 3 mos |
| + | + + + | + + | No evident uveitis. | 2000 | 2000 | 4000 | 5 mos |
| + | + + + + | + + + + | No evident uveitis. | 2000 | 2000 | 4000 | 5 mos |
| | + + + + | ? | Too much vitreous hemorrhage to see anything except marked fibrosis in vitreous. | 4000 | | 4000 | 2 mos |
| | + + + + | ? | Too much vitreous hemorrhage to see anything except marked fibrosis in vitreous. | 4000 | | 4000 | 2 mos |
| + | + + + | 0 | Several retinal veins partially occluded — Diabetes for 18 mos. but no Diabetic Retinopathy | 4000 | | 4000 | 5 mos |
| | 0 | 0 | Normal except veins appear large — Diabetes for 18 mos. but no Diabetic Retinopathy | | | | |
| | + | 0 | Dilatation of venules in temporal periphery. Retinal hemorrhages only in region supplied by a branch of superior temporal vein. White serous exudate in lower vitreous. | 3000 | 3000 | 6000 | 8 mos |
| | 0 | 0 | Amblyopia exanopsia from anisometropia Eye otherwise normal | | | | |
| | + | 0 | Strabismus with partial amblyopia in right eye. Both eyes showed some irregular dilatation in retinal venules; organization of retinal hemorrhages and in the left vitreous was some white serous exudate | 4000 | | 4000 | 3 week |
| | 0 | 0 | | 4000 | | 4000 | 3 week |
| | red | reflex | Aphthous uveitis with recurrent attacks. | | | | |
| | + | + — | Aphthous uveitis with recurrent attacks. | 1800 | 1700 | 3500 | 1 mo |
| - | + + | 0 | Severe diabetic retinopathy. | 2000 | 2000 | 4000 | 5 mos |
| | 0 | 0 | Diabetic retinopathy only—no new formed vessels. | | | | |
| - | + — | 0 | Severe diabetic retinopathy. | 4000 | | 4000 | 6 mos |
| + | + + + | 0 | Severe diabetic retinopathy. | 4000 | | 4000 | 6 mos |

| | Follow-up | RESULTS | | | | |
|---|---|---|---|---|---|---|
| | Period | Final Vision | Subsequent Vitreous Hemorrhages | Diminution in new-formed Vessels | Diminution In Fibrosis | OTHER COMMENT |
| | 18 mos. | 20/15 | 0 | complete | marked | |
| | 16 mos. | 20/15 | 0 | complete | marked | Visible area of healed choroiditis. |
| | 14 mos. | 20/25 | 0 | complete | moderate | Retinal detachment less. |
| | 14 mos. | L.P. | 0 | Still | only faint red | reflex. |
| Sections of posterior segment showed retinitis proliferans, non-specific inflammatory occlusion of retinal vessels, and peculiar thickening of internal limiting membrane. | | | | | | |
| | 14 mos. | 20/30 | 0 | complete | marked | |
| | 12 mos. | 20/20—2 | 0 | complete | slight | |
| | | | | | | |
| | 1 mos. | 5/200 | 0 | moderate | none | |
| | 4 mos. | 20/15 | 1 small | moderate | Questionable | Given 2000 r additional following the small recurrent retinal hemorrhage. |
| | 3 mos. | 20/200 | 0 | ? | ? | Fundus not seen well enough preceding radiation to judge changes accurately. |
| | 3 mos. | 20/25 | 0 | ? | ? | |
| | 5 mos. | 20/40 | 0 | moderate | slight | Retinal detachment only slightly less. |
| | 5 mos. | 3/200 | 0 | moderate | slight | Retinal detachment still ++++ |
| | 2 mos. | 2/200 | 0 | ? | ? | Still too much vitreous hemorrhage to see details with ophthalmoscope. |
| | 2 mos. | 2/200 | 0 | ? | ? | |
| | 5 mos. | 20/200 | 0 | moderate | Questionable | |
| | | | | | | |
| | 8 mos. | 20/15 | 2 small | complete | Questionable | Dilatation of venules less but not completely obliterated. White serous exudate in lower vitreous partly absorbed. Small fresh hemorrhages and cotton wool exudates around disc. |
| | | | | | | |
| | 3 weeks | Insufficient follow-up period to judge results. | | | | |
| | 3 weeks | | | | | |
| | | | | | | |
| | 1 mo. | L.P. | 0 | Questionable | Questionable | Retinal degeneration and optic atrophy. |
| | 5 mos. | 20/50 | 0 | complete | slight | Retinal hemorrhages continued to appear. |
| | | | | | | |
| | 6 mos. | 1/200 | + | marked | Questionable | Initial improvement but fresh vitreous hemorrhages when last examined. |
| | 6 mos. | 1/200 | + | marked | Questionable | |

ent on the effects of the treatment in this regard, nor can we yet determine the permanency of the immediate effects.

In the group of 4 patients with "atypical" Eales' disease, there has as yet been a recurrent hemorrhage only in the patient with an anomalous localized constriction of a retinal vein. However, there has not yet been an appreciable follow-up period for the other three.

In the group of 2 patients with diabetic retinitis proliferans, fresh retinal hemorrhages have continued to appear, and one patient has developed fresh vitreous hemorrhages five months after therapy, even though the new-formed vessels were markedly diminished. It seems unlikely that x-ray therapy could result in permanent benefit for patients in this category until an effective treatment for diabetic retinopathy itself has been discovered.

## COMMENT

Most retinal and vitreous hemorrhages gradually absorb without inducing retinitis proliferans, but sometimes, especially with recurrent hemorrhages, there is proliferation of new vessels and of fibrous tissue on the surface of the retina or disc and into the vitreous. Regardless of the primary cause of the hemorrhages, it seems probable this stimulation of new tissue formation is often a direct result of some fraction of blood in the vitreous, although the components of blood which may be responsible for such a proliferative reaction are not known.

The new-formed vessels extending into the vitreous have very thin walls and histologically are surrounded by a zone of eosin-staining material which appears to be homogeneous transudate and in which delicate sheets of fibroblasts are growing. The thin walls of these vessels can certainly break with actual seepage of blood into the vitreous, as we have observed in 2 cases. Also, it seems theoretically possible that a transudate of serum from the thin-walled, new-formed vessels themselves may account for continued proliferation of new vessels even after the primary retinal and vitreous hemorrhages have cleared. Thus, in certain instances a vicious cycle may be established, the new thin-walled vessels in the vitreous stimulating their own progression by actual hemorrhage or possibly by simple transudate of serum. Our use of x-ray therapy has been directed primarily toward the destruction of new-formed vessels in the vitreous. If the vicious cycle described above plays as large a role as we believe in progressive retinitis proliferans, the results of x-ray therapy may prove to be permanent in those cases where the primary cause of the hemorrhages is self limited rather than of indefinite duration. This happy result cannot be hoped for in diabetes, but may be in many cases of Eales' disease.

The varicosities and tiny dilatations in retinal venules described in 3 of these cases all occurred in areas where retinal hemorrhages had absorbed. For many years these have been known to result at times from retinal hemorrhage and to remain permanently after absorption of the hemorrhage. It seems unlikely they induce more hemorrhages or retinitis proliferans. The vessels are not newly formed and they show little if any shrinkage as a result of even massive x-ray therapy.

We have purposely avoided the clinical terms, periphlebitis and periarteritis, because while these terms denote actual inflammation around vessels, they are commonly used to describe perivascular streaking. As a matter of fact, perivascular streaking is not necessarily indicative of an inflammatory process, but may merely represent a sequela of absorption of large retinal hemorrhages in the area supplied by the vessels.

A discussion of the etiologies of Eales' disease is outside the scope of this paper. Eales' disease may (reportedly) be caused by tuberculosis, endocrine disturbance (such as menstruation) thrombo-angiitis obliterans, hemophilia, capillary fragility, thrombocytopenic purpura, ocular hypotony, low blood calcium, high blood pressure, low blood pressure, foci of infection, and other causes. In any event, we are unable even to give a concise definition of Eales' disease, let alone evaluate the primary causes and explain the facts that 80 per cent of the cases are in males and that the disease is usually bilateral.

SUMMARY

Intensive x-ray therapy to the posterior ocular segment has been given during the past 18 months in a series of patients with ocular diseases characterized by retinal and vitreous hemorrhages and by new-formed blood vessels and fibrous tissue extending from the retina or disc into the vitreous (retinitis proliferans).

A total of 22 eyes in 14 patients have been treated. The ocular diseases were classified as typical Eales' disease in 8 patients, atypical Eales' disease in 4 patients, and diabetic retinitis proliferans in 2 patients.

The irradiation technic devised by Martin and Reese for the treatment of retinoblastoma was utilized to avoid damage to the anterior ocular segment. Total dosages of 3,500 to 15,000 r were given each eye treated. Dosages around 6,000 r are probably optimal.

Depending on the dosage of x-ray given, there was moderate to complete collapse of new-formed vessels and variable regression of fibrosis. There has been only one recurrent hemorrhage following treatment in the 8 cases of typical Eales' disease, an effect which has appeared to be of immediate significance, but the permanency of these immediate results is not established because insufficient time has elapsed since the patients were treated. In the 2 cases of diabetic retinitis proliferans hemorrhages have continued to appear following treatment, and permanent beneficial results can hardly be expected.

## REFERENCE

1. Martin, H. E. and Reese, A. B.: Treatment of retinal gliomas by the fractionated or divided dose principle of roentgen radiation. Arch. Ophth. 16:733-761 (Nov.) 1936.

Martin, H. E. and Reese, A. B.: Treatment of retinoblastoma (retinal glioma) surgically and by irradiation. Arch. Ophth. 27:40-72 (Jan.) 1942.

Martin, H. E. and Reese, A. B.: Treatment of bilateral retinoblastoma (retinal glioma) surgically and by irradiation. Arch. Ophth. 33:429-439 (June) 1945.

## DISCUSSION

MOACYR E. ALVARO, M.D., Sao Paulo, Brazil: The x-ray treatment of hemorrhagic retinal alterations dates back to the end of the second decade of this century and curiously enough it was by the analogy of good results obtained in the treatment of hemorrhages in myoma uteri, that Hessberg, in 1919, tried the x-ray treatment for hemorrhagic glaucoma.[1] In the following years the x-ray treatment for hemorrhagic conditions of the fundus has been used with increasing favor. However there appears to be still considerable difference of opinion regarding the results. A substantial group, the great majority of those who have reported cases of retinal hemorrhages treated with x-rays, seem to approve of the method advocating the use of small doses, totalling 600 r and even less. Thus, Basile, Saul, Zingale, Loewenstein, Reise, Schmerer, Hessberg, Di Marzio, Jendralski, and others have reported good results. Another group, however, is less enthusiastic. Cordes favors the x-ray therapy only for tuberculous periphlebitis in young individuals and discourages it for vascular occlusion due to sclerosis. Schnyder and Foster caution about the possibility of producing inflammatory conditions which in turn might cause retinal detachment. Von Grosz cautioned about the possibility of hemorrhages caused by the x-ray therapy and Stock, Mylius, and Lorey reported cases in which this actually happened, but these were cases in which invariably larger doses had been given.

According to the generally accepted concensus, the action of the x-rays is always bio-negative. The so-called stimulating action is apparently caused indirectly through catabolic substances originated from the primary destructive action of the x-rays on the tissues, through changes in the globulin-albumin relationship and changes in the pH.

An electron can be depicted as a small bullet of one millimeter in diameter revolving in a sphere, the atom, about fifty to one hundred meters in diameter; the proteic molecule has from 800 to 1500 atoms, and a cell from one to one hundred thousand million molecules. The impact of one of those electrons upon the minute part of a cell that stops it (absorbs the rays) may cause several reactions. According to Dessauer, it will cause heat which will coagulate some of the albumin of the cell. To others, it causes changes in the electric charge of the colloids. Be it one or the other, or still some other phenomenon, the fact remains that there will always be changes, as the result of the impact; changes in the ion concentration of mineral salts, changes in the cholesterin contents, in the viscosity of the plasma, and in the permeability of cellular membranes.

Sensitivity to x-rays varies considerably from cell to cell in the different biological species and even in the same species, according to the type of cell. The more differentiated the cell, the less sensitive it is. The action is always directly proportional to the activity of the cell; newly-formed tissue being always more receptive.

White blood cells, except in vitro having nuclei, are naturally more sensitive to x-ray than red blood cells. Lymphocytes are the most sensitive of all, sensitivity decreasing in the following order: monocytes, eosinophiles, myelocytes, and neutrophiles.

The effect of x-rays upon the blood vessels varies with the amount of radiation used. In the retinal blood vessels histologically studied by Hoffmann in enucleated eyes which had been given large doses of x-ray for treatment of malignant tumors, there appeared to be considerable dilatation of both arteries and veins, with changes in the endothelial cells where the nuclei showed marked tumefaction; moreover fibroblasts in great numbers could be seen the same as intense proliferation of the intima and even consequent closing of the lumen of the vessels. When smaller doses are used there is an immediate dilatation of the capillaries as it has been proved by David and Gabriel in their experiments. In order to explain the vasodilatation, several theories have been developed: 1—According to Best, Dale and Dudley, x-rays would be able to liberate histidine and similar substances from the tissues. Histidine would then be transformed into histamine, which in turn would, once in the blood circulation, cause the capillaries to be dilated. 2—According to Ellinger, x-rays would act directly upon the nuclei of the endothelial cells, which appear to be very sensitive to these rays. This would explain the changes found in the intima, even in larger blood vessels. 3—According to the experiment of Sunder Plassmann, nervous terminations, in contrast to the other nervous tissue, are very sensitive to x-rays. In his experiments he was able to show that the nervous terminations which have normally the shape of a reticulum after radiation with small doses of x-ray, had the shape of a conglomerat. Thus, the x-rays would influence directly the terminal vasomotor branches as it has been shown by Jungenburg and Schlepakow in the x-ray treatment of the thyroid gland. There is no reason why the x-rays could not have the same effect upon the nervous terminations of the vasomotor nerves in the vessels of the retina. Even with small doses there seems always to be an increase in permeability of the endothelium of the capillaries, which enables a more active exchange of fluids between the circulating blood and the adjacent tissues.

Let us now state briefly what happens in the retina when hemorrhages occur: The blood flowing out of the blood vessels will be found either to spread superficially amidst the nervous fibers having then the form of a candle flame, or it will penetrate more deeply between the fibers and will appear with a globular form, or else it will break through the internal limiting membrane and into the vitreous. These hemorrhages can be rapidly reabsorbed without leaving any reliquiae, but, more often than not, especially if the hemorrhages recur, degenerative changes may show, deposits of substances originated from disintegration of nervous cells, such as cholesterol and other lipoids may be found, the same as glial or conjunctival proliferation. The time in which a hemorrhage will be completely reabsorbed varies from four days to weeks, months, or even years. Generally speaking the larger the hemorrhage, the longer it will take to disappear and the longer the disintegrating blood cells stay in direct contact with the adjacent nervous cells of the retina the more harmful it is for the latter.

When retinal hemorrhages are due to stoppages in the bloodflow, be it in arteries or in veins, a certain portion of the retina, dependent for its nourishment on the impaired vessel, will deteriorate. In the case of arterial stoppage the circulation of the blood may be reinstated through existing anastomosis as in the case of the coexistance of the two systems—central ar-

tery and cilio-retinal artery, or through new-formed blood vessels and in both cases through gradual enlargement of the caliber of the aforementioned vessels. When venous vessels are stopped, the reinstatement of blood circulation will be even quicker, through adjacent veins, through enlarged capillaries or through anastomosis with choroidal vessels. Moreover, the blood clot can be perforated by one or several channels and circulation along the stopped vein itself, can be resumed. At the same time small collateral blood vessels are gradually enlarged. At first they cannot be seen ophthalmoscopically and if they are over the optic disc they might give the impression of hyperemia or if over the retina they might look like small hemorrhages. Gradually, however, they become visible with the ophthalmoscope and can be easily recognized by their typical aspect of a mesh of tortuous vessels with many anastomoses, forming a real "rete mirabile." These new-formed vessels apparently have no purpose but really they are meant to re-establish the impaired blood circulation. The longer any portion of the retina remains deprived of nourishment through lack of blood circulation, the more it deteriorates and the chances are that the blood vessels adjacent to that part will in turn develop changes which later will cause new hemorrhages. Thus, the sooner blood circulation is restored, the better it will be for the prognosis.

In retinitis proliferans one should discriminate between cases where, following retinal hemorrhages, conjunctival fibrous tracts with few blood vessels are formed at the optical disc and make their way into the vitreous and other cases where there is only a very thin layer of conjunctival semitransparent fibrous tissue sustaining a "rete mirabile" of blood vessels. In the first group we have the true retinitis proliferans, whereas in the second group the new-formed blood vessels are the expression of an effort to re-establish normal bloodflow in a section of the retina where blood circulation was abolished or impaired through the closing of the lumen of arteries or veins.

Retinitis proliferans of the second group, retinal hemorrhages due to various causes as in the complex generally called "Eales' disease", retinal changes due to arteritis and both peri- and endophlebitis can all be successfully treated with x-ray therapy by using small doses. Dilatation and increased permeability of the endothelium of the capillaries will be accessory to an improvement in the blood circulation, avoiding lack of nourishment of the adjacent retina and ultimately fresh hemorrhages and quickening the process of resorption of the existing blood among the nervous cells.

For these cases it would be advisable to use very small doses of 50 r repeated every 5 days. Miescher's three-wave theory would indicate that this should be the ideal interval as the second treatment would coincide with the interval between the first and second wave, when capillary dilatation would have reached its lowest, as it can be readily understood from the diagram.

True retinitis proliferans, (of the first group) where there are fewer new-formed blood vessels and more fibrous tract, does not respond to small doses of x-ray, larger doses being advisable, when tumefaction of the nuclei of the endothelium and accumulation of fibroblasts, the same as proliferation of the muscular layer, may bring about an occlusion of the lumen, discontinuing the bloodflow which as has been pointed out by Drs. Guyton and Reese might be responsible for fresh hemorrhages, establishing a "vicious circle." An action on the plasma, increasing its viscosity might be considered too. The higher sensitivity of the new-formed blood vessels to the x-rays explains the excellent results which were obtained.

When small doses of x-ray are given, the anterior portal may be used, as then no damage can be done to the cornea or to the lens, sensitivity of these to x-ray being much lower than the retinal blood vessels. The cornea starts reacting only when 800 r are reached and the crystalline lens only reacts after 500 r, 50 r sufficing for retinal capillary dilatation. For larger doses, as those which were used by Drs. Guyton and Reese, the temporal portal, or the nasal portal, when there is no hinderance, should be used.

For x-ray therapy of the retina, in its posterior pole, hard rays are advisable as they reach the retina with 100 per cent or even increased to 105 per cent due to secondary rays, whereas soft rays lose their power and only 25 per cent reach the posterior pole of the globe.

X-ray treatment for retinal repeated hemorrhages of different kinds and for retinitis proliferans is justified both by the clinical results seen in the last ten years and by our knowledge of its modus operandi. Moreover the difficulty in establishing the true etiologic diagnosis in many cases and, as a consequence therefrom, the inaptitude to combat the disease by destroying its primary cause, perforce must encourage the pathogenetic approach to the therapy.

The excellent results of x-ray therapy on 22 eyes with retinal posthemorrhagic new-formed blood vessels reported by Drs. Guyton and Reese justify my suggestion of general use of this method so that a larger number of patients,

to be observed over a long period of time, will enable us to reach definite conclusions.

WILLIAM L. BENEDICT, M.D., Rochester, Minn.: Since the application of x-ray therapy to recurrent hemorrhages is not a new procedure, we must consider the variations of the older procedures that have been advocated and used by their authors in their report.

We are somewhat familiar with the remote effects of radiation on ocular structures, even when the eye was not the direct objective of direct x-ray therapy; therefore, the use of x-ray therapy directed to fundal lesions has been restricted largely because of our reticence to initiate pathologic changes that may progress in future years into fibrosis and general degeneration of ocular structures.

I have had no experience with concentrated, directed x-ray therapy as has been employed by the authors. I am familiar, however, with the application of x-rays in the absorption of extravasated blood in the vitreous and their immediate and somewhat remote effect upon the proliferation of new retinal vessels.

I have noted the results of this method of treatment of hemorrhagic diseases of the eye in diabetics. I have been able to follow the effects over a period of from four to six years of mild doses of x-rays applied directly to the eye, either temporally or through the pupil. There is no question that blood in the vitreous will be absorbed much more rapidly following x-ray therapy of from 300 to 1300 r than if the disease is not treated. I am well aware of the fact, however, that retinopathy and diabetes progress with the age of the patient rather than with the severity of the diabetes and also that the most extensive retinopathy occurs in persons whose diabetes began early in life; regardless of the fact that the diabetes may be well controlled by diet or with the use of insulin, vascular changes progress, first on the venous side and later on the arterial side, and eventually proliferation of new vessels and fibrosis of old thrombosed vessels and hemorrhagic areas occur.

Later studies of the eyes so treated of persons who have had repeated hemorrhages into the vitreous and more or less extensive diabetic retinopathy, have shown that the repeated hemorrhages into the vitreous, even after standing for some time, will disappear more rapidly after the application of comparatively small or mild doses of x-rays. However, the proliferative changes in the vessels are not noticeably affected. In no case have I observed diminution in the proliferation of new vessels.

X-ray therapy also has been advocated in the treatment of thrombosis or occlusion of the retinal vessels from one source or another. Most of the cases that we have treated unfortunately have developed such extensive glaucomatous changes that enucleation became necessary and some others have not been followed.

In no case have we seen any definite improvement in the treatment of eyes in which an occlusion of the central vessels with hemorrhage has occurred. We treated one case of thromboangiitis obliterans more than three years ago. There was no noticeable effect, but I must say that the doses used were small and only two applications were given, not more than a total of 725 r. I think, therefore, that we must consider that the new development in this manner of treatment consists in the direct application of the rays of high intensity or large doses over very small areas. We have yet to see what effect it will have upon the proliferation of vessels in cases that are far advanced and the more extensive proliferations that occur not only in diabetic retinitis but are seen some times in Eales' disease. There is no question that the immediate effect on the hemorrhagic extravasation to the vitreous will disappear. That, however, is a virtue that cannot be attributed to the high dosage alone.

I should like to know the authors' opinion regarding the value of direct high-powered therapy on the proliferation of vessels in advanced diabetic retinopathy with proliferation, the so-called desperate cases, as well as what might be expected ultimately in fibrosis and degeneration of the retinal vessels.

ALGERNON B. REESE, M.D., New York, N. Y.: We have not discussed etiology, first because it is not within the scope of this paper, and, second, because we do not know anything about it. As a matter of fact, we do not even know to what condition the term, Eales' disease, should be rightly applied. We do know that the term is used to designate a rather heterogeneous group of cases, as already mentioned by Dr. Guyton.

Our impression is that Eales' disease proper is the following: a bilateral infectious process having a predilection for both the arteries and veins of the retina and frequently affecting also the uvea. A predilection for a causative agent to affect a certain type of tissue is not unusual. An example of this is the preference the herpes virus has for nerve tissue.

The purpose of our treatment is not to promote absorption of hemorrhage, but to dissipate new-formed blood vessels from which hemorrhage might come. The cases which have shown the greatest benefit from the treatment have

been those manifesting the greatest number of new-formed blood vessels of the so-called "rete mirabile" type, with a minimum amount of stroma and organized scar tissue. To date, this regression of the process has been followed by a decrease in the occurrence of retinal and vitreous hemorrhages. As Dr. Guyton emphasized, it is premature to say whether or not the regression and cessation of hemorrhages from this treatment will be permanent.

We are grateful to Dr. Alvaro for pointing out the rationale of the x-ray treatment in these cases. We agree with him that smaller doses of x-ray may suffice and our tendency at present is to reduce the total dose.

Dr. Benedict has rightly emphasized the fact that this treatment consists of directing the rays to the site of the lesion to the exclusion of the vulnerable anterior part of the eye. This is the most important feature of the treatment and cannot be stressed too much. This is the reason we can give large doses with a minimum of untoward effects.

Dr. Benedict asked about the value of this treatment in diabetic retinopathy. Our experience in this regard is confined to two cases neither of which was benefited. We hope to treat a sufficient number to assess the treatment in these cases and in particular those with a large element of new-formed blood vessels.

## In Memoriam

Ross Epting Anderson ..... Jackson, Mississippi
January 15, 1947

Edward Coleman Ellett ..... Memphis, Tennessee
June 8, 1947

Raymond Edwin Elliott ..... Rochester, New York
March 4, 1947

Edgar Leonard Goodman ..... Washington, D. C.
February 28, 1947

Howard Eugene Hamilton ..... Cleveland, Ohio
December 27, 1947

Franklin Fayette Lane ..... Philadelphia, Pennsylvania
January 28, 1947

James Orville MacDonald ..... New York, New York
January 13, 1947

Albert Ernest Rector ..... Appleton, Wisconsin
January 13, 1947

William Augustus Schwartz ..... Phoenix, Arizona
August 9, 1945

George Loring Toby, Jr. ..... Boston, Massachusetts
May 20, 1947

# In Memoriam

EDWARD COLEMAN ELLETT
1869 - 1947

# EDWARD COLEMAN ELLETT

## 1869 - 1947

*In the passing of Doctor Ellett, Memphis has lost one of its finest citizens and the Mid-South its dean of ophthalmology. No man in this section ever exercised such influence on the specialty as did he by his kindly interest in his patients and the problems of his colleagues. Although aware of failing coronary circulation for the past eighteen months he continued in limited practice and maintained his interest in all medical proceedings, local and national. He attended the meeting of the American Ophthalmological Society at Hot Springs, Va., June 5-7, where he participated actively in the discussions of the program. While en route to the Centennial Meeting of the American Medical Association on the night of June 7th he was stricken with an attack of angina, and was taken to a hospital from the train where the end came three hours later.*

*Edward Coleman Ellett, son of Judge Henry T. and Katherine Coleman Ellett, was born in Memphis, Tennessee, December 18, 1869. He was educated in private schools in Memphis, attended for two years the Southwestern Presbyterian University at Clarksville, Tennessee (now Southwestern College at Memphis), and received his A.B. degree from the University of the South, Sewanee, Tennessee. His medical degree was received at the University of Pennsylvania in 1891 where he was also honored with the Alumni Medal for the highest scholastic average in his class. After a year as Resident Physician in St. Agnes Hospital, Philadelphia, and a year as House Surgeon at Will's Eye Hospital, Philadelphia, he returned to Memphis in 1893, confining his practice to diseases of the eye, ear, nose and throat and since 1917 to diseases of the eye only.*

*In 1896 he married Nina Polk Martin and their mutual devotion for a half century made their union one of joy and beauty. Both interested in travel, she accompanied him on journeys to numerous medical meetings in this and foreign lands and before the late war on annual trips to Europe, especially France where he had served during World War 1. She was with him throughout his last illness.*

*Doctor Ellett was Professor of Ophthalmology in the Medical Department of the University of Tennessee from 1908 to 1922, and Chief of Staff of the Memphis Eye, Ear, Nose and Throat Hospital from its opening in 1926 until his death. He was also a member of the staff of the Methodist Hospital.*

*Doctor Ellett contributed generously to the medical literature on Ophthalmology. His papers were mainly on clinical subjects, but his interest in research was keen as evidenced by his constant attendance at meetings of the Association for Research in Ophthalmology and participation in the discussions.*

*He wrote the chapter on "Lacrimal Diseases" in Berens' text book "Diseases of the Eye" and will long be remembered for his advocacy of intracapsular cataract extraction and the use of the corneal-scleral suture, a procedure which he introduced to this country after watching Kalt, and which he lived to see in almost universal use in some form or other.*

*No man has received more honors at the hands of his colleagues. In 1914 he served as Chairman of the Section on Ophthalmology of the American Medical Association and was President of the American Academy of Ophthalmology in*

*1926 and of the American Ophthalmological Society in 1932. In addition he held executive positions in all local and sectional medical organizations having been President of the Memphis and Shelby County Medical Society, of the Tennessee Academy of Ophthalmology and Otolaryngology, and Vice-President of the Southern Medical Association. He was active in the American College of Surgeons and in the Pan-American Congress of Ophthalmology, and was in constant attendance at sessions of the International Congress of Ophthalmology.*

*In November, 1939, the Southern Medical Association honored him with "Ellett Day" dedicated to a review of his achievements as a physician, citizen and soldier. In October, 1942, Doctor Ellett was Associate Guest of Honor of the American Academy of Ophthalmology and Otolaryngology and was given the Award of Merit in recognition of his services as President, Member of the Council and Instructor, as well as Scientist, Teacher and Soldier. In May, 1943, the Memphis and Shelby County Medical Society honored him with a testimonial dinner to celebrate his fiftieth year of medical practice in Memphis, at which were nine Memphis ophthalmologists and otolaryngologists who had been associated with him in private practice during those years.*

*For many years a member of the Board of Directors of the National Society for the Prevention of Blindness and a Vice-President at his death, he gave freely of his time to the labors of this lay organization in the field of sight conservation. In 1939 in cooperation with the St. Louis Society for the Blind he was given the Leslie Dana Medal "for outstanding achievements in the prevention of blindness and the conservation of vision."*

*A member of the original American Board for Ophthalmic Examinations he helped to conduct the first examinations for certification in Ophthalmology at the first meeting in Memphis some thirty years ago. This interest was maintained to the end and he continued to review candidates examination papers until last year when he retired from The American Board of Ophthalmology after serving as President for several years. This, too, was the occasion of a testimonial by the Board.*

*During World War I, Doctor Ellett served in France as Commanding Officer of Base Hospital 115 with the rank of Lieutenant Colonel and received a citation for "exceptionally meritorious and conspicuous service." Discharged as a full Colonel in the Medical Corps of the United States Army Reserve he has been known to all his Memphis colleagues as "The Colonel," a term of honor, endearment and respect, which he accepted modestly.*

*In June, 1942, he received the honorary degree of LL.D. from Southwestern College, and in September, 1943, the honorary degree of D.Sc. from the University of the South.*

*His gentleness and kindly interest in his patients was rewarded by an enormous practice which he was delighted to share with his associates. A deft surgeon, his inclinations were always toward the simplest and safest procedures. In the past two years his practice was limited to ophthalmic consultation and surgery but his manual dexterity and steadiness was maintained to the very end and was praised by all who watched him.*

*Courteous gentleman, beloved physician, skilled surgeon, wise counsellor and staunch friend, his great influence will live on, but in the hearts of all who knew and loved him, his death leaves an aching void.*

RALPH O. RYCHENER

TRANSACTIONS
American Academy of Ophthalmology and Otolaryngology
*Official Publication of the American Academy of Ophthalmology and Otolaryngology*
Published under the direction of the Editorial Board
WILLIAM L. BENEDICT, Rochester, Minnesota, *Editor*
W. HOWARD MORRISON, Omaha, Nebraska, *Associate Editor*
HENRY L. WILLIAMS, Rochester, Minnesota, *Associate Editor*
Printed by Douglas Printing Company, 109 North 18th Street, Omaha 2, Nebraska

All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, the American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

# Editorial

## Section on Instruction

The program for the Section on Instruction of the fifty-second annual meeting to be held in Chicago, October 12-17, 1947, at the Palmer House, appeared in full in the March-April issue of the TRANSACTIONS, with the exception of the names of the instructors in the course on Occupational Ophthalmology. The lecturers for this course and their subjects are listed in the present issue (p. 566).

The condensed schedule of instruction courses and the advance order sheet appear again in this issue of the TRANSACTIONS. Orders for tickets are now being received. Each Fellow of the Academy sending in an order is notified by postcard of the tickets reserved for him. In cases where an order is not clear or where there is a discrepancy in the amount of money due, a letter is written to clarify the situation. To expedite the work in the office, it is hoped that these letters will receive your prompt attention.

As there seems to be misunderstanding on the part of some Fellows regarding the courses, may we please call your attention to the following points:

1. An *individual* course lasts for only one hour. It may be given on two, three, or four different days, each presentation being a repetition of the first.

2. A *continuous* course lasts two or more hours as indicated in the program. It is not possible to buy a ticket to only *part* of a continuous course.

3. The complete program of papers to be presented at the scientific sections held in the Grand Ball Room will be printed in the July-August issue of the TRANSACTIONS. The scientific papers in ophthalmology will be presented during the morning sessions. If you wish to attend these sessions, do not order tickets for otolaryngology courses. The scientific papers in otolaryngology will be presented during the afternoon sessions. If you wish to attend these sessions, do not order tickets for ophthalmology courses.

4. Orders are filled in compliance with first choices when possible. Please list second and third choices in the event first choices are not available.

5. *No tickets will be sent through the mails. All orders are to be picked up at the Advance Order Registration Desk in Chicago.*

6. Please do not ask to change an order that has already been filled.

7. A limited number of reprints of the Section on Instruction, as it was printed in the March-April TRANSACTIONS, are available to members who have lost their copies.

## Diversion Club

The Diversion Club of the Academy has completed the plans for the three day all-expense post Academy convention vacation at the famous French Lick Springs Hotel at French Lick, Indiana. No doubt many of you will want to enjoy this most pleasant outing. You may have three days of golf, horseback riding, skeet shooting or complete relaxation, or, if you wish, you may participate in a bridge tournament. There will be prizes for golf, skeet shooting and bridge. We know that many of you will want to have your wives join you in this delightful trip.

French Lick, in October, is one of the most beautiful spots in America. The weather at that time of year is delightful and invigorating.

It will be necessary to limit the number of reservations to one hundred, owing to the railroad facilities.

The over-all charge for this three day vacation is $82.50 per person from Chicago and return. If you wish to make this trip by automobile your railroad fare will be adjusted in the over-all fee charged.

The train for French Lick will leave the Dearborn Street Station over the Monon Railroad. The time of departure may be learned at the Registration Desk of the Academy in the Palmer House. A secretary will be assigned to take care of the details of the trip for those who have made reservations.

All correspondence relative to the trip should be directed to Dr. C. H. McCaskey, Chairman of the Diversion Club, 608 Guaranty Building, Indianapolis 4, Indiana. All checks should be made out to the Diversion Club of the Academy and mailed to the foregoing address.

If you wish to take this trip please fill in the blank below, stating whether you will travel by train or automobile.

### POST CONVENTION VACATION

C. H. McCaskey, M.D., Chairman, Diversion Club
608 GUARANTY BLDG., INDIANAPOLIS 4, IND.

1. I wish to make reservations for the post Academy convention vacation at French Lick Springs for_____persons.

2. I shall travel by train_____.

3. I shall travel by automobile_____.

NAME_____

ADDRESS_____

# COMMITTEE ON STANDARDIZATION OF TONOMETERS

## Report on Certification of Tonometers

DURING the past months certified Schiötz tonometers have at last become available for purchase. The Committee on Standardization of Tonometers wishes to give an account of the steps by which this favorable state has been reached. For almost a generation prior to the war, Schiötz tonometers, produced by a great number of independent and unsupervised manufacturers all over the world, have shown an increasing deterioration in the reliability of reproduction of the official Schiötz design, and had reached the point where each ophthalmologist needed to establish the normal range of readings of his particular instrument by direct clinical measurements. Instruments produced by different reputable manufacturers gave widely diverging readings on the same eye. These were the considerations which led the Academy to appoint a Committee for the Standardization of Tonometers.

### I. PROCEDURE FOR DETERMINING SPECIFICATIONS

The first step taken by this committee was to establish tonometer testing stations for the examination of tonometers submitted to them by ophthalmologists and institutions. The primary purpose of these testing stations was to see what could be done by way of recalibration and repair in order to make the readings taken with these instruments mutually compatible. These testing stations are still in operation and will continue their service. Reports on some aspects of their work have already been submitted, and further reports will be given from time to time.

The early work of these testing stations, particularly the New York Station established by Dr. Schoenberg and Dr. Posner, and the Chicago Station established by Dr. Kronfeld, included the collection of data that became of prime value in the subsequent plans for certifying tonometers. At these stations many instruments were weighed, measured, and tested, and the deviations in design which they showed in comparison with the official Schiötz design became known. By comparing the readings of two instruments that differed from one another in some particular, the significance of this difference in the final reading could be evaluated.

On the basis of this knowledge gathered from a study of several hundred tonometers, the Committee decided to attempt to set up standards and specifications for tonometer construction, and to invite the American manufacturer to co-operate in meeting these standards. It was agreed that once acceptable specifications had been established, arrangements would be made to certify tonometers submitted by the manufacturers which would be found to conform to the specifications. It was felt that the measurements leading to certification should not be made by the Committee itself, but should be made at appropriate cost by some independent agency. This would free the Committee from any possible imputation of partiality to any particular manufacturer, and would also leave the Committee free to consult and advise with any manufacturer who sought its help in overcoming difficulties involved in his particular design. It was not the purpose of the Committee to attempt to develop a new and improved design for the tonometer, but merely to see to it that certified instruments should conform to existing design within reasonable tolerances.

Through the initiative of Dr. Schoenberg, the Committee was most fortunate at this point in obtaining the offer of assistance from the Electrical Testing Laboratories, Inc., of New York City. This is a commercial organization engaged in testing a wide variety of electrical and optical devices. The Electrical Testing Laboratories, Inc., offered to test tonometers submitted to it, at cost, and to certify those instruments that were found to conform to the agreed specifications. Mr. William F. Little, Chief of the Photometric Division of the Electrical Testing Laboratories, Inc., has been most helpful to us in this work, and has given most generously of his time and thought to our problems. Much help was also obtained from Mr. Raymond Mehrlust, engineer in charge of the development of the Sklar tonometer.

A subcommittee consisting of Drs. Schoenberg, Kronfeld and Friedenwald was appointed to draw up the original draft of specifications for the Schiötz tonometer. After Dr. Schoenberg's death, his place on this subcommittee was taken by Dr. John McLean. Dr. Kronfeld undertook a special study to find out whether those measurements initially decided on actually included those most significantly related to the clinical efficiency and reliability of the instrument. At the same time, Mr. Little made measurements on a batch of fifty instruments furnished him by one of the manufacturers. The data which he obtained were analyzed by the subcommittee in order to discover how large were the variations to be expected from instrument to instrument under current manufacturing practice.

The variations found on these tests were used as a provisional basis for setting up tolerances, it being obviously desirable not to set tolerances so close that current methods of manufacture would have to be revised, and the price of the product consequently greatly raised. The provisional tolerances were then modified on the basis of the following considerations: (1) The influence of the proposed permissible variations on the accuracy and reliability of the tonometric measurement was analyzed in relation to the theory of tonometry as developed by Dr. Friedenwald,[1] and in relation to the clinical measurements on variant instruments that had been gathered by Dr. Kronfeld. In case the proposed permissible variations had only negligible effects on the tonometric measurement, the possibility of enlarging the tolerances was discussed with the manufacturers. In case the proposed permissible variations had an appreciable effect on the tonometric measurement, the manufacturers were asked whether they could conform to more rigid specifications without major and expensive changes in the method of manufacture. (2) In respect to some of the proposed tolerances, the manufacturers pointed out that while a group of tonometers could be produced with very little variation using their machine tools at a single fixed setting, the production of a second batch would involve resetting their tools and result in some new variations.

As a result of these considerations, a provisional set of specifications was drawn up and submitted to all American manufacturers known at that time to be engaged in, or planning to be engaged in the production of Schiötz tonometers, with the request that they comment on the specifications and indicate whether they would be willing to accept such specifications for their future manufacture. The only comments which the Committee received were from one manufacturer who regarded the tolerances as too wide in certain respects. As a result of some more recent discussions with various manufacturers, the specifications have been modified slightly. The present specifications are given in Table

TABLE I

SPECIFICATIONS No. 3 FOR SCHIÖTZ TONOMETERS (April, 1947)

All tonometers are to carry a serial number. The manufacturer of the tonometer is to be designated on the tonometer.

| *Test* | *Specification* *Standard* | *Limits* |
|---|---|---|
| Weight, grammes | | |
| Plunger, lever and pointer | | |
| Scale indication 5 | 5.5 | ±0.15 |
| Scale indication 10 | 5.5 | ±0.25 |
| Tonometer without handle | 16.5 | ±0.5 |
| Load 7.5 | 2.0 | ±0.02 |
| Load 10.0 | 4.5 | ±0.02 |
| Friction between plunger and sleeve | Negligible | |
| Friction between handle and sleeve | Negligible | |
| Foot plate, millimeters | | |
| Diameter | 10.1 | ±0.2 |
| Curvature | 15.0 | ±0.25 |
| Diameter of curved surface | 9.0 minimum | |
| Plunger millimeters | | |
| Diameter (measured 0°-90°) | 3.0 | ±0.03 |
| Curvature | 15.0 | ± .75 |
| Edge curvature-smooth, rounded | | 0.1 maximum |
| Scale indication | | |
| On 15 mm. standard | —1 | ±0.2 |
| On 16 mm. standard | 0 | ±0.2 |
| On own block | 0 | ±0.2 |
| Micrometer | | |
| Scale 5 | 5.0 | ±0.25 |
| Scale 10 | 10.0 | ±0.25 |
| Scale 18 | 18 | ±1.0 |
| Wobble | | |
| Plunger | 0 | 0.4 maximum |
| Fulcrum | 0 | 0.4 maximum |
| Extension of plunger below foot plate, millimeters | 0 | 3.0 maximum |
| Pointer, millimeters | | |
| Depth | | 3.0 maximum |
| Thickness | | 0.25 maximum |
| Distance between scale and pointer | | 1.0 maximum |
| Plunger contact with hammer | Perpendicular | 5-10 scale |
| Hammer, underside smooth, free from pits | | |
| Schiötz Chart correct | | |

I. It is proposed that as new and improved designs of the instrument, or methods of manufacture are developed progressively more stringent specifications may, from time to time be proposed, and that the reliability of the certified product may be still further enhanced at some future date.

Arrangements were then made with the Electrical Testing Laboratories, Inc., to certify Schiötz tonometers that conform to the agreed specifications. The Electrical Testing Laboratories, Inc., agreed to establish a scale of charges for its testing on a nonprofit basis. The Electrical Testing Laboratories, Inc., also agreed to make the results of its tests available to the Committee on Standardization of Tonometers for inspection and analysis.

During the development of this program, the suggested procedure for certification was submitted to and approved by the American Academy of Ophthalmology and Otolaryngology, the American Ophthalmological Society, and the Section of Ophthalmology of the American Medical Association. The form of certification which the testing laboratory has been authorized to issue is the following:

ELECTRICAL TESTING
LABORATORIES, INC.
2 East End Avenue at 79th Street
New York 21, N. Y.

CERTIFICATE OF
INSPECTION AND TEST

Schiötz Tonometer No. .....
E. T. L. No. .....
Manufactured by .....
Inspected and test on .....
and found to comply with Specification No. ..... for Schiötz tonometers, including requirements as to weights of plunger lever and pointer, of tonometer without handle and loading weights; friction; dimension and curvature of foot-plate and plunger; scale calibration; wobble; construction details. Specifications supplied upon application.

**Electrical Testing Laboratories, Inc.**
Inspected and Tested by .....
.....

Head, Photometric Division
Order No. ..... Date .....
Note: Specification No. ..... is approved by the Committee on Standardization of Tonometers of the American Academy of Ophthalmology and Otolaryngology. Arrangements for certifying tonometers have been approved by:

The American Academy of Ophthalmology and Otolaryngology.
The American Ophthalmological Society.
The Section of Ophthalmology of the American Medical Association.

## II. METHODS OF MEASUREMENT

The methods of measurement used in testing the tonometers submitted for certification are based on those worked out in the checking stations by Drs. Schoenberg and Posner, and by Dr. Kronfeld, with some refinements suggested either by Mr. Little of the Electrical Testing Laboratories, Inc., or by the manufacturers. These methods have for the most part been described,[2] and will not be given in detail here. These methods were demonstrated to the members of the Academy at an exhibit "Standardization and Rehabilitation of Schiötz Tonometers" by Drs. Kronfeld and Ricci at the meeting of the American Academy of Ophthalmology and Otolaryngology in 1944.

1. Plunger weights are determined with an accuracy of 0.01 gms. Within the tolerances currently accepted this degree of accuracy seems sufficient. Any considerable increase in the accuracy of measurement would involve very considerable difficulties since the plunger is weighed in the tonometer with the lever arm resting upon it. If the plunger is not exactly vertical during the weighing, some friction develops that limits the accuracy of the measurements. Consequently, if greater accuracy were desired, very time consuming adjustments of the tonometer during the weighing process would be required. The much less critical over-all weight of the tonometer is measured with an accuracy of 0.1 gms.

2. No satisfactory method for measuring the friction between plunger and sleeve, or between handle and sleeve has so far been devised. With new and well constructed instruments held in a strictly vertical position, the friction is too small to measure. It was decided, therefore, to accept instruments in which no resistance could be felt when the plunger was moved up and down in its sleeve while its tip rested on the examiner's finger.

The chief sources of friction in instruments of current design are the following: (a) In order that the plunger may be removable for cleaning, the disc which is attached near the upper end of the plunger has to be removable, and in current models is readily screwed on or off. It is necessary that the plunger should not rotate while the disc is screwed on or off. In order to avoid rotation of the plunger, a small pin is attached to it and this pin moves in a narrow slot within the sleeve of the tonometer when the instrument is assembled for use. Cutting this slot is technically a difficult procedure, and the edges of the slot are not always perfectly smooth. Manufacturers who have consulted us,

have been urged by the Committee to develop some alternative method of attaching the disc to the plunger that would avoid the necessity of the pin and the slot. (b) The second source of undue friction that has led to the rejection of some tonometers arose in the following way. The plunger is currently made of steel. Some manufacturers made the hammer of the lever arm out of brass. When such an instrument has been used a short time, a minute pit develops on the undersurface of the brass hammer, where the softer metal is indented by knocking against the hard upper tip of the plunger. When such a pit has developed, the tip of the plunger tends to stick in the pit and as the plunger moves up and down in the sleeve, its axis is displaced in relation to the axis of the sleeve, causing the plunger to lock in the sleeve. Instruments in which the hammer is made of harder metal, and in which the underside of the hammer is, as required by the specifications, smooth and free from pits, are free from this cause of friction.

3. Diameters and surface curvatures. The diameters of plunger and foot plate are measured with a micrometer with an accuracy considerably better than 0.01 mm. The radius of curvature of the foot plate and of the foot of the plunger is determined by resting the surfaces on thin metal discs of known diameters, and seeing whether light comes through the crack between the opposing surfaces mainly near the center of the concave surface or mainly near the edge. This method of measurement is not very satisfactory, and the tolerances given in the specifications represent the limits of accuracy of the testing methods rather than those of current manufacturing practice. The actual variations in acceptable instruments in this respect are probably considerably smaller than those permitted for certification.

4. Scale indications can be read with an accuracy of approximately $\pm 0.1$ scale unit. Standard blocks and the standard micrometer on which the scale readings are tested are in our experience reliable within the limits of the reading errors of the tonometer scale. In case instruments are developed in which the scale magnification is considerably larger than that of the conventional Schiötz, new problems in the reliability of the test blocks and micrometer will probably be encountered.

5. Wobble is tested in the Schiötz by holding the instrument on a flat plate, and determining the influence on the scale reading of lateral movements of the plunger, and of backwards and forwards movements of the fulcrum. Relatively large tolerances had to be allowed for these variations but, as will be noted below, it is unlikely that the full effect of the wobble enters into the tonometric measurement.

### III. RELIABILITY OF CERTIFIED TONOMETERS

In performing a tonometric measurement in clinical practice, there are obvious limits in the accuracy and reproducibility of the reading quite independent of any structural variations between different tonometers. The position of the pointer cannot be estimated closer than $\pm 0.25$ scale unit. During the reading the instrument cannot be held accurately to the desired vertical position, nor accurately centered on the apex of the cornea. The patient's eye cannot be held accurately in the direction of vertical gaze. Variations in the tension of the extraocular muscles and in the weight of the lids upon the eyeball cannot be controlled. It seems useful to lump together these factors under the term "clinical reading error," and to consider them separately from the variations in readings that are dependent upon variations in tonometer characteristics.

Repeated readings made with the utmost care with the same instrument on the same eye, indicate that the probable variations in readings due to the clinical reading error is of the order of magnitude of ±0.5 tonometer scale unit. In the region of the upper limit of normal intraocular pressure, i.e., 25-30 mm. Hg, this probable variation contributes an uncertainty of approximately ±2.5 mm. Hg or approximately ±8 per cent in the estimate of intraocular pressure.

Since variations in readings due to variation in tonometer construction will add to those due to uncertainties in the clinical measurement, a reasonable goal in respect to tonometer certification would be to reduce the variations in tonometer construction to such a level that the sum of the variations in readings due to instrumental errors would be less than half that produced by clinical reading errors. This goal has not yet been achieved in the specifications so far accepted, but actual manufacturing practice appears to do better than just meet the accepted limits. Table II gives the present estimate of the influence of the allowed tolerances in tonometer construction on the pressure reading. The estimates are based either on theoretical considerations or on actual clinical tests on variant instruments, and refer to readings at the upper limit of the normal intraocular pressure.

Some comment on these estimates is indicated. The influence of variations in plunger weight decreases with rising intraocular pressure. In the pressure region of 25-30 mm. Hg, both theoretical calculations and clinical tests agree that the influence of this variation is percentually roughly equal to the per cent variation in weight. Theoretical considerations indicate that the variation in tonometric measurements due to variation in the over-all weight of the tonometer would be proportional to the weight variation if plunger and over-all weight varied in the same proportion. Since the influence of variation in plunger weight has already been accounted for, the effect of variation in over-all weight on the tonometric reading should be somewhat less than proportional. The estimate given in the table is a compromise between the theoretical conclusions and the clinical measurements which, in this case, are not in satisfactory agreement.

No satisfactory data are available to estimate the effects of variations in foot plate curvature, and the provisional setting up of this variation as negligible may have to be revised in the light of future data.

The area of cross section rather than the diameter of the plunger enters directly into the theory of tonometric measurements. Both clinical measurements and theory are in agreement in

TABLE II

| *Tonometer characteristic* | *Maximum allowed variations* | *Estimated influence on tonometric reading* |
|---|---|---|
| Plunger weight | | |
| Scale indication 5 | ±2.7% | ± 3.0 % |
| 10 | ±4.6% | |
| Tonometer weight | ±3.0% | ± 2.0 % |
| Foot plate curvature | ±1.7% | negligible |
| Plunger diameter | ±1.0% | ± 2.0 % |
| Scale indication | | |
| On micrometer scale 5 | ±0.25 scale unit | ± 6.25% |
| Plunger wobble | ±0.2 | ± 2.5 % |
| Fulcrum wobble | ±0.2 | ± 2.5 % |
| Maximum error if all deviations summate | | ±18.25% |
| Maximum probable error assuming that individual deviations are as likely to be positive as negative | | ± 8.25% |

estimating the effect of this variation on the tonometric measurement as double the allowed per cent variation in plunger diameter.

The effect of variations in scale readings are estimated in the region around scale reading 4 corresponding to the average normal reading with the 5.5 weight. There are sound reasons for believing that in well constructed instruments the effect of wobble will be only partially exhibited in a tonometric reading. This conclusion is based on the experience that repeated measurements on the micrometer are reproducible within the limits of accuracy of scale readings ($\pm 0.1$ scale unit), in spite of the larger allowed wobble tolerance. The estimated wobble effect is, therefore, given as one-half that of the permitted wobble measurement, and may be regarded as an upper limit rather than as an exact estimate.

The influence of the sum total of all these variations may be calculated in two ways. If we assume that all the variations will, in a given case, operate in the same direction, then the total effect would be approximately $\pm 18$ per cent error in the pressure reading, or approximately $\pm 5$ mm. Hg error in the reading at the upper limit of normal intraocular pressure. This method of estimating the effect of the maximum allowed variation seems, however, to be grossly unfair, since it is most unlikely that in any given instrument all of the variations will influence the tonometric measurement in the same direction. For instance, if the plunger is overweight, it is rather more than less likely that it will also be oversized in diameter. These two variations will have opposing influences on the tonometric reading, and will tend to cancel each other out. It seems fair, therefore, to estimate the total effect of the allowed variations by assuming that they are independent. The estimate of $\pm 8$ per cent as the maximum error due to allowed variations in tonometer construction is obtained by taking the square root of the sums of the squares of the estimates for each individual allowed variation.

Moreover, only in the most exceptional cases would the actual variations in the physicial characteristics of an individual tonometer approach the maximum allowed tolerance in every single aspect. Consequently the probable variations among certified tonometers will be much smaller than the maximum possible variation. Too few instruments have as yet been certified to permit a rigorous analysis on this score, but a preliminary estimate indicates that the instrumental error among certified instruments will probably be much smaller than the clinical reading error.

During the period July, 1946 to May, 1947, two hundred and ten instruments submitted by manufacturers were measured by the Electrical Testing Laboratories, Inc. Of these, two hundred and five were found to conform to the required specifications and were certified. The remaining five instruments were readjusted by the manufacturer and the defects corrected. On resubmission all five were found to be acceptable and were certified. It is evident that those manufacturers whose instruments were included in the two hundred and ten submitted during this period of review have achieved a high degree of uniformity and reliability of their product.

An analysis of the deviation in these two hundred and ten instruments showed that the items where these instruments gave nearest to the maximum tolerated deviations were the following:

Micrometer scale reading 10
Wobble at plunger
Thickness of pointer
Distance between scale and pointer

In all other respects these instruments conformed very closely to the desired standards. If we consider only the de-

viations at micrometer scale reading 10, and those in respect to plunger wobble as sources of variations, the maximum deviations in these certified tonometers entail a maximum error of ±5 per cent, in the tonometric reading attributable to instrumental variations. This amounts to ±1 mm. Hg in the critical region of upper normal intraocular pressure levels. Since this is appreciably smaller than the clinical reading error, our Committee believes that its goal of making available to the profession tonometers that are devoid of clinically significant instrumental errors is a feasible one.

COMMITTEE ON
STANDARDIZATION OF TONOMETERS

Francis H. Adler, M.D.
Harry S. Gradle, M.D.
Trygve Gundersen, M.D.
D. O. Harrington, M.D.
Peter Kronfeld, M.D.
John McLean, M.D.
Adolph Posner, M.D.
Algernon B. Reese, M.D.
Jonas S. Friedenwald, M.D.
*Chairman*

SUBCOMMITTEE ON
CERTIFICATION OF TONOMETERS

Peter Kronfeld, M.D.
John McLean, M.D.
Jonas S. Friedenwald, M.D.

## REFERENCES

1. Friedenwald, J. S.: Contribution to the theory and practice of tonometry, Am. J. Ophth., 20:985, 1937.
2. Posner, A.: Standardization and checking of Schiötz tonometers, Arch. Ophth., 30:1, 1943.

# AMERICAN ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES

## The Program of a Small Eye, Ear, Nose and Throat Society

In many average towns there is a scarcity of opportunity for graduate instruction. Moreover, there often exists a lack of co-operation within the specialty groups. This situation was experienced and considered unfortunate in Reading, Pennsylvania. To remedy it the Reading Eye, Ear, Nose and Throat Society was founded September 24, 1941.

Reading is a typical North American industrial community with a population of approximately 110,000. Among its physicians are eighteen who limit their work to ophthalmology and otorhinolaryngology. The surrounding area has a population of about 300,000. In this section are forty-five eye, ear, nose and throat men, all of whom belong to the local Eye, Ear, Nose and Throat Society.

Until the new group was founded, Reading was handicapped by an absence of possibilities for graduate education in the specialties and by lack of co-operation within the profession. Now harmony and mutual understanding prevail because of a well integrated organization which benefits physicians and patients alike. The individual practitioner as well as lay and professional groups are encouraged in increasing measure.

Much of the credit for the success of the local society must go to the American Association of Eye, Ear, Nose and Throat Society Secretaries. This group consists of secretaries of local and regional societies. It is Pan-American in scope and is fostered and supervised by the American Academy of Ophthalmology and Otolaryngology. The Reading Eye, Ear, Nose and Throat Society has benefited greatly by continuous advice from the national organization. It has stayed in close contact with its officers from the initial draft of the constitution to the latest development of activities.

### MEETINGS

Programs have to be arranged carefully to give members what they desire. Interest is scant if papers are presented in monotonous sequence. Attendance is good when educational values are given with "trimmings" as background for relaxation. The routine described hereafter has been in force for some time. Needless to say it is undergoing constant changes because progress ceases wherever the schedule of performance is kept rigid.

The meetings are held in a men's club in the center of town every month except August. They begin at six in the evening with a cocktail hour followed by dinner. This get-together period has been found to be remarkably helpful. Opinions are aired and dissonances harmonized previous to official discussion. Edges of possible tempers are dulled and a mellow state of mind prevails which fosters unanimity of purpose. About eight o'clock a short business meeting is held. Because divergent views have already been straightened out disagreements are rare and the business meeting is brief. The scientific session which follows has topics alternating between ophthalmology and otorhinolaryngology. Numerous guests are invited and are usually present: anesthesiologists, pathologists, opticians, psychologists, the executive personnel in charge of the program for the prevention of blindness, and others. In addition to

routine scientific programs, meetings with groups having related interests have been found stimulating and have added a new viewpoint. Such programs are comprised of topics of mutual concern, and a wider horizon of medical interest is given to all who attend. These special meetings are as follows:

*Academy Meeting*

All the members of the organization are urged to attend the annual meeting of the American Academy of Ophthalmology and Otolaryngology. Those who are not Academy members are encouraged to go as guests. If possible, train reservations are made for a joint trip. A sitting room is engaged at the hotel for informal get-togethers and for entertainment of friends encountered at the sessions.

A week or so after the Academy meeting, members review, at the local meeting, instructional conferences and lectures. Round table discussions follow. In this way the value of the instruction is multiplied, and consideration is given to subjects which previously had not seemed to be of consequence. But more than that: by preparing summaries an added didactic value is given to those courses which had been selected by Fellows of the Academy as of primary importance to them. Thus the teaching potential of the Academy is extended and magnified.

*Joint Meeting with the County Medical Society*

The topics of these meetings are selected to be of interest to everybody present. For instance: Symposium on Vascular Disease; Symposium on Glaucoma for the General Practitioner; The Medical Management of Sinus Disease. At first it was difficult to engage the attention of the County Medical Society. For a long time many general practitioners, annoyed by the fact that few, if any specialists attended their meetings, appeared to consider specialists "high-hat." At the same time they felt that joint programs would be too technical. The results obtained in this community have shown this opinion to be without foundation. Actually a great deal of mutual understanding has come out of these meetings which are presided over jointly by the presidents of the County Medical Society and the Eye, Ear, Nose and Throat Society.

*Joint Meeting with the State Academy*

The April meeting is held as a part of the annual meeting of the Pennsylvania Academy of Ophthalmology and Otolaryngology. Several years ago a number of eastern Pennsylvania eye, ear, nose and throat societies decided to meet jointly once each year. They formed the Eastern Pennsylvania Association of Eye, Ear, Nose and Throat Physicians. This organization was subsequently enlarged and became the Pennsylvania Academy of Ophthalmology and Otolaryngology. The new Academy has a two-day program each year. Meetings are held in country clubs or resorts, places removed from metropolitan centers. Personal relations are fostered more successfully in such environments than they would be within the hubbub of a city. Old friendships are renewed and new acquaintances formed. Social intercourse is considered as important as attendance at the scientific sessions. The environment, with its country vistas, a game of golf, a walk in the woods, five o'clock tea, a cocktail or two, do much to attain that friendliness which is essential for the successful practice of ophthalmology or otolaryngology.

In addition, physicians who are unable to be members of local societies are eligible for Associate Membership, on recommendation and other investigation. Associate Membership was established especially for a certain type of physician: namely, the individual who is recognized in his own small town as an

eye, ear, nose and throat specialist but who is doing, by necessity, some general work which makes him ineligible for membership in his own specialist society.

While the Pennsylvania Academy prefers diplomates it has made it its policy to provide graduate education also for men who are not eligible to take either of the Boards. It encourages these associate members to limit their work in the future and to try to fulfill the other requirements of the Board of Ophthalmology or the Board of Otolaryngology.

*Joint Meeting with the Diplomates Association*

The Diplomates Association was founded on the initiative of the local Eye, Ear, Nose and Throat Society. In past years it had become evident through their enthusiasm that diplomates of other specialties liked the meetings: perhaps because of the quality of the programs, but perhaps only because of the good fellowship and the resultant understanding and co-operation in hospitals and private practices. Some years ago, on invitation by the Eye, Ear, Nose and Throat Society, a special diplomates dinner was held which resulted in the formation of the new organization. Topics for the meetings of the Diplomates are of interest to the entire group. For example: The Present Status and the Future of Anesthesia; Allergy in the Specialties and in General Practice; Atomic Energy in Medicine.

*Joint Meeting with the Local Dental Society*

This co-operative experience has been eminently satisfactory. Topics of borderline interest are discussed by dentists, dental surgeons, maxillofacial surgeons, otolaryngologists, and ophthalmologists. The viewpoint of the dentist and the attitude of the otolaryngologist are modified, and a great deal of common ground is created. Knowledge and understanding of facial anatomy and physiology have been deepened in both groups. The patients in this county are gradually becoming aware of something new in the practice of medicine. The eye or the ear, nose and throat man no longer exudes a feeling of self-sufficiency; nor does the dentist. An integration of dental and medical service has come into being, for the benefit of surgeon and patient alike.

*All Day Hospital Meeting*

The day begins with eye, ear, nose and throat surgery in one of the local hospitals. The operations are followed by an ophthalmologic dry clinic, an otorhinolaryngologic dry clinic, and a dental dry clinic. Lunch is served at the hospital. Afterwards a paper is read by an out-of-town speaker. Scientific and technical demonstrations are presented. The rest of the afternoon is given over to golf and friendly get-together. The evening begins with a cocktail hour succeeded by a banquet. The banquet is addressed by an eminent speaker who is the guest of honor of the occasion. If the afternoon speaker is an ophthalmologist, the evening speaker is an otolaryngologist, and vice versa.

*Clinics at Medical Centers*

Once a year a trip to a university is undertaken. On this occasion the Society is divided into an Eye Section and an Ear, Nose and Throat Section. Surgical and dry clinics are given at teaching hospitals. They are followed by ward rounds and lectures. The sections come together in the evening for dinner. The day ends with attendance at the Section of Otolaryngology of the College of Physicians to which the Reading Eye, Ear, Nose and Throat Society has a standing invitation.

*Local Speakers*

One month is reserved for presentations by local talent. Reminiscences and

"I had a case" papers are discouraged. Original research is fostered in so far as this is possible in a small town. For example, a lucid explanation of the optical errors inherent in testing with prisms was much appreciated.

*Study Sessions*

Several times a year the members of the organization meet to review literature, to hear digests of other meetings, and to summarize recently published books. Ophthalmological and otolaryngological reporters are appointed whose duties are to gain comprehensive knowledge and to impart it intelligently to the membership at large. The reporters are the younger and newer men with enthusiasm, elasticity of mind, ability to interpret, and time for study. Study sessions are quite informal. As round table affairs in every sense of the term they contribute a great deal to the professional knowledge of each participating individual.

*Christmas Get Together*

In December, wives and friends are invited. The topics are selected for their wide general interest. For example: Symposium on Contact Lenses. A paper was read by a physician. He was followed by a contact lens technician. A technical movie, demonstration on patients, and an all around discussion completed the topic. The program is part of the annual Christmas Dinner Dance, a most enjoyable affair. The wives seem to be pleased with an invitation to participate in scientific proceedings. Doubtless they take home a great deal of pertinent information. Perhaps they get a clearer understanding of their husband's profession and of his day to day problems.

*Annual Picnic*

Once during the summer the Society meets in the early afternoon for swimming, other sports and fun. A picnic and clam bake follows. Then comes a short scientific session, lasting about half an hour. Extensive round table discussions end the evening, ranging from unusual cases to office procedures and from surgical technics to reminiscences of visits to clinics here and abroad.

*Officers*

In March occurs the election of officers for the next year. They will take over in September. Electing the officers six months ahead of time puts them in position to become familiar with their duties. Also, they are given a chance to look around so that they can tentatively line up speakers. Above all they are made to feel they have a special interest in their Society. Instead of having this special interest for one year only they have it for eighteen months because of their early election. Thereby a beneficial increase in the continuity of administration is achieved.

The executive personnel of the Society serves for one year with the exception of the Secretary who, by custom, is re-elected year after year. In this way there exists rotating responsibility. Everybody is given a chance to function as an administrator, and "growling" is converted into constructive criticism and into action. In addition, this rotation of officers puts the governing of the group on the broadest base possible, it prevents the formation of a clique, and it makes every member responsible for his organization.

NATIONAL SUPERVISION

The American Association of Eye, Ear, Nose and Throat Society Secretaries helps local secretaries to formulate intelligent programs. The Association is in the process of evolving long range program possibilities for continuous instructional development, year after year. It expects to assist in the coordination of local activities for sections of the United States.

An added educational feature is under consideration. Through this plan each local society could subscribe to an instructional series presented by the American Academy of Ophthalmology and Otolaryngology. One whole year would be allocated to each subject, for instance: Sinusitis, Glaucoma, Allergy, Detachment. The necessary material would be sent to one member of each society, a man sufficiently trained and interested to spend the time required for study. He would give a short paper covering the monthly assignment, previous to the regular meeting of the group. It is hoped that this plan will be put into effect in the not-too-distant future.

## CONCLUSION

A small Eye, Ear, Nose and Throat Society has found it expedient and successful to institute routine and special programs. With this system one does not go to "the same old monthly affair." Rather, one looks forward with eagerness each month to an experience which may be challenging. This experience should be ever changing and should not tend to become shopworn, stereotyped and dull.

Stimulation derived from attending the monthly meetings has had an effect on the work of each eye, ear, nose and throat man in this community. The benefits accrued are manifold, and a very important one is the fact that specialization has lost much of its limitation. Instead of becoming increasingly more and more confined to a small subdivision of medicine each man is constantly tying up with other professional and civic groups. It is precisely this interrelation and association with others that has made "The Reading Plan" successful.

The annual program of the Reading Society should not be considered a prototype for other organizations. It is no more than a tentative formulation of the desire for additional education of a group of average practitioners. Improvement will continue and SUGGESTIONS FOR CHANGES ARE MOST WELCOME. Constant experiments are of importance in the development of ideas. New thoughts should be given a try-out on a small scale, and audience reaction should be studied and interpreted critically.

Heretofore, graduate education of practitioners in small communities consisted of attendance at national and sectional meetings, clinics, and courses. It is hoped this will be enhanced and augmented by programs in local societies: logical in long range planning, intelligent in execution as well as gratifying and enjoyable for every individual member.

PAUL C. CRAIG, M.D.
232 North Fifth St.
Reading, Pa.

# OCCUPATIONAL OPHTHALMOLOGY

## Notice

The program for Dr. Kuhn's twelve-period instruction course on **Industrial Ophthalmology** to be presented by the Section on Instruction at the Academy meeting, October 13-16, 1947, is as follows:

*Monday, October* 13, 2-5:30 *p.m.*

**Opportunities and Responsibilities of Ophthalmologists.** Derrick Vail, M.D., Chairman, Joint Committee on Occupational Ophthalmology.

**What Constitutes an Industrial Eye Program.** Hedwig S. Kuhn, M.D., Secretary, Joint Committee on Occupational Ophthalmology.

**Visual Testing Technics.** Henry Imus, Ph.D., (By Invitation), Office of Naval Research, Navy Department, Washington, D. C.

*Tuesday, October* 14, 2-5:30 *p.m.*

**Fact Finding Methods.** N. C. Kephart, Ph.D., (By Invitation), Industrial Vision Institute, Purdue University, Lafayette, Ind.

**Eye Protection Equipment and Program.** John Roche, M.D., (By Invitation), Director, Industrial Section, National Safety Council.

**Compensation and Medical-Legal Problems.** C. O. Sappington, M.D., (By Invitation), Industrial Health Consultant, Chicago, Ill. Harry H. Nelson, (By Invitation), Director, Workman's Compensation, Industrial Commission, Wis.

*Wednesday, October* 15, 2-5:30 *p.m.*

**Toxic Hazards.** Ralph McLaughlin, M.D., Consulting Ophthalmologist, Carbide & Carbon, W. Va.

**Plant Experiences—Round Table Discussion.**

Jean Felton, M.D., (By Invitation), Medical Director, Monsanto Chemical Co., Oak Ridge, Tenn.

Edward Showalter, M.D., (By Invitation), Western Electric, Chicago, Ill.

Walter Fleischer, M.D., (By Invitation), Medical Director, Rustless Iron Steel Div., American Rolling Mill, Baltimore, Md.

David Cook, Ph.D., (By Invitation), Industrial Relations Director, General Analine & Film Corporation, New York, N. Y.

Carl Dietz, (By Invitation), Assistant Vice President, Wyman Gordon Co., Harvey, Ill.

*Thursday, October* 16, 2-5:30 *p.m.*

**Visual Skills and Correcting for the Job.** Hedwig S. Kuhn, M.D.

**Seminar—Individual Problems of Members of Course.** Faculty.

### INTRODUCTORY STATEMENT

*During the war, a very intensive and extensive questionnaire was sent all the shipyards in the country that were under contract with the Maritime Commission. The purpose of this questionnaire was to secure information on eye accidents, frequency, type, treatment, loss of eyes (partial or total), and all the other problems which were involved. Special emphasis was placed on diagnosis and care of flash cases as these formed a large percentage of eye accidents in the shipyards. This material as a whole, is being summarized and studied and will be published in the near future. It will give an overall picture of eye accidents in shipyards involving at least a half million workers. One of these shipyards was located in southern Indiana as a purely war operated set-up. The consulting ophthalmologist of this yard has made a special study of their eye accidents and has submitted a report which we thought was an excellent example of the problems of shipyards from the ophthalmological angle.*

*There are a number of comments that seem pertinent to this report. The statement "since the employee was not compelled to wear his safety equipment . . . . ." points to one of the most serious of all problems in the shipyards during the war. In order to get production, the emphasis was put on care of eye accidents rather than on supplying and enforcing safety procedures and equipment. It is known, of course, that there was a shortage of safety goggles. While this added to the problem, it is to be noted that the medical profession did not and does not condone this attitude on the part of management, and will continue to work toward eliminating inadequate protection wherever found during peace time. Also, it is to be regretted that visual acuity was not taken before the removal of a foreign body. That practice was also the result of war pressure and is in direct opposition to the number one rule in the plant*

*or private office where foreign bodies are treated, namely, make an accurate acuity of each eye separately before the injured eye is touched. It is significant that in 1944 (the peak year) the cost of eye care in this plant came to $6,500 per month and that 60 per cent of all the accidents were eye cases. After the installation of medically controlled emergency eye care, this cost was considerably cut and the quality of professional care increased.*

*By and large, the experience in industry has shown that the use of the infra-red lamp in flash cases is not advisable. (A statement to this effect was issued by the Joint Committee of Occupational Ophthalmology several years ago in the Journal of the American Medical Association.) The same relief can be obtained by the use of ice compresses.*

*Most ophthalmologists, working in industry, would take exception to the use of atropine, not only because of the danger of glaucoma but because of the unnecessarily long period of blurring of the worker's vision. Here, we should like to again call attention to the previously reported use of homatropine in oil which will relieve ciliary spasm for from 24 to 36 hours instead of the usual one or two hours that an aqueous solution of homatropine produces. There are many industries in which the blurring of vision through the routine use of atropine would be an enormous handicap. Since the same result is obtainable with homatropine in oil, we would suggest that this point be given serious consideration.*

*The use of patches following the removal of a foreign body seems to be determined by local problems as they exist in the plant, in the region, and in the follow-up routine of eye accident cases. In the Calumet district, a patch is used in every case where a corneal foreign body is removed by a spud. It is good protection even though it may be in use only for an hour or so. It involves no hardship on the part of the patient, and we have never found any relationship between a patch and a corneal ulcer if the proper medication is used after the removal of foreign body. On the other hand, patches are rarely used in the Pittsburgh area as employees refuse to co-operate. For this reason, cauterization has been reluctantly substituted in many instances.*

*This is a most illuminating paper and one for which we should compliment the authors for the intensive study of a problem which was certainly not in the ordinary realm of their work prior to the war, and was carried on under the pressure and hardship of war practice. We all learn by this generous type of service and its presentation.*

*At no time has the medical profession been supplied with even the desired minimum amount of information on toxic hazards to eyes, what they consist of, what effects are produced locally or systemically, and what treatment is indicated. It was the tremendous good fortune of the Joint Committee to have discovered the brilliant work of Miss Van Arsdell. Here, we found the starting point for supplying this detailed information which was so badly needed not only by the medical professions but by Safety Directors in Industry and Public Health Services. The paper presented here will form the preliminary statement of a Manual on Toxic Hazards which the Joint Committee is sponsoring with the cooperation of the National Society for the Prevention of Blindness. This Manual will be made available in the very near future and will include detailed tables of the toxic substances themselves, their effects and indicated treatments. We felt, however, that it was urgent to provide the information in this fine paper immediately to the ophthalmologists who are eager for it.*

HEDWIG S. KUHN, M.D.
*Secretary Joint Committee*
*Occupational Ophthalmology*

## A REPORT OF THE INDUSTRIAL EYE ACCIDENTS AT EVANSVILLE SHIPYARDS

HOWARD C. SLAUGHTER, M.D., *and*
WILLIAM M. COCKRUM, M.D.
EVANSVILLE, IND.

THIS is a report on the Evansville Shipyards, which was operated by the Missouri Valley Bridge & Iron Company at Evansville, Indiana. This shipyard was an open yard; that is, the ships were constructed outside. The yard was not in existence in 1940 and out of existence in 1946. The yard was constructed hurriedly for assembling ships rapidly. There was very little consideration as to the employment hazard in the construction of the yard.

This office was approached in the spring of 1944 by the shipyard to take over the handling of all eye injuries as they were having considerable time lost and the expense of the eye injuries was running very high. From that point on

an attempt to keep a rather complete record of the activities of the eye cases at the shipyard was made, and this paper tries to draw some conclusions between the systems of handling the patients prior to the engagement of us and after we were engaged.

The Missouri Valley Bridge & Iron Company gave each employee a physical examination, which was done by the employment office. The eye examination consisted of testing vision at 20 feet and color perception. A vision of 20/30 with correction was accepted for general employment; that is, the employee had to have at least 20/30 vision in one eye. If an employee had a vision of 20/40 in one eye, or both eyes, he was then called a handicapped employee. He was hired for a specific job and could not be transferred to another department or to different work without consent of the department head under which he was working. There was no waiver of visual impairment signed by the employee and an employee with vision of 20/30 in one eye only was employed with handicap.

The records at the employment office were available to us. The safety department furnished and requested that each employee wear such safety devices as were generally accepted for his type of employment. These were available in adequate numbers and were furnished without charge. However, some workers, at their own expense, obtained specially ground safety glasses and welder's goggles in shades that were not supplied by the company. The recommendations for the shades of flash goggles and safety shields, etc., were the same as other industrial plants. It must be borne in mind, however, that the majority of the labor at this shipyard were green labor; that is, having little or no experience in industrial employment. Although the employee was not compelled to wear his safety equipment, we found that after one or two visits to the First Aid Station a greater percent of the workers wore their safety devices.

When an employee was injured the usual routine was to obtain a pass from his foreman and then report to First Aid. However, the yard was so arranged that the foreman frequently was not available to issue such passes. These passes were not required by the First Aid Station but were issued to give the impression that the employee was being observed in the time consumed in visits to the First Aid Station. At the First Aid Station the history was obtained by the receptionist as to time and place and what the employee was doing at the time of the accident. The employee was then turned over to a registered nurse. Visual acuity was not taken. The registered nurse handled all foreign bodies that did not require a sharp instrument to remove.

When the shipyard was first set up all cases that the registered nurse was not able to handle were turned over to the Surgeon-in-Chief, who was not an ophthalmologist. If he was unable to handle the case, the case was then turned over to one of the ophthalmologists in the community. The patient was sent to the ophthalmologist at the company's expense, both as to time and transportation. It was found that this procedure consumed a great deal of the employee's time. Later, assigning of the cases was left to the judgment of the registered nurse who usually sent the cases directly to an ophthalmologist, so the case was not seen by the Surgeon-in-Chief. If the employee was not seriously injured and could return to work, he was returned to work without a lost time record. Otherwise, he was discharged on sick leave as of the day and time of the injury. In order to return to work he had to be released by the medical doctor that was handling his case. He took his release to the First Aid Station where the nurse turned it over to the

Time Department and his closed time chart was again opened. If he refused treatment, his time card was closed.

Frequently an employee was released by the medical doctor stating that he could do light work. However, such people were turned down by the Time Department as the man was hired at a scale and this scale had to be maintained in order to hold up Navy and Union standards. An insurance report was sent in on all employees that received more service or medication than was normally supplied at the First Aid Station. A report was sent to the Indiana Industrial Insurance Board, the insurance carrier, and one was kept by the First Aid office.

To repeat, when the Missouri Valley Shipyards first opened, all injuries were referred to the Surgeon-in-Chief, and those who required special treatment were sent to ophthalmologists in the community for further treatment. At first all eye patients were handled by one man. However, as employment grew the situation became quite impossible for one man to handle, so the employees were sent out to several ophthalmologists. The cost of this procedure increased the bill for eye injuries immensely. Both the price of the treatment and the abuse the employee made of the fact that he had left the yard, along with the difficulty in transportation between the yard and various offices, all contributed to the increased cost. Also, to facilitate transportation, attempts to group the cases were made. This would usually end up by one man being seen by several ophthalmologists. This, in turn, led to confusion in the insurance forms, the fee charged, and unsatisfactory follow-ups.

From statistics obtained from the First Aid Station of the Evansville Shipyards it was estimated that from 15 to 18 patients on an average per day, including Sundays, left the Evansville Shipyards for treatment by eye specialists. These men were seen at $3.00 for the first visit and $2.00 for each subsequent visit, unless the man was seen after office hours, when the charge was $5.00 for the first visit and $2.00 for each subsequent visit. To this price must be added the time spent away from their employment, which averaged two hours at $1.20 an hour and $1.00 for transportation.

From March 7 to April 7, 1944, 382 patients were treated in this manner. For one month after this date 385 patients were seen by the contract eye physician at the shipyard. Seventy-five of these were seen in the office at the above quoted prices. These patients were seen in the office due to their working hours and cases which could not be handled adequately with the equipment at the shipyard. This group also included those hospitalized. This left 310 patients that were seen at the shipyard on which no clerical work was done; the patient did not lose over 30 minutes from his work; and the patient was usually seen several times.

It was estimated by the shipyards that at least $1,500.00 a month was saved, plus a great amount of clerical work. This is a very conservative estimate. However, from reviewing Charts I and II one can see that there was a great decrease in the amount of compensation paid and a decrease of nearly $1,400.00 on doctor bills that were paid in the last six months of 1944.

The Indiana law, under which these cases were settled, allows for the following compensation: for the permanent loss of sight of an eye or its reduction to 1/10 of normal vision with glasses, 150 weeks; for any permanent reduction of sight of an eye, compensation shall be paid for a period proportionate to the degree of such permanent reduction. The American Medical Association Table on Visual Impairment was

used in computing the percentage reduction of visual efficiency. The compensation is based on 55 per cent of the average wage with a minimum of $20.00 per week and a maximum of $36.50. Since all of the employed were receiving the maximum salary, they would receive a compensation of $20.07½ a week. However, from this amount allowed is deducted the amount the patient received for temporary total disability. That is, the compensation he received when he had been off work for seven days and up to the time he went back to work. For the loss of one eye he would receive a total of $2,705.00. For the loss of both eyes he would receive the maximum, or $5,500.00.

At the time this office signed the contract with the Missouri Valley Bridge & Iron Company to supply eye care and direction of eye care to the employees, there had been rather a lax system of survey and inspection done by the First Aid Station in the cases of eye injury. One of the first things that was done was to instruct the nurses in a manner of locating and recording position of a foreign body, using the familiar clock-dial system. At this time also the use of fluorescein as a staining agent in all eye cases was enforced. At this time also a routine was set up for the care of all the more common eye complaints that came through the First Aid Station.

The treatment table was stocked with the following items: pontocaine ½%, fluorescein 5%, normal salt solution in a dispenser, zinc sulphate ¼%, pontocaine ½% and adrenalin 1/1000 in equal parts, 1% silver nitrate and a solution of ¼% zinc sulphate with 20 minims of 1/1000 adrenalin ½ oz., homatropine 5% and atropine 1%. The nurses were instructed never to use the silver nitrate, nor the homatropine, nor the atropine, and were instructed to use fluorescein followed by a copious wash with normal salt solution on each eye that came in for inspection. Foreign bodies that could be lifted off without curetting were to be removed and recorded. Rust rings were to be referred to the office of the consulting ophthalmologist or held over until the next visit at the First Aid Station by the ophthalmologist. Flash was to be treated by the instillation of one drop of adrenalin and pontocaine and then a fifteen-minute exposure to the infra-red lamp. This treatment of flash we found to be highly efficacious in a large series of cases of this type. Prior to the use of infra-red we had been in the habit of using cold applications.

Foreign body allegations were to have two drops of ½% pontocaine instilled in the eye, followed by a two-minute waiting period, then the eyeball and eyelids were carefully examined with a loupe. A routine for examination of the eyeball consisted of the individual looking up, down, to the right, to the left. The upper lid was everted and examined and the inferior cul-de-sac was inspected. When the foreign body was discovered, its location, character, and size were recorded. Any abrasion of the cornea or eyeball that became apparent through staining with fluorescein was recorded. If an employee stated that something hit his eye with great force and yet no foreign body was found, the eye was stained with fluorescein. If a staining area was found, the patient was referred to the ophthalmologist. We are cognizant of only one case of an intraocular foreign body missed by the nursing force in their routine inspection during a two year period. This case was discovered in a routine homatropine refraction when the individual came to our office as a private patient.

Generally, the following routine for visiting the shipyard was followed: At 8:30 each morning one of us went to the First Aid Hospital where we saw all of

the employees who were coming off the graveyard shift and all the employees who were going on the day shift who had eye complaints. The fact that we were to be there at 8:30 a.m. was generally announced throughout the yard so that all were familiar with this practice. We made no discrimination among the men seen at this time as to whether their eye complaints were of a compensable or non-compensable nature. Prescriptions were issued as we would have done in our office in private practice. We should like to emphasize that we explained to the non-compensable cases at this initial visit what their eye situation was in order to avoid any future conflicts in the employee's mind. Any injuries that necessitated refraction, visual field studies, slit lamp study, or careful ophthalmoscopy were referred to the office.

Prior to the routine use of pontocaine it had been our custom to use other anesthetics (butyn holocain) in a like manner. This was stopped because of the numerous cases of sensitivity that resulted. This was done with no discrimination but simply in an effort to stop the lost time and trouble that arose from these cases. Some such cases were severe enough to require hospitalization. In the year and a half since the use of pontocaine was put into effect we have seen no instance of dermatitis of the lids that could be attributed to its use.

Cases of conjunctivitis that came into the First Aid Station were treated routinely with zinc sulphate instillations and were instructed to return to the First Aid Station for the next visit of the ophthalmologist. Any cases which came in during the swing shift were either asked to return to the First Aid at 8:30 the following morning or were referred directly to the ophthalmologist's office, depending upon the degree of discomfort and type of complaint encountered.

The use of all eye ointments at the yard was prohibited unless prescribed by the consulting ophthalmologist. It had been a semi-routine practice, prior to our contract, to issue some anesthetic ointment to the employees who complained of flash burn. Many of the employees were in the habit of carrying tubes of these preparations around with them. This was strenuously discouraged, again due to the great number of cases of dermatitis and allergic conjunctivitis encountered in this group. We may add in this respect that we have seen three or four cases of dermatitis from the use of pontocaine ointment although as we mentioned above, pontocaine in liquid form has never given us any trouble.

We have had a very small number of corneal ulcers following removal of foreign bodies but these have been treated routinely by immediate hospitalization for the administration of aqueous penicillin locally to the eye along with hot fomentations every two hours and atropine instillations. Generally, these cases cleared up promptly and in two or three days they were sent back to work.

We should like to state that we have used atropine routinely in eyes showing ciliary congestion unless the individual was doing some specialized form of work. All these patients were kept on the job with dark glasses with little or no trouble. We had seen one case of glaucoma only while the yard was open and this was a case that had received no atropine. No discrimination on the use of atropine was followed as far as age was concerned. The majority of the cases that were employed at the shipyard were over 35. The larger per cent fell into the 40 to 55 age group, or at least in the group of patients that we saw a great number were over 40 years of age. They received atropine for ciliary congestion just as the younger group did.

CHART I

| | 1943 FIRST HALF | 1943 LAST HALF | 1944 FIRST HALF | 1944 LAST HALF | 1945 FIRST HALF |
|---|---|---|---|---|---|
| Total Number of Man Hours Worked | 15,369,352 | 15,299,214 | 20,930,444 | 20,367,614 | 12,639,752 |
| Total Number of Eye Injuries | 7,376 | 7,082 | 15,052 | 13,686 | 7,497 |
| Total Number of All Injuries | 13,828 | 14,120 | 29,641 | 26,788 | 12,451 |
| Percentage of Eye Injuries of All Injuries | 53% | 50% | 50% | 52% | 60% |
| Total Number of Lost Time Injuries | 306 | 153 | 520 | 485 | 258 |
| Total Number of Eye Lost Time Injuries | 169 | 67 | 116 | 80 | 25 |
| Percentage of Eye Lost Time Injuries of All Lost Time Injuries | 55% | 44% | 22% | 17% | 7.7% |
| Number of Days Lost as Result of Eye Injuries: | | | | | |
| (a) Foreign Bodies | 242 | 204 | 217 | 241 | 28 |
| (b) Flashes | 277 | 185 | 227 | 173 | 361 |

CHART II

All lost time cases are not included, but cases on which there was unusual expense on hospitalization and cases on which compensation was paid either with or without lost time.

| | (1*) 1943 LAST HALF | 1943 FIRST HALF | 1944 LAST HALF | 1944 FIRST HALF | 1945 FIRST HALF |
|---|---|---|---|---|---|
| No. Cases | 2 | 8 | 102 | 58 | 25 |
| No. Lost Time | 2 | 8 | 87 | 57 | 14 |
| Hospitalized | 2 | 6 | 11 | 12 | 4 |
| Compensation Paid | 2 | 5 | 20 | 12 | 4 |
| Total Days Lost | 82 | 119 | 538 | 325 | 93 |
| Eq. Salary Lost | $ 736.00 | $ 978.02 | $5,108.70 | $3,135.16 | $990.10 |
| Compensation Paid | $4,138.55 | $2,939.58 | $3,711.48 | $ 728.50 | $171.08 |
| Eyes Lost | 1 total | 1 - 80% | 2 eyes - 30% | None over 10% | No visual loss (2*) |
| Hospital Bill | $ 147.17 | $ 118.15 | $ 601.90 | $ 334.10 | 0 |
| Doctor Bill | $ 275.00 | $ 188.00 | $1,870.00 | $ 416.00 | $51.00 |

1* One eye lost total before 1943 in construction of yard.
2* One case with low visual loss and hospitlization not yet settled.

Note: This chart is rather incomplete up to the last half of 1944. In comparing Chart I in the last half of 1944 there was only a total of 80 eye lost time accidents, 58 of which are included on this chart. In 1943 the first half of the year there was a total of 169 cases lost time accidents but two of these required added expense. This can be explained in the fact that the patients' eyes were covered with patches and the Navy would not allow them to return to work until their eyes were uncovered. The last half of 1944, the majority of eye cases had no patch to wear and if they required that, we considered them serious enough to be hospitalized in order that the recovery time would be cut down. This does not include doctor bills on cases which did not require hospitalization or on which no compensation was paid that were seen in the office at regular fee. One case of retinal detachment, occurring four months after a head injury, is not included in this chart. It was operated on, but the procedure was not successful.

We have followed a system, for these years, of employing no patches after removal of a foreign body and find that our results both in lost time and recovery of the wound appear to be improved over the method of patching the injured eye. The healing appears to be more rapid, there are fewer cases of corneal ulcer and the individual is not handicapped by the loss of stereopsis.

From the review of this paper, these conclusions can be drawn. In spite of the fact that atropine was used quite freely on cases with ciliary congestion in all age groups, glaucoma was not precipitated in a single case. We did observe one patient that had glaucoma that subsequently had a bilateral operation by another ophthalmologist in Evansville. This man had received no atropine and was seen but once at the shipyard, and, realizing he probably had glaucoma, was sent to the office where tonometer readings confirmed our suspicions. He was not a patient in this office. Also, from reviewing Charts I and II these things can be brought out: In 1944, employment increased 34.7 per cent over 1943. There was a 101 per cent increase of all accidents and a 98 per cent increase in the number of eye accidents over 1943. There was a 118 per cent increase in the number of lost time accidents, including all accidents. However, there was a decrease of 17 per cent in the number of eye lost time accidents in 1944 over 1943 with a greater decrease in the last half of 1944. There was, however, a 5 per cent increase in the days lost from foreign bodies in 1944 over 1943 with a 7 per cent decrease in the days lost from flashes in 1944 as compared to 1943. There was a decrease in the amount paid in compensation because of visual loss in 1944 as compared to 1943.

## FIRST AID FOR CHEMICAL EYE INJURIES

P. M. Van Arsdell
CHICAGO, ILL.

All foreign material which enters the eye is irritating and may be dangerous depending on the nature of the substance. The first rule for treatment consists of immediate copious irrigation directly in the eyes with plain water for at least five minutes. Prompt and plentiful water irrigation will save the sight of many eyes, and any one can and should institute this treatment at the time of the accident.

All chemicals entering the eye are immediately and potentially dangerous. Of those which contact the eyes directly, the concentrated acids and alkalis are most damaging; while for the lower concentrations the alkalis or alkali producing salts are far more dangerous to sight than the acids and acid producing salts. Alkalis penetrate deeply through a continuous chemical action with the tissues. The main reaction produces soap which continuously dissolves the eye tissue, and the longer it remains in contact, the deeper the penetration causing more extensive damage. Should solid particles remain after irrigation, a trained person should remove them as soon as possible.

There are a number of rules which should be kept in mind after the eyes have been thoroughly irrigated and in order not to undo the benefits of a thorough irrigation the following points should be learned by everyone.

(1) Untrained persons *should not* attempt any further treatment than the irrigation. Many well meaning individuals, at the sight of a person in great pain, naturally wish to do all they can to alleviate the suffering. If an eye kit containing argyrol

Benson & Associates, Inc., Chicago, Ill.
Reprinted from *Industrial Medicine*, April, 1947.

or yellow oxide of mercury ointment is at hand, they attempt medication which can be damaging to the patient.

(2) The best procedure after on-the-scene irrigation is to get the injured person to the plant dispensary or first aid room if such is provided, and lacking these facilities, to the nearest eye physician. This should not be delayed; for in all chemical contacts, speed and promptness of treatment are the essence of sight preservation.

(3) No ointments should be used on burns of any type (chemical, rays, or others) as they are impossible for the physician to remove and hinder his work at a later time.

(4) Do not attempt any neutralization such as acids for alkali contacts or vice versa. In the first place valuable time will be consumed in preparation of such solutions, and secondly it has been proved many times over that they are far too damaging to a normal eye to be effective when damage has already occurred.

(5) Whenever any disturbances of vision occur such as dimness, blurring, double vision, temporary or partial blindness an ophthalmologist should be consulted as soon as possible with full particulars ready as to type of work, presence of chemicals, or heat or any other pertinent facts with regard to possible chemical or light exposure.[23a]

Every day an appalling number of persons temporarily or permanently lose their sight through accidents. The joy of vision is tacitly accepted by the great majority who are not aware that their sight may be lost as the result of an accident. In industry alone, nearly 8,000 were blinded in one or both eyes in 1945. Authentic figures are apparently not available for the total number of eye accidents that occur, as a search has shown the number to range from 80,000[47a] to over 300,000[16,34,35] per year.

Chemical eye injuries have become an increasingly serious problem throughout many industries which are using more chemicals of a greater variety than ever before. In one plant alone the incidence of chemical eye injuries has more than doubled in a period of four years. Records were kept during the period from 1942 to 1945 inclusive and the following table summarizes the findings. Dispensary cases are those treated in the plant, and referred cases are those severe enough to warrant further treatment by an ophthalmologist.

TABLE I

| Year | Dispensary Cases | Referred Cases |
|---|---|---|
| 1942 | 663 | 85 |
| 1943 | 583 | 69 |
| 1944 | 926 | 82 |
| 1945 | 1394 | 97 |
| Total | 3566 | 333 |

From the foregoing table kept consistently, it is easily recognized that the incidence of chemical eye irritations and burns is far greater than the statistics would indicate.[26a]

Chemicals are responsible for a fairly large number of eye injuries either by direct contact with the material, or as a result of accumulation of the chemical within the body through inhalation or absorption through the skin.

The presence of chemicals in the working area is a potential eye hazard for every worker — whether he cleans the test tubes or directs research. Splashes, dusts, or vapors may at any time accidentally contact the eyes, with damage to the lids, conjunctiva, or cornea; and in the case of cumulative exposure, damage may occur to the optic nerve, the brain or the blood stream, which eventually may cause many types of disorders of vision.

The most easily recognized injuries are those occurring when splashes or

dusts enter the eyes. For both the injured worker and for those who wish to help him, only one cardinal first aid rule should be followed:

> Promptly wash the eyes with plenty of water for at least 5 minutes.*

After such treatment the remaining course of procedure is to see an ophthalmologist as soon as possible.[19]

Throughout the literature, the majority of the number of qualified persons who have written on the subject emphasize this procedure. Experimental animals in large numbers have given their eyes to prove these rules, and case histories of eye accidents due to chemical contacts reported in medical journals throughout the world have proved their efficacy.

Prior to 1920, it was customary to recommend neutralizing agents such as mildly acid solutions for alkali burns, and mildly alkaline solutions for acid burns *before* any other treatment was given, and this is still practiced at present, but only *after* the prompt and thorough washing has been done. Then it is given, if necessary, by the physician or other qualified persons. According to many reports on the subject, the best results, and that means saving some of the sight of the eye, are accomplished by means of the continuous water washing before any other treatment is applied.

From a purely theoretical standpoint, it would seem to the chemist that neutralization should be the proper course, but in actual practice such a procedure adds considerably to the pain, damage and irritation of an already highly inflamed, extremely sensitive area.[6a,26a] Even when undamaged, mildly acid or alkaline solutions do not feel too good to the eyes, but when they have been injured such a procedure immediately following an accident is comparable to pouring mild acids or caustics on open flesh wounds. Removal of foreign material by means of water washing leads to less permanent damage than use of chemical neutralization. A further advantage is gained by using water in that it may be speedily and effectively applied and no time is wasted as it would be if it were necessary to prepare the neutralizing solutions of the proper strength.

The involuntary reaction of the injured person when foreign material reaches the eye is first a spasmodic closure of the eyelids, followed almost immediately by a tendency to rub the eye. Both of these reactions must be combatted swiftly when chemicals reach the eyes. Dilution by water requires that the eyelids be held open by force if necessary and no rubbing, as that may work the chemical further into the recesses of the eye, making it necessary to use instruments to effect complete removal.

Ordinary dusts or particles, nature removes in her own way by means of tears, but when such chemicals as acids, alkalis, corrosive salts or organic chemicals enter the eye, the penetrating action is so swift that nature must be augmented by use of plenty of water from outside to wash away the damaging material. Also the pain caused by such entry is extremely great and the quick dilution helps to alleviate the distress. When the lids clamp shut as they often do from the spasm caused by the pain, the hands must be used to hold open the eyes so that effective washing may take place.

### ANATOMY OF THE EYE AND ITS RELATION TO CHEMICAL CONTACT

The lids, the conjunctiva, and the cornea are the external portions of the eye which can be damaged by the entry of chemical splashes or dusts. Corro-

*See references 1, 3, 5, 8, 10, 11, 14, 17, 20, 21, 23, 24, 25, 26, 26a, 28, 29, 30, 31, 32, 35, 36, 40, 44, 46, 47, 51, 53.

sion of any of these portions is extremely painful,[7] and unless promptly cared for may lead to serious damage.

When the outer portions of the lids are injured, swelling and closure may result. However, when chemicals are splashed into the eyes and there is a raw surface in contact with the conjunctiva, an adhesion of the lids and conjunctiva may occur unless proper precautions are taken during treatment of the chemical burn. This difficulty has been met with particularly when lime burns occur or when any caustic is splashed in the eyes and the two raw surfaces come together.[20] Medically, this condition is known as symblepharon.

The conjunctiva is the mucous membrane of the eye. It lines the inner surfaces of the eyelids, and also covers the forepart of the sclera and cornea. The portion lining the inner surfaces of the eyelids is thick and opaque having a considerable blood supply which is responsible for the red appearance when the lid is everted. The part lying over the eyeball is loosely connected to the bulb of the eye and is thin, transparent, and has only a small number of veins. Over the corneal portion, the conjunctival layer is epithelium only. Lymphatic tissue is liberally distributed over the conjunctiva ensuring a constant lubrication of the forepart of the eye.

Whenever this conjunctival layer is injured either by scratching or by chemical reaction, considerable pain ensues, and the reaction is to rub or to tightly close the eye. Conjunctivitis or irritation of the conjunctival layer occurs when foreign substances enter the eye causing a reaction of the tissues.

The cornea is the transparent portion of the eyeball through which the light penetrates to the deeper mechanism of the eye. In the case of chemical splashes, the thin conjunctival layer is easily penetrated by chemical reaction allowing the corrosive chemical to contact the cornea. Chemical action on the cornea may be extremely serious if not fatal to the vision as the action of the chemical may be either one of clouding the cornea to the point of opaqueness, or the cornea may be so severely corroded that the vision is either completely destroyed or extremely distorted. When such chemicals as lime and ammonia attack the cornea, penètration to the inner chambers of the eye is rapid and blindness may result from destruction of the inner structures.

Inhalation or absorption of certain types of chemicals affect the eyes in such ways as abnormal sensitivity to light, dimness of vision, and partial or total blindness which may or may not be permanent.[6,23,34]

As has been discussed, the eye is so constructed that the overlying conjunctiva and lids form a constantly bathed protection for the eye, but when this coating is contacted by strong chemicals for seconds only, this outer protective layer is penetrated and damage to the visual organs occurs. Permanent damage is controlled by three factors:[11]

(1) Strength and character of the chemical
(2) Length of time before initial treatment
(3) Type of initial treatment

## CHEMICALS IN RELATION TO EYE DAMAGE

Vapors, fumes, dusts, and splashes are all potential eye irritants whether they are derived from acids, bases, salts, or organic compounds. For splashes and dusts, the effects are immediate, while those from fumes and vapors may be cumulative, although it is difficult to make a sharp demarcation.

*Acids.* — Concentrated acids such as sulfuric, nitric, hydrofluoric, mixed acid, acetic, and picric withdraw water from the cells, and unite with the cellular proteins forming acid albuminates.[11]

TABLE II

| ACID | CONC. | TREATMENT | RESULTS |
|---|---|---|---|
| $H_2SO_4$ | 95% | None | Death from brain penetration |
| " | " | Neutralization after 10 seconds with 2% $NaHCO_3$ | Death from brain penetration |
| " | " | Washed after 10 seconds with plenty of water | Scarring cornea |
| " | 50% | None | Scarring of cornea |
| " | " | Neutralized after 10-15 seconds with 2% $NaCHO_3$ | Scarring of cornea |
| " | " | Washed with plenty of water | Normal eyes for all but one after 2-3 days. Only one of group showed opaque cornea |
| $HNO_3$ | 68% | None | Destruction of eyes |
| " | " | Neutralized immediately with 2% $NaHCO_3$ | Destruction of eyes |
| " | " | Water irrigation immediately | Marked scarring of cornea, but no destruction. After 15 seconds of $HNO_3$ contact, all eyes destroyed in spite of any treatment. |
| " | 30% | Neutralized by 2% $NaHCO_3$ | 100% of all eyes treated, degenerated |
| " | " | Washed only | 40% loss of eyes, 60% permanent corneal scars |
| " | 20% | None | Marked corneal scarring |
| " | " | 5-20 seconds contact | 60% cleared with either water or 2% $NaHCO_3$, but the water treated eyes cleared 2-3 days sooner than neutralized eyes |

The reaction is immediate and very painful as the heat of tear dilution causes severe burning with great destruction of the eye tissue.

Cosgrove and Hubbard,[11] in presenting their study on the effect of water washing of acid burns in contrast to neutralization, have shown the following effects from acids of varying strengths when they are placed in the eyes of rabbits. Table II summarizes their findings.

Highly concentrated acids usually produce some opacification with perforation of the globe or extensive symblepharon due to the contact of raw surfaces produced when the conjunctival layers are burned. The milder acid burns tend to recover uneventfully. As the concentration of acids is lowered, it has been found that the corneal epithelium has a highly protective effect against the penetration and damaging effects of acids. In a study of the $p$H levels at which eye damage occurs it was found that for certain acids, such as tannic and tungstic, at high $p$H were capable of producing lesions because of the greater protein affinity of the anion.[13] It was also shown that the following acids increase in the order shown in affinity of the anion for protein — hydrochloric, acetic, metaphosphoric, sulfosalicylic, picric, tungstic, and tannic. The maxi-

TABLE III

| ALKALI | CONC. | TREATMENT | RESULTS |
|---|---|---|---|
| NaOH[11] | | None | Degenerated eye followed by death |
| " | " | Neutralized with 2% acetic acid | Degenerated eye followed by death |
| " | " | Washed with plenty of water | 50% treated-cloudy cornea; 50% degenerated eyes |
| KOH[20] | 10 | None | Opacities in a few minutes |
| NaOH[20] | 5 | None | Opacities in a few minutes |
| " | 2 | None | Perforation of cornea in 7 days |
| " | 0.8 | None | Transient opacity for 3 days |
| $NH_4OH$[11,50] | 28 | None | Cornea destroyed |
| " | " | Neutralized with 2% acetic acid after 1 minute | Cloudy cornea |
| " | " | Water washing after 4 minutes contact | Transparent cornea |
| " | " | 5 minutes contact any treatment | Permanent scarring |
| $NH_4OH$[20] | 10 | None | Devastating lesions, corneal ulcer healing in 18 days, leaving densely opaque cornea |
| " | 7 | None | Opacity for 14 days, only slight residual clouding |
| " | 3.5 | None | Transient corneal opacity for 3 days |
| $Ca(OH)_2$[20] | 25 | None | Opacities within a few minutes |

mum lesions produced by each are essentially the same. From these studies it was also determined that certain specific features of acid eye burns contrasted markedly with those produced by alkalies.[13]

(1) Slower penetration and sharper demarcation of the lesion.

(2) Little tendency for the corneal epithelium to slough off.

(3) Little tendency to show exudate or leukocytic infiltration of fibrinous iritis.

(4) Little evidence of selective involvement of blood vessels.

(5) Marked tendency of the relatively opaque cornea to heal.

(6) A clinical course that is predictable within a few hours after injury.

These generalizations were determined with *p*H ranges from 1 to 9. As a rule the larger or higher molecular weight anions tend to penetrate less than the smaller or lower weight group. Definitive studies of this type are an aid in later treatment following first aid, and as so little data has been available on any but the most common acids, these experiments help to define the extent of damage that can be expected upon accidental eye contacts with the less common acids.

*Alkalis.* — Alkali contacts present a much more difficult problem as the injury to the eyes is more extensive and intensive as well as more rapidly destructive especially in dilute solutions. Alkalis combine with cell fats and albumins forming alkali albuminates and

soaps.[11] They penetrate much deeper into the epithelium and cause the tissues to die. As the action is extremely rapid, any treatment may not aid in preserving the vision.

The severity of alkali burns depends only slightly on the nature of the cation. The factors influencing the extent of the lesions are the concentration of the alkali,[20] the duration of contact, the *p*H of the solution, and the penetrability. Generally, sodium, potassium, and calcium hydroxides produce a porcelain white opacification of the cornea immediately after exposure. The final outcome with regard to sight for any of these is poor. The potassium hydroxide produces even more severe reactions than the sodium. The duration of exposure may naturally be prolonged if dusts of lime or lye lodge in the folds of the eye and are not removed. In this event the corneal opacities become progressively worse as there is no lessening of the concentration of the irritant, and the particles act as "feeders" to the irritation. Heat is also produced when concentrated alkalis or lime enter the eye, however lacrimation tends to lower the temperatures. In treatment, surface irrigations with water should be instituted immediately after the contact in order to prevent the development of corneal lesions. Speed in treatment is paramount as the chances of retaining sight are poor in any event.

Throughout the literature[6a,6,23,27,34,26a] a number of tables have been presented by numerous authorities showing the extent and type of damage to be expected from chemicals and contacts with compounds or inhalation of vapors. The notable exceptions in such tables are the lack of data on metallic and non-metallic salts which are in constant and increasing use throughout industry. These salts based on the reactions they produce on skin contact can be as damaging to the eyes as the acids and metals from which they are derived. A generalization, based on knowledge of reactions on skin contacts, would seem to apply even more specifically to the damage to be expected on eye contacts as the continuous moisture present on the eye area is even more conducive to dissociation of the ions than that of the skin which in comparison is relatively dry.

Aluminum chloride, antimony chloride, and other halogen salts have a strongly acid reaction on contact with moisture, hence, the eye damage in addition to that caused by the metallic ion, will also be in proportion to the amount of acid ions released in the eye tissues when the eye moisture is contacted.

Such salts as sodium cyanide, calcium acetate, potassium, sodium or ammonium sulfide in solution show an alkaline reaction; the damage to be expected on eye contacts with salts of this type would tend to follow the general pattern of weak alkali solutions as discussed previously. For the latter sulfide salts, in addition to the highly alkaline effect, the free hydrogen sulfide would also produce the light sensitivity and other irritations commonly encountered with low concentrations of this compound.

For either the acid or alkaline type of salt the reaction due to the *p*H of the solution should not be as great as that of the purely acid or purely alkaline contacts. However, the other compounds produced by the dissociation reaction would not only add their effect but also could enhance the irritation produced by the acidity or alkalinity of the stronger ion in solution. A further fact which may also markedly influence these effects is that particles of these salts when lodged in the eye tissues may be difficult to remove and the "feeder" action of such particles is the same as that discussed under alkaline effects. Table IV shows the salts which may be expected to be irritating to the eyes, although little work appearing in

TABLE IV

| COMPOUND | SKIN REACTION | EXPECTED EYE REACTION |
|---|---|---|
| Chloride salts of Ammonium Aluminum Antimony[4,12,15] Barium Bismuth Calcium Cobalt Copper Iron Nickel Lead Strontium Tin Zinc | Solutions are astringent and drying. Dry salts are hygroscopic and may produce burns. Antimony, copper, nickel, tin and zinc produce itching and burning followed by the development of dermatitis. | Irritating from acid reaction, concentrated solutions or crystalline salts may produce corrosion and burning of eye tissues. Conjunctivitis and corneal lesions can be expected from those producing dermatitis and skin burns. |
| Arsenic | Absorbed through the skin. Low concentrations produce inflammation and later pigmentation with eczema. | Highly irritating to mucous linings of eye area. |
| Barium | Eczema and cyanosis, irritating to sweat glands may bleach hair and eyebrows. | Highly irritating, conjunctivitis and corneal lesions might result from dry salt contact. |
| Cadmium[38] | Irritation and inflammation. Ulceration occurs in nasal septum on inhalation. | Irritation of conjunctiva, prolonged contact might damage cornea considerably. |
| Mercuric (corrosive sublimate) | 1-5% solution produce irritation, drying, and corrosion. Absorption from the intact skin may be sufficient to produce general poisoning. | Highly irritating on initial contact with even low concentrations. Mercuric salts have a high protein coagulating power hence the eye might be permanently clouded even on slight contact with a highly concentrated solution. |
| Mercurous | No reported effect. | Might produce a slight irritation. However mercurous salts are practically insoluble, and effects would be negligible. |
| Nitrate Salts of Al, Bi, Ca, Cu, Co, Fe, Pb, Mg, Ni, K , Na, Sr, Ba, Cd, and Hg | Astringent and drying in high concentration in crystalline form. May produce dermatitis if contact is prolonged. | Irritating and may produce conjunctivitis. |
| Silver[18,41,43] | Burns skin and mucous tissues turning them gray and finally black after exposure to light. Silver compounds under the skin may cause gray to black skin coloration. | Produces burns on eye tissues which may penetrate very deeply. When silver salts enter the body silver may be preferentially deposited in the whites of the eyes changing the color to mouse gray. |

the literature corroborates this theory which is based solely on the skin reactions which have been fairly exhaustively studied.

Sulfate salts act as astringents and irritants[15] to the skin. Cobalt and nickel cause dermatitis. The eye reactions can be expected to follow this general pattern, conjunctivitis occurring in those producing dermatitis and extreme irritation and burning from those salts having astringent and irritating effects on

the skin. Zinc sulfate has been reported to cause conjunctivitis.

Halogen salts follow the same general pattern as those reactions outlined for the chlorides only with the possible exception that as the molecular weight of the halides increases,[52] the solubility also decreases.

Carbonate salts with the exception of the alkali metals are fairly insoluble. On the skin they produce a drying effect, the alkali and alkaline earth salts react as strong bases, hence the effect on the eye contact would parallel that for the lower alkali concentration previously discussed.

Chromates,[2] either dusts or vapors, on the skin produce dermatitis of an ulcerative type known as "chrome holes." On contact with eye tissues, irritation, conjunctivitis, irritation of the mucous membranes, lacrimation, chemical burns, and eyelid inflammations are produced. Generally the immediate reaction is not so serious, but vapors, dusts, and sprays from the chromates, in view of the fact that contact is over a prolonged period, are in daily contact with these tissues and in many instances have produced all of the above reactions. The length of time necessary for such manifestations depends largely upon the degree of susceptibility of the individual.

Sulfide salts on the skin in high concentration loosen the hair and soften the skin in much the same manner as alkaline solutions. In the eyes the effect is one of alkali combined with hydrogen sulfide which produces conjunctivitis and a high sensitivity to light.

As soon as eye contact with any chemical occurs, water washing should be the rule as promptly as possible. Following the washing, a visit to the ophthalmologist should be insisted upon, as no chances should ever be taken with the eyesight. As a further aid in preserving sight an accurate description of the type of contact should be given in order to aid the doctor as much as possible in instituting the correct treatment. In this way many complications may be avoided. For convenience, the following tables have been divided into three groups, inorganic, organic, and miscellaneous. The eye reactions have also been set forth, as far as information is available into internal, to cover the more immediate effects of contacts; external, to cover a number of reactions which seem to more specifically affect the lids; and the chronic or permanent effects, which occur either quickly or are long delayed as a result of continuous inhalation or absorption of the chemicals. Damage to the eyes may be caused by inhalation, ingestion, or contact. With many of the organic and a number of the inorganic compounds the effects in the beginning are due to vapors or dusts entering the eyes from the atmosphere, but the more chronic and serious reactions result from inhalation of the vapors and gradual absorption from the lung tissues over a considerable period. Many chemicals act too swiftly for the ultimate damages to the eyes to be determined, especially on ingestion, but the others manifest themselves very dramatically at the beginning by their effect on the optic nerve.

In some groups such as the halogenated hydrocarbons, certain interesting conclusions may be drawn showing, especially in the gaseous series such as methyl chloride, methyl bromide, and methyl iodide, that the toxic reaction tends to become more pronounced as the molecular weight increases; however, it is difficult in most instances to make any generalizations as all of the possible types of damage have either not been tabulated in the literature or are presently unknown. Many of the chemicals have been adopted for general use so recently that studies of chronic effects have not or cannot yet be evaluated.

Generally, whenever new compounds are introduced, careful checks for eye damage should be maintained. While animal studies may not indicate damage, the differences in body chemistry may be sufficient for toxic effects to be noted in the higher species and be completely absent in the lower.

The purpose of this article has been to stress the importance of the use of water in copious amounts as the most effective first aid measure. This simple procedure is effective and will minimize the chances of blindness and much extended duration of pain when chemicals are accidentally contacted in the eye area.

The list of chemicals known to affect the eyes is constantly growing. Physiological studies of industrial chemical contacts are continuously broadening in scope as new chemicals are taking their places in manufacturing procedures. With greater knowledge not only being derived from the clinical laboratory, but also from case histories, the ultimate knowledge of effects of chemicals on the body should eventually be recorded. When this is accumulated and even before, greater emphasis on safe handling of all chemicals should become part of the educational program for industry. Ideally all hazards should be eliminated, but until such a goal is reached first aid measures and adequate protection must continually be brought to the attention of every one who uses any type of chemical for any purpose.

## REFERENCES


1. Alvis, E. O.: J. Indiana M. A., 27:335 (1934).
2. Anon.: J.A.M.A., (Belgian Letter) 109: 1922 (1937).
3. Anon.: J.A.M.A.,Queries and Minor Notes 119:1467 (1942).
4. Barillet, F.: L'Industrie Chimique, v. 29, No. 332, p. 73 (1942).
5. Barkan O. and Barkan, H.: J.A.M.A., 83: 1567 (1924).
6. Bridges, C. D.: Job Placement for the Physically Handicapped, New York, McGraw Hill, 1946, p. 105.

6a Carpenter C. P. and Smyth, H. F., Jr.: Am. J. Ophth., v. 29, No. 11, p. 1363 (1946).

7. Cashell, G. W., Shay, H. B., and Bodenham, D. C.: Brit. J. Surg., v. 31, No. 124, p. 373, (1944).
8. Cogan, D. G.: J.A.M.A., 122:435 (1943).
9. Cogan, D. G. and Grant, W. M.: Arch. Ophth., 33:106 (1945).
10. Cosgrove, K. W.: Safety Eng., 54:214 (1927).
11. Cosgrove, K. W., and Hubbard, W. B.: Ann. Surg., 87:89 (1928).
12. Feil, A.: Presse med., 47:1133 (1939).
13. Friedenwald, J. S., Hughes, W. F., Jr. and Herrmann, H.: Arch. Ophth., 35:98 (1946).
14. Gibson, A. and Carlisle, J. M.: Sight Saving Rev., v. 15, No. 4, p. 220 (1945).
15. Gocher, T. E. P.: Northwest Med., v. 44, No. 92, p. 467 (1945).
16. Greenburg, L.: National Safety News, 41: 26 (1940).
17. Hannum, J. E.: Ind. & Eng. Chem., 19:817 (1927).
18. Harker, J. M. and Hunter, D.: Brit. J. Dermat., 47:441 (1935).
19. Hiestand, C. V. and Atkinson, W. B.: Kentucky Med. J., 29:31 (1931).
20. Hughes, W. F., Jr.: Arch. Ophth., v. 35, No. 4, p. 423 (1946).
21. Kaplan, I. D.: Gigiena Truda i. Tek Bezopasnosti, No. 1, p. 52 (1936).
22. Koopman: Deutsche Ztschr. f. d. ges. gerichtl. Med., 36:383 (1936).
23. Kuhn, H. S.: Industrial Ophthalmology, St. Louis, C. V. Mosby, p. 269 (1944).

23a Kuhn, H. S.: Am. J. Nursing, 47:24 (1947).

24. Lukianova, E.: Sovet. vestnik oftal., 6 pt. 1, 126 (1935).
25. Machlin, I. M.: Vestnik, oftal., 18 pt. 2. 193 (1941).
26. Mann, I., Pirie, A., Pullinger, B. D.: Am. J. Ophth., v. 29, No. 10, p. 1215 (1946).

26a McLaughlin, R. S.: Am. J. Ophth., v. 29 No. 11, p. 1355 (1946).

27. McNally, W. D.: Toxicology, Indust. Med (1937).
28. Middleton, A. B.: Illinois M. J., 67:35 (1935).
29. Moss, O. W.: New Orleans M. and S. J. 89:302 (1936).
30. North, E P. and Jones, V L.: J. Missouri M. A., 26:51 (1929).

31. Oaks, L. W.: Am. J. Ophth., 28:370 (1945).

32. Osburn, J. M.: California & West. Med., v. 62, No. 1, p. 22 (1945).

33. Rabinowitch, I. M.: Canad. M. A. J., 52: 345 (1945).

34. Resnick, L.: Eye Hazards in Industry, Columbia Univ. Press (1941).

35. Ridley, F.: J. Indust., Hyg. & Toxicol., 21: 439 (1939).

36. Rubin, H. H. and Arieff, A. J.: J. Indust. Hyg. & Toxicol., 27:123 (1945).

37. Sabata, J.: Oft. Sbornik, 4:92 (1929); Abs. Am. J. Ophth., 13:748 (1930).

38. Schwartz, L. and Otto A.: Ztschr. f. Hyg. u. Infektionskr., 104:364 (1925).

39. Schwartz, L.: Dermat. and Syph., v. 50, No. 1, p. 25 (1944).

40. Sigrist, A.: Ztschr. f. Augenh., 43:176 (1920), Abs. J. Indust. Hyg. & Toxicol., 2:199 (1920).

41. Subal: Wien. klin. Wchnschr., v. 35, No. 21, p. 493 (1922).

42. Tabershaw, I. R., Fahy, J. P. and Skinner, J. B.: J. Indust. Hyg. & Toxicol., 26:328 (1944).

43. Thies, O.: Zentralbl., f. Gewerbehyg., N. S. 3:315 (1926).

44. Thies, O.: Zentralbl. f. Gewerbehyg., N. S. 5:83 (1928).

45. Townsend, J. G.: Sight Saving Rev., 13: 219 (1943).

46. Uhde, G. I.: Am. J. Ophth., 29:1090 (1946).

47. U. S. Dept. of Labor, Div. of Labor Standards, Washington, D. C. (1946).

47a Van Atta, F. A.: Private Communication, (Dec.) 1946.

48. Van Ordstrand, H. S. Hughes, Robert, DeNardi, J. M. and Carmody, M. G.: J. A. M. A., 129:1084 (1945).

49. von Oettingen, W. F.: Suppl. to Occupation & Health, 1-6, Int. Labor Office, October, (1939).

50. Waardenburg, P. J.: Abst., J.A.M.A., 83: 802 (1924).

51. Walker, S., Jr., and Auten, H. L.: J.A.M.A., 116:1356 (1941).

52. White, R. P.:Occupational Affections of the Skin, New York, P. B. Hoeber, (1929).

53. Whitney, L. H.: Indust. Med., 14:336 (1945).

54. Wolff, E.: Brit. J. Ophth., 10:196 (1926), Lancet, 1:1212 (1926).

## CONTINUING BIBLIOGRAPHY ON OCCUPATIONAL EYE PROBLEMS

ADAM, A. L., AND KLEIN, M. *Electrical Cataract, notes on a case and a review of the literature.* Brit. J. Ophth., 29:169, April, 1945.

ANON. *Standing Orders for Nurses in Industry.* Amer. Mutual Liability Ins. Co., Indus. Nursing, 4:15-22, Sept. 1945.

ANON. *Vision Checking Speeded By New Sight Screener.* Scient. Am., 174:268, June, 1946.

ANON. *Eyes Saved from Chemicals.* Science News Letter, 50:311, Nov. 16, 1946.

ANON. *Visual Standards Should be Raised for Post War Jobs.* Scient. Am., 172:234-5, April 1945.

ANON. *Industrial Medical Service—for the smaller plant.* Indust. Med., 15:192-194, March, 1946.

BAUER, L. *Eye Injuries Hurt More.* Safety Eng., 92:18020,50-51, Oct. 1946.

BRODSKY, B. S. *The Use of 70 Per Cent Alcohol in Combating Sequels of Prolonged Retention of Lead from Indelible Pencil.* Sovet. Med., (Nos. 4-5) 8:28, 1944.

CSAPODY-MOCSY, MARION. *Test Charts for Women's Needlework.* Klin. Monatsbl. f. Augenh., 109:251, March-April, 1943. (Abstract Am. J. Ophth., 29:1051, Aug. 1946.)

CHECHIK-KININA, E. *Treatment of Perforating Wounds and Burns of the Eye with Lysozyme.* Vestnik oftal., 24:42, 1945. (Abstract Arch. Ophth., 36:5,625, Nov. 1946.

COGAN, D. G. AND GRANT, W. M., *Keratitis Due to n-butyl Alcohol.* Arch. Ophth., 34: 248, Sept. 1945.

CRUTHIRDS, ARCHIE EDWARD. *Importance of Sulfhydryl in the Treatment of Corneal and X-Ray Burns.* Am. J. Surg., 72:500-509, Oct. 1946.

HINSHAW, JOSEPH H. *Legal Ophthalmology,* Am. J. Ophth., 29:111, Sept. 1946.

INCIARDI, J. A. *Technic of Screening Eyes in Mass Examinations.* Eye, Ear, Nose & Throat Monthly, 24:419-422, Sept. 1945.

MANN, IDA, AND ARCHIBALD, DOROTHY. *A Study of a Selected Group of Women Employed on Extremely Fine Work.* Brit. M. J., 1:378-90, March 18, 1944.

MINTON, *J. Ophthalmic Problems and Visual Standards.* Brit. J. Ophth. 30:298-304, May, 1946.

MITERSTEIN, B. AND KORNBLUETH, W. *Transplantation of Mucous Membrane (Denig operation) in Severe Burns.* Harefuah, 29: 152, Oct. 1, 1945.

MUMFORD, E. W. *Ophthalmological Guidance for Nurses in Industry.* N.S.P.B., Pub. No. 420, 1945.

NELSON, J. H., *Ideal Seeing Conditions. The Study of the Human Visual System as a Basis for Prescribing Lighting.* Brit. J. Indust. Med., 2:224-232, Oct., 1945.

ORR, H. C. *Ray Cataracts.* Tr. Ophth. Soc. U. Kingdom, (1944) 64:249-252, 1945.

PAVISIC, Z. *Early Transplantation of Mucous Membrane from the Mouth and Conjunctiva in Burns of the Eye.* Ophthalmologica, 108: 297-304, Dec., 1944.

PERERA, G. A. *Sympathetic Ophthalmia Following Subjunctival Rupture of the Eyeball.* Am. J. Ophth., 28:186, Feb., 1945.

PETER, A. L., AND ROSEN, E. *The Importance of Injecting Air Into Tenon's Capsule Even in Cases of Foreign Bodies Within the Eyeball.* Am. J. Ophth., 28:1140-1144, Oct., 1945.

POOLE, F. E. *Selective Placement of Workers: A Medical Man's Viewpoint of Workers:* Soc., 44:1060, Oct., 1945.

SANDERS, W. *Treatment of a Perforating Corneal Wound with Penicillin and Sulfadiazine.* J.A.M.A., 127:197, Feb. 17, 1945.

SCHWEISHEIMER, W. *Eye Troubles of Machinists.* Ind. Arts and Voc. Educ., 34:154, April, 1945.

SCHWEISHEIMER, W. *Eye Troubles of Arc Welders.* Ind. Arts and Voc. Educ., 34:205-206, May, 1945.

SHAH, M. A. *Glass Blowers Cataract.* Indian M. Gaz., 80:400-402, Aug., 1945.

STEWART, D. STENHOUSE. *Some Observations On a Tendency to Near-Point Esophoria and Possible Contributory Factors.* Brit. J. Ophth., Jan., 1945.

STUMP, N. F. *Spotting Accident-Prone Workers by Vision Tests.* Factory Manag. and Maint., 103:109-112, June, 1945.

STUMP, N. F. *A Statistical Study of Visual Functions and Industrial Safety.* J. Appl. Psychol., 29:467-70, Dec., 1945.

TIFFIN, J. *Vision for the Job.* Personnel, 22: 270-271, Jan., 1946.

TISHER, P. W. *The Opportunity for Prevention of Blindness in Industry.* Connecticut M. J., 10:382, May, 1946.

U. S. ARMY M. DEPT. *Corrective Lenses in Welder's Goggles.* Bul., 6:186-188, Aug., 1946.

WOODBURY, D. O. *Bringing Eye Tests Up to Date.* Science Digest, 19:11-15, April, 1946.

WOLF, ERNEST. *Effects of Exposure to Ultra Violet Light on Visual Thresholds.* Proc. Nat. Acad. Sci., 31:236-241, Aug., 1945.

# OFFICIAL PROGRAM

# TRANSACTIONS

## AMERICAN ACADEMY
*of*
## OPHTHALMOLOGY
*and*
## OTOLARYNGOLOGY

Vol 51 No 6

**JULY - AUGUST**
*1947*

Published bimonthly, February, April, June, August, October, and December, by the Douglas Printing Company, 109 North 18th Street, Omaha 2, Nebraska, for the American Academy of Ophthalmology and Otolaryngology. Editorial and business office, 100 First Avenue Building, Rochester, Minnesota. Subscription price $3.00 yearly to members, $6.00 yearly to non-members. Entered as Second-Class Matter November 28, 1938, at the Post Office at Omaha, Neb., under Act of March 8, 1879.

**ANNUAL MEETING OCTOBER 12-17, 1947, CHICAGO**

* * *

# I. INTRAOCULAR FOREIGN BODIES IN SOLDIERS

HELENOR CAMPBELL WILDER
WASHINGTON, D. C.
BY INVITATION

RETAINED foreign bodies were found in 731 of 3,882 eyes of soldiers studied at the Army Institute of Pathology during World War II. Although nearly all of these foreign bodies were introduced during combat and training, a few were the result of pre-induction injuries, with recent exacerbations of pain and inflammation leading to enucleation. All globes with penetrating wounds were examined by x-ray and were searched for foreign bodies. In many instances the particles were so small or so deeply embedded in organizing hemorrhage or inflammatory membrane that they were not recovered from the gross specimens and only became visible on microscopic examination. These could not be subjected to magnet test, but sections containing them were stained with Prussian blue. Those which gave a positive iron reaction were classified as probably magnetic, although the degree of their magnetic quality could not be determined; whereas those giving no reaction were regarded as nonmagnetic. The Federal Bureau of Investigation assisted in the more specific identification of many of the foreign bodies by spectroanalysis. In many eyes the particles were multiple (figs. 1, 2), and in a few both magnetic and nonmagnetic materials were found. The following table demonstrates a preponderance of retained nonmagnetic foreign bodies in eyes of soldiers who, in all but a few instances, had sustained military injuries.

INTRAOCULAR FOREIGN BODIES

| | |
|---|---|
| Ferrous | 273 |
| Nonmagnetic | 452 |
| Both | 6 |

Ferrous foreign bodies (figs. 2, 3), although outnumbered by nonmagnetic ones, formed the largest relatively homogeneous group. Some were more strongly magnetic than others, and it is possible that many which were weakly magnetic on direct contact may have been clinically nonmagnetic.[2] Siderosis, with deposits in the cornea (fig. 4), lens (fig. 5), filtration angle, iris, ciliary epithelium (fig. 6), and retina (figs. 7, 8), occurred in 27 eyes with retained ferrous particles. In one eye a rather extensive iron reaction was noted only 23 days after injury, but in this case many ferrous particles provided multiple foci of diffusion. In some instances in which ferrous foreign bodies had been retained for much longer periods, early encapsulation by scar tissue (fig. 9) had prevented dissemination of the metal, and only a local iron reaction was obtained with Prussian blue. In hematoxylin and eosin sections, basophilic staining, as well as pigmentation, was frequently observed around the sites of the ferrous material (fig. 10), but rarely around other metals and never to so marked a degree.

Army Institute of Pathology
Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

The nonmagnetic foreign bodies comprised a variety of materials. Among the metal substances, copper, brass, lead, nickel and zinc alloy were identified; among the nonmetallic, glass, vegetable matter, eyelashes, rock, shale and clay predominated.

Copper and brass were most numerous among the nonmagnetic metals. Unfortunately chalcosis could not be demonstrated by special staining, and therefore it was impossible to study the intraocular distribution of copper.

Many nonmagnetic foreign bodies were readily identified by gross or microscopic examination more specifically than by magnet test. Copper fragments (fig. 11) could be recognized both in microscopic sections and on gross examination by their characteristic color. Lead (fig. 12), in comparatively large pieces, was sectioned in the celloidin blocks with little damage to the microtome knife. Glass, of course, was easily identified on gross examination, and vegetable matter and eyelashes (fig. 13) on microscopic examination. Nonmagnetic foreign bodies were found not only as primary missiles or in association with magnetic material, but also as material retained after extraction of a magnetic missile or as implants along the path of a primary missile which had passed completely through the eye. Eyelashes were the most common of the secondary foreign bodies. In one instance a fragment of chrome green paint remained in the choroid, although the piece of metal from the bumper of a jeep which it had camouflaged had been extracted by magnet. Intraocular particles of stone, clay, and vegetable matter frequently were retained after land mine explosions. In severe head injuries, skin (fig. 14) and bone (fig. 15) were sometimes implanted and behaved as inactive foreign bodies, unlike conjunctival epithelium (fig. 16) which finds the interior of the globe a fertile field for growth.

Some of the eyes in this series were removed because of the severity of the initial injury and before secondary pathologic changes had appeared. In these, partial evisceration, disorganization, massive intraocular hemorrhage (fig. 17), and collapse of the globe were the kinds of damage which led to early enucleation. All stages of organizing hemorrhage were present in eyes removed after longer postoperative periods (figs. 18, 19).

Abscesses were frequently seen around the sites of foreign bodies of various types, almost invariably around vegetable matter (fig. 20). In the vitreous chamber, the subsequent formation of a pyogenic membrane resulted in retinal detachment (fig. 21) and phthisis bulbi (fig. 22). Glaucoma, a less frequent sequela of intraocular foreign body, was present in association with siderosis bulbi. It also occurred occasionally when the missile lodged in the anterior chamber and the inflammatory process was confined to the anterior segment (fig. 23). Foreign body granulomas appeared around all kinds of foreign bodies, but the formation of giant cells seemed to be stimulated particularly by eyelashes (fig. 24) and vegetable matter (fig. 25). In some instances large syncytial cells were arranged around implanted hairs in such a way as to suggest proliferated sheath cells. Phagocytized melanin granules were usually present in them, however, confirming the opinion that they were more probably true histiocytic giant cells. The tendency of vegetable matter to stimulate a giant cell reaction is evidenced in a case in which cotton fibers and carbon particles were introduced into the vitreous chamber by a dynamite explosion. Endophthalmitis developed and the eye was enucleated six weeks following in-

jury. Incarcerated in an inflammatory cyclitic membrane the cotton fibers (fig. 26) were all surrounded by giant cells, whereas none appeared around the carbon particles (fig. 27).

Sympathetic uveitis developed in 5 patients with retained intraocular foreign bodies. In one instance, lead (fig. 28) had been introduced in combat two months before enucleation and in another, wood (figs. 29, 30) had been introduced in a pre-induction injury thirty-six years before enucleation. In the latter, following the old corneal perforation, the eye retained a small amount of vision until three months before enucleation, when it became inflamed and blind. Uveitis appeared in the sympathizing eye one month before enucleation. One eye retained a fragment of glass until enucleation one and one-half months after injury. One was removed three months following the introduction of an unidentified nonmagnetic foreign body. In the fifth case the time interval between injury and enucleation was not given, but about one week after injury, severe uveitis was reported to have developed in the sympathizing eye. The foreign body was magnetic.

A variety of infections followed introduction of foreign material into the eye. Masses of cocci (fig. 31), bacilli, and unidentified fungi (fig. 32) were sometimes seen in vitreous abscesses. Saprophytic fungi were frequently found in and around wood particles (figs. 33, 34, 35).

## COMMENT

A wide variety of foreign bodies was found in eyes of soldiers following injuries in training and combat. Of these approximately 37 per cent were magnetic, 62 per cent nonmagnetic, and 1 per cent mixed. This apparently confirms previous observations[1,2,3] that in military injuries nonmagnetic intraocular foreign bodies predominate. They do, undoubtedly, in eyes submitted for pathologic examination, but many instances of successful magnet extraction do not come to the pathologist's attention; and, even in eyes that are enucleated, nonmagnetic foreign bodies are often secondary to ferrous missiles which may have been extracted. The infections and reactions associated with the different types of foreign bodies ran the gamut from staphylococcus infection, resulting in purulent panophthalmitis, to the mild reactive inflammations, associated with siderosis bulbi and intraocular hemorrhage without infection. The formation of giant cells was particularly abundant around vegetable matter and eyelashes. Of particular interest were 5 cases of sympathetic uveitis associated with retained nonmagnetic material in four and with a retained ferrous foreign body in one. Phthisis bulbi was a common end result, and glaucoma was comparatively rare, as in any series of penetrating wounds.

I wish to thank Lawrence P. Ambrogi and Eleanor V. Paul for x-rays, careful gross examinations and magnet tests, as well as for their preparation of sections which always included the foreign body sites.

## REFERENCES

1. Pokrovsky, A. J.: Characteristics of intraocular foreign bodies in military and civil injuries, and the technic of their removal, Vestnik Oftal., 22: 4, 1943.
2. Stallard, H. B.: War surgery of eye; analysis of 102 cases of intra-ocular foreign bodies, Brit. J. Ophth., 28:105-135 (March) 1944.
3. Dansey-Browning, G. C.: Ophthalmic treatment in the field, 1943, Brit. J. Ophth., 30:26-35 (Jan.) 1946.

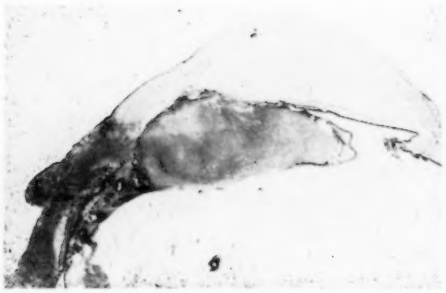


FIG. 1. Multiple ferrous foreign bodies in cornea, ciliary region, lens, and vitreous. Scar of entrance of intraocular foreign bodies at the limbus. Enucleation twenty-three days after injury in action by bomb explosion. A.I.P. Neg. 97290.

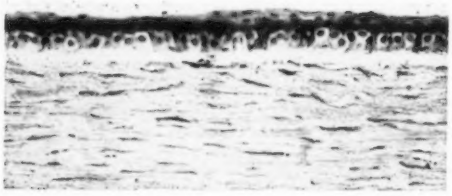


FIG. 4. Siderosis bulbi, cornea. Iron pigment phagocytized by the cells of the interlamellar spaces. Enucleation two years after injury. A.I.P. Neg. 100247.

FIG. 5. Siderosis bulbi, lens. Iron pigment in an anterior capsular cataract. Enucleation nine months after injury. A.I.P. Neg. 100248.

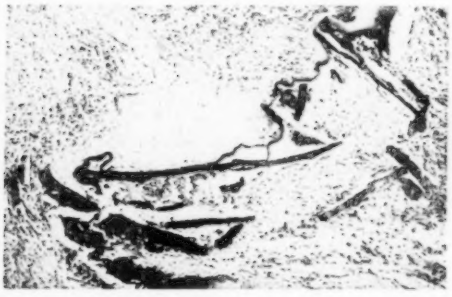


FIG. 2. Multiple ferrous foreign bodies in an inflammatory cyclitic membrane. Magnet extraction of one steel particle was performed on the day of injury six weeks before enucleation. A.I.P. Neg. 99319.

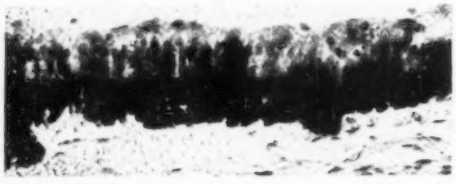


FIG. 6. Siderosis bulbi, ciliary epithelium. Iron pigment in the normally nonpigmented inner layer. Enucleation eleven months after injury. A.I.P. Neg. 100246.

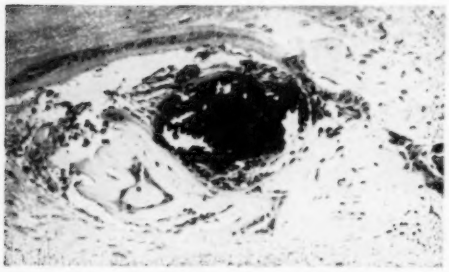


FIG. 3. Ferrous foreign body in lens capsule incarcerated in an inflammatory cyclitic membrane. Enucleation one year following a penetrating wound by a screw driver. A.I.P. Neg. 99322.

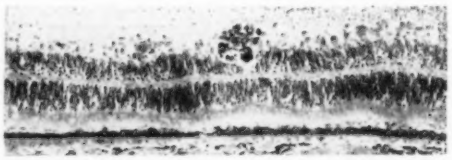


FIG. 7. Siderosis bulbi, retina near equator. Iron pigment around a retinal vessel. Enucleation two years after injury. A.I.P. Neg. 99252.

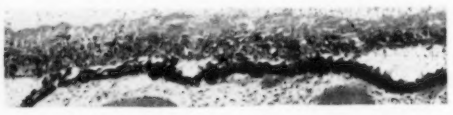


FIG. 8. Siderosis bulbi, peripheral retina. Iron pigmentation and atrophy. Enucleation two years after injury. A.I.P. Neg. 99250.

FIG. 9. Scar tissue around the site of a ferrous foreign body in the posterior chamber preventing the dissemination of iron pigment. Enucleation two months after injury. A.I.P. Neg. 97284.

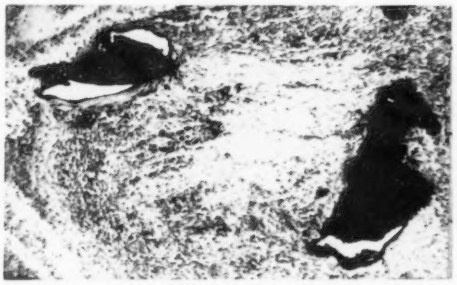


FIG. 12. Lead in inflammatory tissue in the vitreous chamber. Enucleation five months after injury. A.I.P. Neg. 99318.

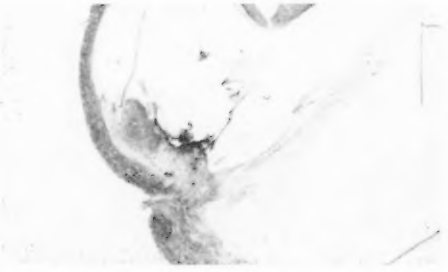


FIG. 10. Iron pigment and basophilic staining around the site of a ferrous foreign body in the vitreous chamber. Enucleation forty-one days after injury. A.I.P. Neg. 97286.

FIG. 13. Vegetable matter and cilium in inflammatory tissue in the vitreous chamber, following a land mine explosion. Enucleation two months after injury. A.I.P. Neg. 97289.

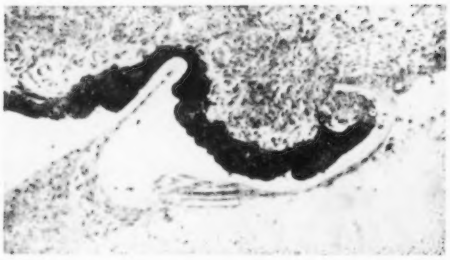


FIG. 11. Copper wire in early anterior capsular cataract. Lens capsule recoiled outward at site of entrance. Chronic iridocyclitis with posterior synechia. Enucleation six days after injury. A.I.P. Neg. 99321.

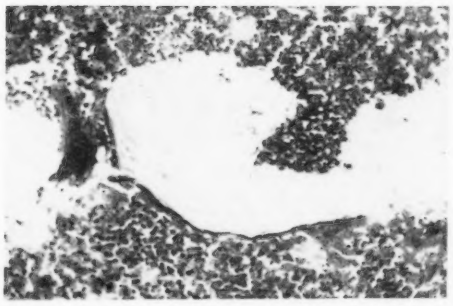


FIG. 14. Epidermal epithelium with keratic debris in a vitreous abscess. A zinc alloy primary missile and hairs were also retained until enucleation four days after injury. A.I.P. Neg. 97361.

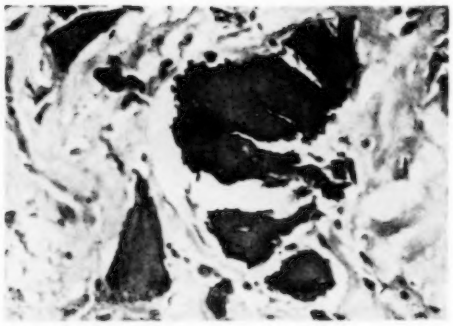


FIG. 15. Implanted bone incarcerated in a cyclitic membrane following a skull wound. Enucleation nine months after injury. A.I.P. Neg. 97356.

FIG. 16. Hyperplastic conjunctival epithelium around a thorn incarcerated in an inflammatory cyclitic membrane. Enucleation four months after injury. A.I.P. Neg. 97296.

FIG. 17. Expulsive hemorrhage following a penetrating wound at the limbus by a copper fragment retained until enucleation nine days after a booby trap explosion. A.I.P. Neg. 84004.

FIG. 18. Beginning organization of hemorrhage in the track of a lead foreign body, extending from the corneal wound of entrance to the chorioretinal wound of lodgement at macula. Enucleation sixteen days after injury from gun discharge. Magnet extraction attempted on the day of injury. A.I.P. Neg. 83982.

FIG. 19. Organized hemorrhage forming a traction band between the scar of entrance at the limbus and the chorioretinal scar of lodgement of a lead foreign body. Cicatricial distortion of globe. Enucleation six months after injury. A.I.P. Neg. 97836.

FIG. 20. Abscess around a vegetable fragment in the angle of the anterior chamber and in its track through the lens. Enucleation twenty-four days after injury. A.I.P. Neg. 97293.

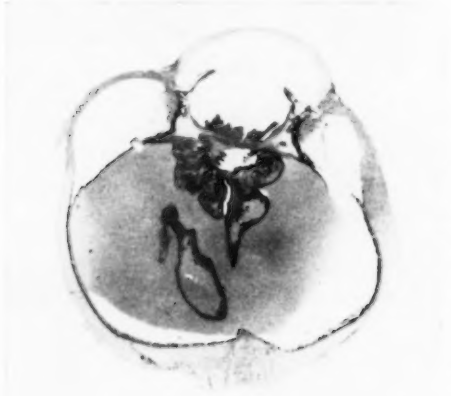


FIG. 21. Vitreous abscess at the site of a brass foreign body. Detachment of retina by a pyogenic cyclitic membrane. Early phthisis bulbi. Enucleation two months after injury. A.I.P. Neg. 99314.

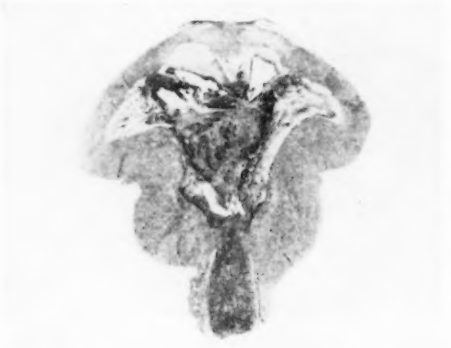


FIG. 22. Phthisis bulbi following lodgement of vegetable matter in the vitreous chamber eleven years before enucleation. A.I.P. Neg. 99316.

FIG. 23. Chronic glaucoma following lodgement of a vegetable matter in the anterior chamber fourteen years before enucleation. A.I.P. Neg. 99313.

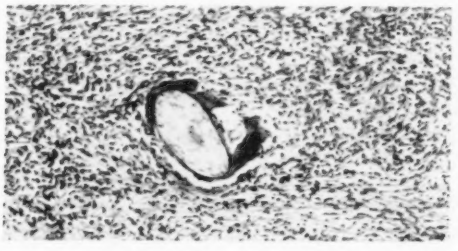


FIG. 24. Giant cell reaction to cilium in an inflammatory cyclitic membrane. Enucleation five weeks after injury. A.I.P. Neg. 97363.

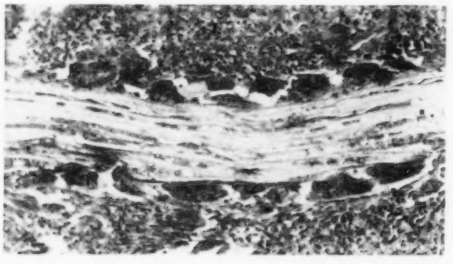


FIG. 25. Giant cell reaction to vegetable matter in an inflammatory cyclitic membrane. Enucleation two months after injury. A.I.P. Neg. 97288.

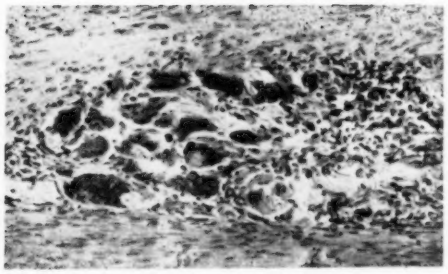


FIG. 26. Giant cell reaction to cotton fibers in an inflammatory cyclitic membrane. Enucleation six weeks after dynamite explosion. A.I.P. Neg. 99317.

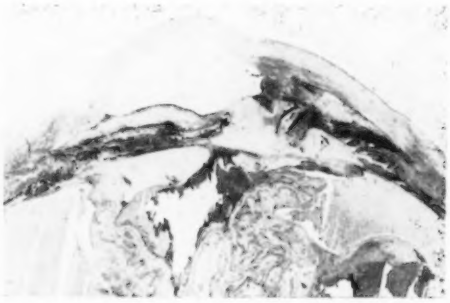


FIG. 29. Recent sympathetic uveitis in an eye with wood foreign bodies retained thirty-six years after a penetrating wound through the cornea. A.I.P. Neg. 97358.

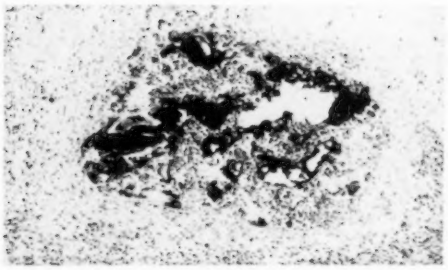


FIG. 27. Same eye as in fig. 26. No giant cell reaction to carbon particles in the cyclitic membrane. A.I.P. Neg. 99320.

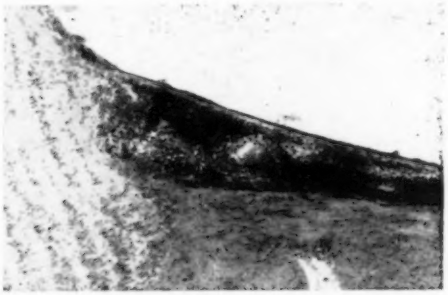


FIG. 30. Same eye as in fig. 29. Choroid at optic disc. Sympathetic uveitis. A.I.P. Neg. 97362.

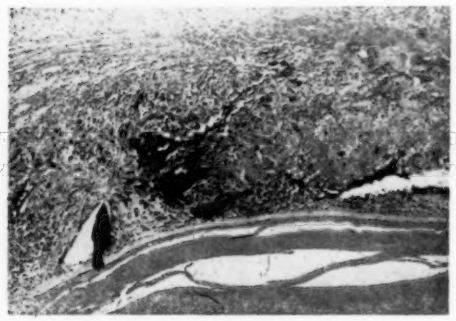


FIG. 28. Sympathetic uveitis evidenced by granulomatous inflammation of the uveal tract involving the iris. Lead foreign body in the iris. Posterior synechia. Enucleation two months after injury. A.I.P. Neg. 97294.

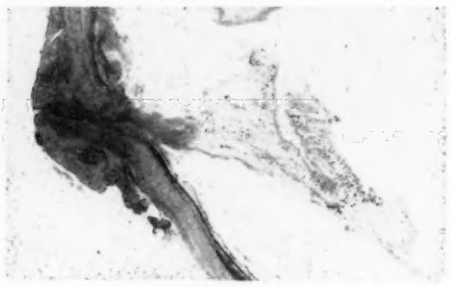


FIG. 31. Colonies of cocci in a vitreous abscess following a penetration of the sclera by a copper fragment which lodged in the vitreous. Enucleation two weeks after the explosion of a dynamite cap. A.I.P. Neg. 97291.

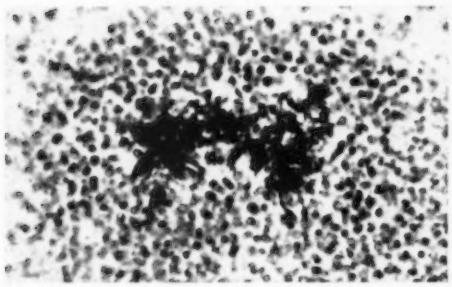


FIG. 32. Colony of unidentified fungi in a vitreous abscess. A minute nonmagnetic foreign body was found in the abscess on gross examination. Enucleation followed penetrating wound sustained in Italy. Duration unknown. A.I.P. Neg. 97283.

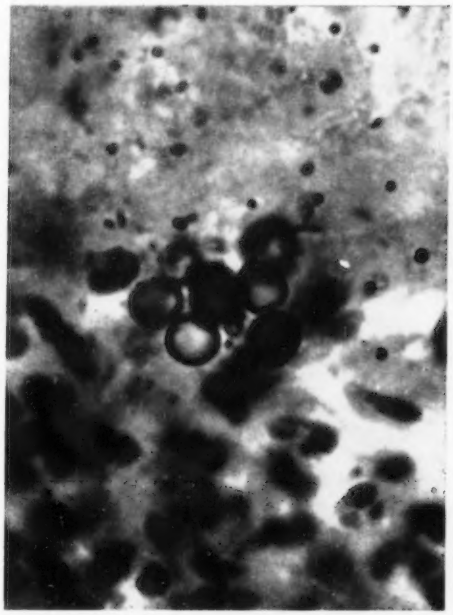


FIG. 34. Same eye as in fig. 33. Brown yeast-like organisms, probably saprophytic, in vitreous abscess. A.I.P. Neg. 99240.

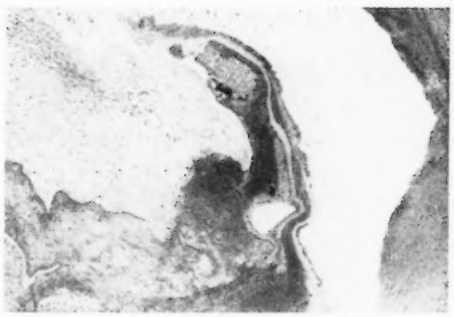


FIG. 33. Wood fragment prolapsed with retina through corneal wound in purulent panophthalmitis. Enucleation eight days after injury. A.I.P. Neg. 97355.

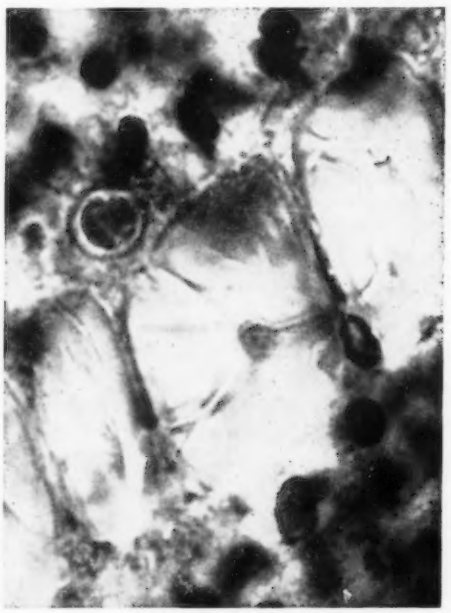


FIG. 35. Same eye as in figs. 33 and 34. Saprophytic fungi associated with wood particle in vitreous abscess. Double contoured cell and mycelium. A.I.P. Neg. 99241.

# II. EXPERIMENTAL WORK ON INTRAOCULAR FOREIGN BODIES

GILBERT C. STRUBLE, M.D.
WEST LOS ANGELES, CALIF.

MANY instances of magnetic foreign bodies involving the posterior segment of the eye were encountered in the ophthalmologic services of the army. Because of an increasing number of such cases, particularly of foreign bodies free in the vitreous, and since some approaches and technics appeared to offer advantages over other methods, my colleague at the Crile General Hospital, Eye Surgical Center, Major Leo Croll, and I decided to study the problem using such experimental material as was then available to us.

The results of this preliminary work using the Lancaster hand magnet have been published[1] and are no doubt familiar to some of you. The author has subsequently carried this investigation further using the giant magnet in the new series of experiments. Because of its connection with the subject under discussion, Dr. Gundersen has kindly invited me to present this material this afternoon. The time allotted to this discussion does not permit me to describe these experiments in detail. The procedures, however, are well demonstrated in the film to follow.

## EXPERIMENTAL STUDIES USING THE LANCASTER HAND MAGNET WITH FOOT SWITCH

The question investigated was as to what distances various sized particles could be drawn through the vitreous and through the uvea and out of the scleral incision. Particles of steel shavings measuring 1, ½ and ¼ millimeter in diameter were used because they were the size most often encountered clinically. The experiments were carried out on eyes of freshly killed hogs less than twelve hours dead which were kept refrigerated until used. It is realized many factors exist here not present in the injured human eye and that no organizing exudate, fibrin, or hemorrhage was present in the experimental specimens.

Direct observation of metal particles in the *vitreous* under magnetic attraction proved very interesting. The foreign body would move toward the magnet to some degree regardless of the distance involved. When the distance was too great for removal it would at once return to its original position on releasing the foot switch. At times the final resting place of the foreign body was a little closer than at the beginning but usually it would come to rest at its original position. The experimental data accumulated is shown in table I. On many occasions when the foreign body was under observation in the critical zone it would touch or nudge the retina or ciliary body during the magnet application and then return to its original position. With the foreign body in the certain zone the *success of the extraction on the first application of the magnet depended upon the initial impact of the foreign body against the inner layers of the globe.* The ½ and 1 mm. fragments, each in the proper zone, delivered without difficulty. The ¼ mm. fragments, due to their small size and lack of impact, had to be aided in their path

Formerly at Crile General Hospital, Cleveland, Ohio. Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

| Steel Fragment | Certain Zone I Distance in mm. | Critical Zone II Distance in mm. | Failure Zone III Distance in mm. |
|---|---|---|---|
| ¼ mm. | 3 | 3 to 7 | Greater than 7 |
| ½ mm. | 6 | 6 to 11 | Greater than 11 |
| 1 mm. | 11 | 11 to 16 | Greater than 16 |

TABLE I

through the uvea. In no instances were these fragments sharply pointed. They were, as a matter of fact, small, square or oblong particles with blunt ends.

*Foreign Bodies Embedded In the Retina and Choroid*

The eyes were prepared as before. The foreign body, however, was embedded in the choroid and retina at varying distances from the scleral section. Efforts at removal with the magnet were repeated in the usual manner. This series of experiments was inconclusive insofar as the ability to remove foreign bodies at distances greater than 4 mm. One consistent fact was noted: where the fragment was pulled laterally for a distance of 3 or 4 mm. toward the sclerotomy opening and magnet, a tear or gutter of the retina or choroid was created throughout the length of the movement of the particle. When the foreign body was embedded in the choroid or retina directly overlying the scleral opening it was always pulled through by the magnet on the first attempt with a minimum of trauma. This is further argument for precise pinpoint localization of a metallic foreign body embedded in these structures at the time of surgical removal.

EXPERIMENTAL STUDIES USING THE GIANT MAGNET WITH BLUNT TIP*

This work was carried out using highly magnetic steel particles of the following sizes: ¼x¼ mm., ¼x½ mm., 1x¾ mm., 1½x¾ mm., 2x¾ mm., 2½x¾ mm., and 3x¾ mm. All the particles used had relatively blunt ends. The work, as before, was carried out on eyes of freshly killed hogs less than twelve hours dead which were kept refrigerated until used. The particles were placed in the extreme posterior portions of the globes tested because of the following reasons:

1. The giant magnet exerts its maximum pull at this distance.
2. This was the distance at which this particular model has its greatest clinical application.

The investigations may be divided into four classifications:

CLASSIFICATION 1. *Experimental work on magnetic foreign bodies lying free in the vitreous.* The eye was placed on its side in a specially prepared acrylic mannequin* and the upper one-third of the sclera was outlined with methylene blue and excised. The dissection extended from the limbus, posterior to approximately 3 mm. from the optic nerve. The choroid and retina were now carefully removed throughout the same extent, care being taken to avoid loss of vitreous or to disturb the position of the lens. The previously chosen, measured, and weighed metallic fragment was then inserted into the vitreous near the posterior pole of the eye. The distance in millimeters from the foreign body to the anterior plane was carefully measured on each occasion prior to removal. In each instance the foreign body was placed in the vitreous at a depth corresponding as closely as possible with the

* Mueller Giant Eye Magnet.

* Designed and furnished through the courtesy of Major Stephen F. Smolczynski, Acrylic Eye Laboratory, Valley Forge General Hospital.

FOREIGN BODIES FREE IN THE VITREOUS

| Size in mm. | Weight | Distance to anterior plane of cornea | Particle Responds Magnet distance to eye | Delivered | Remarks |
|---|---|---|---|---|---|
| ¼ | 1.0 mg. | 19 mm. | Yes at 7 cm. | No | Moved forward to 9 mm. from anterior plane and retracted to 14 mm. |
| ¼ | 1.0 mg. | 12 mm. | Yes at 7 cm. | No | Moved forward to back of lens but retracted to original site. |
| ½ | 2.8 mg. | 18 mm. | Yes at 8 cm. | No | Moved forward to region of lens but retracted almost to original location. Lacked sufficient impact to embed in ciliary body or be drawn around lens into the anterior chamber from any distance. |
| 1 | 4.6 mg. | 19 mm. | Yes at 8 cm. | No | Moved forward at times as far as the ciliary body but would not embed and retracted to 2-3 mm. behind the lens. At other times could be brought forward to the ciliary body and remain there but could not be brought into the anterior chamber. |
| 1½ | 7.1 mg. | 19 mm. | Yes at 8 cm. | Yes | Moved forward slowly and finally embedded in the ciliary body or could be drawn into the anterior chamber. |
| 2 | 9.3 mg. | 17 mm. | Yes at 9 cm. | Yes | Moved forward more rapidly. Drawn into the anterior chamber or embedded in the ciliary body. |

TABLE II

optic axis of the eye so that a straight pull with the magnet could be made on each occasion. The results of this work are shown in table II and in the summary of the findings in this series of experiments.

SUMMARY OF FINDINGS CONCERNING FOREIGN BODIES FREE IN THE POSTERIOR VITREOUS

1. Particles less than 1 mm. in size could be brought forward temporarily, but they retracted back into the vitreous as the current was turned off. At times they occupied a location more anterior than in the beginning. None could be drawn into the anterior chamber, and none would embed in the ciliary body. Rate of movement very slow. No hazard to the lens.

2. Particles 1 mm. in size moved forward slowly without hazard to the lens. They would at times remain in contact with the ciliary body but would not embed. They usually retracted back into the anterior vitreous.

3. Particles larger than 1 mm. could be consistently brought forward. They would embed in the ciliary body or could be drawn into the anterior chamber. Particles up to 1½ mm. moved relatively slowly. Particles larger than 1½ mm. moved a little faster, but injury to the lens was a negligible factor, especially if the magnet was moved toward the eye slowly inside the 10 cm. distance.

CLASSIFICATION 2. *Magnetic foreign bodies embedded in the retina.*

The specimens were prepared in the manner above described. The previous-

FOREIGN BODIES IN THE RETINA

| Size in mm. | Weight | Distance to anterior plane of cornea | Particle responds (Distance?) | Delivered | Retina Separated | Comment |
|---|---|---|---|---|---|---|
| ¼ | 1.0 mg. | 21 mm. | ? | No | No | Not of sufficient size or weight to disengage or e evate retina. |
| ½ | 2.8 mg. | 22 mm. | Yes | No | Elevated a little | Not of sufficient size or weight to disengage. Elevates retina a little at times. |
| 1 | 4.6 mg. | 20 mm. | Yes | No | Detached | Slowly disengages and moves forward to the ciliary body but does not embed. Retracts back into vitreous behind lens when current is turned off. Could not be brought into the anterior chamber. |
| 1½ | 7.1 mg. | 22 mm. | Yes | Yes | Elevated during disengagement | Disengages suddenly and moves rapidly through vitreous and embeds in the ciliary body or could be brought into the anterior chamber. |
| 2 | 9.3 mg. | 22 mm. | Yes | Yes | As above | As above. On one occasion struck and bruised posterior lens capsule. Embeds in ciliary body or can be brought into the anterior chamber. |
| 2½ | 9.7 mg. | 21 mm. | Yes | Yes | As above | Disengages suddenly. Cuts very rapidly through vitreous and embeds in ciliary body or can be brought into anterior chamber. |
| 3 | 11.1 mg. | 22 mm. | Yes | Yes | As above | As above. |

TABLE III

ly chosen, measured, and weighed foreign bodies were placed on the end of a toothpick which was then inserted until the particle was buried in the retina. In those instances where difficulty was experienced in embedding the particle in this manner, the tip of the Lancaster hand magnet was applied to the outside of the sclera at the posterior pole at the level of the particle and the current applied. This proved to be an effective method whereby the particle could be embedded in a relatively uniform manner with a maximum of trauma to the globe. The results of these investigations appear in table III and the summary of the findings in this series.

## SUMMARY OF FINDINGS CONCERNING MAGNETIC FOREIGN BODIES IN THE RETINA

1. Particles less than 1 mm. did not disengage from the retina but could be seen to move on magnet application.

2. Particles 1 mm. in size may disengage but cannot be delivered.

3. Particles over 1 mm. in size will disengage and can be brought forward and delivered either through an approach over the ciliary body or through the anterior chamber.

4. Particles over 1 mm. in size suddenly disengaging appear to snap free under great tension and to move very rapidly through the vitreous, creating a

definite hazard to the posterior lens capsule.

5. The larger the particle, the greater the force of impact on intervening tissues.

6. The speed of particles through the vitreous after being suddenly disengaged from the retina appears definitely greater and more hazardous than in those cases where the particle was lying free in the vitreous.

7. All particles except those ¼ mm. in size caused some separation of the retina. The particles which disengaged slowly if at all (1 mm.) at times appeared to cause more retinal separation than the larger particles which disengaged suddenly.

CLASSIFICATION 3. *Magnetic foreign bodies lying between the choroid and retina.*

Using careful layer dissection of the sclera, a 6 mm. opening was made in the antero-posterior meridian of the globe, beginning close to the optic nerve and extending in the direction of the limbus. The dissection was continued at one end of the section until a black, glistening, choroidal bead presented. At this point, the blunt tips of a pair of Stevens' scissors were inserted between the choroid and sclera and the remaining scleral fibers severed with a single cut. Using a cyclodialysis spatula, the choroid was separated from the sclera for a short distance (about 3 mm.) to one side of the section. The exposed choroid in this area now carefully incised with the tip of a cataract knife, care being taken to avoid injuring the retina. The previously chosen, weighed, and measured magnetic foreign body was now introduced through the scleral and choroidal incision until it was lying on the retina beneath the choroid. The scleral incision was then closed with a previously placed suture, the eye was placed on its side in the mannequin, and the oval area of sclera, choroid and retina removed in the manner previously described. The giant magnet was now turned on to low power. The current was gradually increased to one-half full power by adjusting the hand reostat* and with the setting at this point, the eye mannequin, containing the prepared specimen, was gradually moved toward the tip of the magnet. Careful scrutiny of the foreign body lying under the retina was made during this procedure, using a binocular loupe for greater magnification and the hand slit-lamp** as a light source. Measurements were made with the millimeter ruler to determine at what distance the particle first showed evidences of being magnetic under attraction. As the foreign body was brought inside this zone of magnetic attraction, the reostat of the giant magnet was alternately turned to full power. This procedure was continued until and after the magnet tip was in contact with the cornea of the specimen. The results of this investigation appear in table IV and the summary of the findings in this series.

SUMMARY OF FINDINGS CONCERNING MAGNETIC FOREIGN BODIES BETWEEN THE RETINA AND CHOROID

1. Particles 1 mm. or less in size would not perforate the retina and could not be removed. Those between ½ and 1 mm. in size would elevate the retina somewhat.

2. Particles 1½ mm. or larger in size could always be delivered. The larger the size, the more rapidly they would perforate the retina. Disengagement was always very sudden and under great tension. Their progress then through the vitreous was very rapid and their impact on intervening tissues se-

* It was found to be impracticable to carry out prolonged tests with the magnet set at full power because of the overload on the electric circuit and the danger of overheating.

** Shahan's model.

FOREIGN BODIES BETWEEN THE RETINA AND CHOROID

| Size in mm. | Weight | Distance to anterior plane of cornea | Particle Responds Magnet distance to eye | Delivered | Retina separated | Comment |
|---|---|---|---|---|---|---|
| ¼ | 1.0 mg. | 21 mm. | ? | No | No | Only questionable movement of particle. No retinal separation. |
| ½ | 2.8 mg. | 21 mm. | Yes at 5 mm. | No | Lifted a little | Repeated application caused only slight retinal elevation. |
| 1 | 4.6 mg. | 21 mm. | Yes at 1 cm. | No | Lifted a little | Repeated application caused only moderate retinal elevation. |
| 1½ | 7.1 mg. | 21 mm. | Yes at 4 cm. | Always | Yes | After repeated applications moving the particle and retina back and forth the foreign body eventually worked through the retina (45-60 sec. exposure time.) On one occasion it suddenly perforated the retina and embedded in the posterior lens capsule. |
| 2 | 9.3 mg. | 20 mm. | Yes at 9 cm. | Always | Yes | Retina quickly perforated. Foreign body moves through the vitreous faster than can be followed, embedding in lens, iris, or could be drawn into the anterior chamber. |
| 2½ | 9.7 mg. | 20 mm. | Yes at 11 cm. | Always | Yes | As above. |
| 3 | 11.1 mg. | 20 mm. | Yes at 12 cm. | Always | Yes | As above. |

TABLE IV

vere enough to penetrate the lens, iris, or ciliary body.

CLASSIFICATION 4. *Magnetic foreign bodies lying between the choroid and sclera.*

The specimens were prepared in the manner just described, except that the choroid was not opened. The previously chosen, weighed and measured magnetic foreign bodies (the same particles were used in all the experiments) were now placed between the choroid and sclera. The incision was closed and the eye prepared as before. The same procedure was used in approaching the magnet tip, with the foreign body site under close scrutiny. The results of this investigation are shown in table V and in the summary.

SUMMARY OF FINDINGS CONCERNING MAGNETIC FOREIGN BODIES BETWEEN CHOROID AND SCLERA

1. Particles less than 1 mm. in size failed to respond noticeably.
2. Particles 1 mm. in size responded slightly.
3. Particles 1½ mm. in size and over produced an increasing degree of choroidal separation and retinal elevation.
4. In no instance was the choroid and retina perforated or the foreign body delivered.

FOREIGN BODIES BETWEEN THE CHOROID AND SCLERA

| Size in mm. | Weight | Distance to anterior plane of cornea | Particle responds Distance magnet to cornea | Delivered | Choroid & retina elevated | Comment |
|---|---|---|---|---|---|---|
| ¼ | 1.0 mg. | 20 mm. | No | No | No | None |
| ½ | 2.8 mg. | 20 mm. | No | No | No | None |
| 1 | 4.6 mg. | 20 mm. | Slight movement with tip in contact with cornea | No | Slightly | None |
| 1½ | 7.1 mg. | 21 mm. | Yes at 3 cm. | No | Slightly more elevation | None |
| 2 | 9.3 mg. | 20 mm. | Yes at 9 cm. | No | Marked elevation | None |
| 3 | 11.1 mg. | 20 mm. | Yes at 12 cm. | No | Very marked elevation | Prolonged and repeated exposures failed to cause penetration of the choroid or retina. Some distortion of the contour of the scle a occurred and severe separation of choroid was noted. |

TABLE V

One additional finding not previously noted is probably worth while recording. In the first series of experiments using the giant magnet, the long, slender, tapering tip was used to localize the point of application in an effort to determine, if possible, whether a direct pull through the center of the cornea along the optic axis of the eye appeared to be more effective than an oblique pull over the ciliary body region avoiding the lens. There is some evidence to indicate more pull was exterted on the foreign body using the oblique application than the direct frontal application. This may be because of two separate factors: (1) The oblique approach distance was two to three millimeters closer to the foreign body; (2) The reluctance of the lens is not a factor in the oblique approach. This finding is rather well demonstrated in the moving pictures of this procedure which will be shown later. It is realized, however, that with the long, tapering tip, maximum pull at a distance was not obtained in either instance.

As previously stated, it is realized that many factors exist in these specimens that are not present in the injured human eye. We are probably safe in stating, however, that given a foreign body problem with identical equipment available, it would be logical to assume the results here obtained represent the best which could be expected. Probably because of the additional factors of fibrin, hemorrhage, exudate, and so forth, the results would be inferior to those outlined.

## REFERENCES

1. Struble, Gilbert C. and Croll, Leo J.: Technical refinements in the removal of magnetic foreign bodies from the posterior segment of the eye, Am. J. Ophth., 29:151-161 (Feb.) 1946.

# III. THE X-RAY LOCALIZATION OF INTRAOCULAR FOREIGN BODIES

WILLIAM H. DROEGEMUELLER, M.D.
GREELEY, COLO.

TO THE ophthalmologist, the problems in the localization of intraocular foreign bodies involve more than the purely technical aspects of the methods of localization now available. The purpose of this discussion will be to present such problems. The x-ray technics of localization and the calculations used are highly perfected with little call for improvement unless it would be in the better visualization of the less opaque foreign bodies. With regard to the perfection in roentgenologic methods of localization Pfeiffer[1] has stated, "With consumate care it is possible for the roentgenologist to localize a foreign body and give the points of its position with greater accuracy than the surgeon can utilize at operation." Under ideal circumstances that statement is without doubt true. Of some significance is the fact that we as ophthalmologists in transposing a foreign body position from the roentgenologist's chart are subject to more or less error. It is such conditions in the problem of localization that it is desired to bring to attention for evaluation.

There are other potential sources of error in the commonly used methods. Whether the Sweet method, the Comberg contact lens technic, or markers of various types sutured to the exposed portion of the eye are used, the basis for the localization is the relationship revealed between the foreign body and the orientation marking device that has a known position to the eyeball proper. Upon this relationship the foreign body position is calculated and charted. In analysis, slight head movements or eye movements due to faulty fixation or in judgments involved and calculations in the charting can affect the accuracy of the localization. When we add to such a circumstance, the difficulty of the transposition of the charted site to the eyeball proper at surgery, it is apparent that, at the time the scleral incision is made, there is reason to question whether the selected position represents the maximum in localization accuracy. There is no certainty as to the exactness of the preceding steps, and it would seem well to hold such a localization as approximate.

Such an evaluation, that the methods we commonly use are only approximate, does not discredit the methods or necessarily our ability to locate a charted position upon the eyeball. The fact remains that we lack the proof of the accuracy in the localization and the surgical approach to the foreign body site. Though this seems somewhat exaggerated, which it is in the type of case seen in civilian practice, it was a definite actuality in cases of battle casualties that were complicated by multiple and small fragments in and about the orbit and eyes that were irritable or blinded by cataract or vitreous hemorrhage with the problem of fixation. In addition, the experience of checking the primary localization, as will be described, revealed how often, even in favorable cases, one may be in gross error or in error of a degree that must be evaluated in another important consideration in foreign body extractions, that is in terms of possible trauma produced in the foreign body removal.

The checking of the primary localization can be accomplished by a method described by Colonel Struble[4] in 1942 as pinpoint localization. The calculated foreign body site is exposed, but before

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

opening the sclera a small shaving of lead is sutured to the eyeball at the chosen position and the operation is interrupted to make lateral and postero-anterior roentgenograms. From the relationship of the foreign body to the check marker, the accuracy of the selection is established, or the necessity of readjustments to the additional evidence is revealed. Obviously, the time consumed in the interruption of the operation and the necessity of roentgenologic co-operation at this time, are undesirable features. It does add to the certainty of the localization which brings up the question as to the need for accuracy in localization.

It is apparent that absolute localization is essential in any attempt to remove a nonmagnetic foreign body, and usually the attempt is only justified after a careful consideration of the localization findings. Struble and Croll's[5] studies upon the technical aspects of foreign body removal have shown that for the full utilization of the magnetic power available, especially for the small magnetic and the weakly magnetic fragments, accurate localization is most desirable. In the usual cases of magnetic foreign bodies where magnetic power can make up for inaccuracies of localization, the answer lies not in the recovery of the foreign body but in an evaluation of the trauma produced in the extraction. An argument in favor of accurate localization would be the hypothesis that the least traumatizing pathway of removal would be directly out of the eyeball without the production of any lateral drag upon the retina or choroid. But such is a hypothesis and there are, no doubt, cases where the foreign body can be drawn away from the retina with less harm than through it. However, in the attempt to evaluate the effects of different pathways of removal it would seem necessary not only to know the relative foreign body position but its size and depth from the sclera, its shape and the plane of least width as well. Such information is made possible by extended localization procedures.

The roentgenogram of the antero-posterior exposure will often reveal foreign bodies obscured by the bony densities in the lateral views. The postero-anterior plate should be made with the head tilted to throw the petrous portion of the temporal bone just below the orbit. Preliminary roentgenograms, with a Comberg lens for orientation as to the anterior eye boundary and when made in stereoscopic views for the depth relationships revealed, are of value along with the clinical conditions, in planning further localization procedures. If contrary to clinical evidence a negative x-ray report should never be taken as proof as to the nonexistence of an intraocular foreign body.

(By use of lantern slides the author illustrated several methods of localization of foreign bodies by use of especially applied markers attached to the eyeball. In checking the localization, for the ease in identification and for the spacial plane relationships revealed, a 1½ mm. gold ring from a locket chain was demonstrated as used instead of a lead shaving. Also illustrated was a method of determining the foreign body site upon a wax model of the eye. In this method the contact lens with markers was placed upon the model in the position and plane as recorded at the taking of the roentgenograms. From a study of the stereoscopic plates the position of the foreign body was calculated upon the model. The model with the foreign body position and anatomical reference points was used in the surgical approach for reference.—Ed.)

Such checking of the foreign body site is not new. Spaeth[3] describes the use of wire rings of silver sutured at the limbus, the ora serrata and the

equator for nonmagnetic foreign body cases. That it is practical to use the method routinely, was recently described by Struble and Croll.[5] If conditions are such to prevent the check at operation, the procedure can be carried out in two stages. This is probably preferable in nonmagnetic cases since it allows a more detailed study than can be made from wet plates in the limited time at operation. The method can be modified for circumstances where an attempt at extraction is not successful and, rather than produce added trauma by repeated blind efforts, a ring can be left at the trial site and the case re-evaluated.

The reports on the Berman localizer, an instrument that reveals the presence of magnetic and some larger nonmagnetic fragments by an intensity sound signal, are encouraging for here is a method to check foreign body localizations. (The instrument is easily used at operation and in this respect is more desirable than the x-ray procedure described. It has an added feature in that the position of the foreign body can be checked if the position changes.) It would seem fair to state from reports that the Berman localizer is not as sensitive as the x-ray and of course will not be as available.

Time does not permit the discussion of procedures of value in special cases, as the bone free method of Vogt to confirm the presence of small fragments in the anterior segment. Parallax studies and the technic of injecting air into Tenon's capsule, as described by Spackman,[2] deserve special mention. These latter procedures are of value in determining whether a foreign body is intra- or extraocular. Parallax studies can be augmented by the use of scleral markers and the effect of the motion studied with the wax model. In this regard, it seems preferable to explore the site if questionable, and, if no foreign bodies are found, to leave a marker for further study. For fragments posterior to the eye, in a situation where exploration and the leaving of markers is impractical and where there is no doubt as to the correctness of the exposure which must be tangential in the foreign body vicinity, the use of air in Tenon's capsule would seem the method of choice.

The routine use of stereoscopic plates has a definite value. The source of error produced by movements is not as frequent as would be expected. Vertical movements blur the picture if fusible but do not alter the spacial relationships. Lateral movements in a degree to still allow fusion would alter the depth relationships but to a relative degree. For practical purposes, intraocular foreign bodies can be considered as fixed in position. (Observations upon vitreous fragments in response to the magnet were that, on release from the attraction, they returned to their former position.) Small movements of foreign bodies in special circumstances are probable and give rise to false projections; a situation that was encountered infrequently and, when such did occur, called attention to this possibility.

In conclusion, the ophthalmologic problem in x-ray localization of foreign bodies is in critically evaluating the technic used under the circumstances so that in the extraction of a foreign body, the added trauma to the eye is minimal.

## REFERENCES

1. Pfeiffer, R. L.: Localization of intraocular foreign bodies with the contact lens, Am. J. Roentgenol., 44:558, 1940.
2. Spackman, E. W.: Localization of intraorbital foreign bodies, Arch. Ophth., 18: 704, 1937.
3. Spaeth, E. B.: Principles and practice of ophthalmic surgery, Philadelphia, Lea and Febiger, 1941, p. 842.
4. Struble, G. C.: Personal communication.
5. Struble, G. C. and Croll, L. J.: Technical refinements in the removal of magnetic foreign bodies from the posterior segment of the eye, Am. J. Ophth., 29:151, 1946.

# IV. SURGERY OF INTRAOCULAR FOREIGN BODIES

TRYGVE GUNDERSEN, M.D.
BOSTON, MASS.

THE probability of injury to any part of the body by flying missiles can be predicted by the relative surface of that part exposed. In a frontal view of the body's silhouette, which measures approximately 4.2 square feet, the extremities comprise approximately 35 per cent of the silhouette, the head and neck 12 per cent, etc. The incidence of battle wounds corresponds roughly to these figures. The spherical eyeball has a silhouette surface of only .78 square inches or only 0.1 per cent of the total body surface. Both eyes would expose only 0.2 per cent. But the incidence of ocular casualties is much higher than this, at least ten times as high. One reason for this is certainly the fact that almost any injury to an eye results in disability, be it a small corneal abrasion, or even a minute foreign body under the upper lid. A perforating wound of the globe by the minutest foreign body will almost always result in the soldier's evacuation, which a similar wound of the auricle might not.

Accurate statistics on this subject are difficult to obtain principally because of two factors. 1. Inaccurate records and omissions in reporting casualties. It was found that the registrar in one of our large evacuation hospitals was reporting eye injuries as maxillofacial casualties to the Army surgeon. There was no classification on the registrar's form for eye injuries. It was also common practice to classify patients with multiple wounds according to the most extensive wound only. 2. Recounting of cases, especially during an active period when evacuation is hurried.

The following statistics were obtained in two ways: 1. Survey of records of the 6th General Hospital to which I was assigned during the entire war. Duplicate records were kept on each eye patient seen. The following table (I) gives a summary of these records. 2. "Spot checks" of all hospitals in the North African and later the Mediterranean theaters of operation were made at regular intervals. Circular form letters were sent to all hospitals even those in the Army zone every three months requiring information regarding the total hospital census, the eye census and all ocular diagnoses for one definite day. A compilation of these figures is given in table II.

In comparing these figures it is noted that in the 6th General Hospital there was a higher incidence of eye injury (5.63 per cent), eye battle casualty (3.38 per cent) and eye disease (9.83 per cent) than in all hospitals combined, namely eye injury (1.96 per cent), battle casualty (2.57 per cent) and eye disease (1.80 per cent). This difference is due to the more accurate records which can be kept by one interested individual plus the fact that for a part of the period concerned, the 6th General Hospital functioned as one of six eye treatment centers in the two theaters.

## NEW EXPERIENCES WITH INTRAOCULAR FOREIGN BODIES

### *High Velocity Fragments*

It was learned early in the recent war that the increased velocity of flying missiles caused a different type wound than

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

SHOWING NUMBER AND CLASSIFICATION OF PATIENTS SEEN — EYE SECTION — 6th GENERAL HOSPITAL MARCH 1943 TO DECEMBER 1944

| | No. of Cases | % of total hospital admissions | % of patients seen including O.P. | % of patients with eye diagnosis |
|---|---|---|---|---|
| Total hospital admissions | 26,223 | 100 | | |
| Battle casualties | 7,090 | 27 | | |
| Injuries | 4,504 | 17.2 | | |
| Disease | 14,629 | 55.8 | | |
| Total patients seen in Eye clinic (Including out patients) | 6,101 | ..... | 100 | |
| Refractions | 3,075 | ..... | 50.4 | |
| Out patients | 1,090 | ..... | 17.86 | |
| Total patients admitted to hospital with eye diagnosis | 1,936 | 7.37 | 31.74 | 100 |
| Primary eye diagnosis | 1,172 | 4.46 | 19.2 | 97.8 |
| Secondary eye diagnosis | 764 | 2.91 | 12.54 | 2.2 |
| Eye patients admitted to hospital as battle casualties (3.38% of all the patients admitted to hospital as B.C.) | 241 | 0.91 | 3.95 | 12.50 |
| Eye patients admitted to hospital due to injury, accidental (5.63% of all hospital admissions due to injury, accidental) | 255 | 0.97 | 4.17 | 13.17 |
| Eye patients admitted to hospital due to disease (9.83% of all hospital admissions due to disease) | 1,440 | 5.49 | 23.62 | 74.33 |
| Total glasses ordered (44.68% of refractions) | 1,374 | | | |

TABLE I

had been encountered before. A tiny shell fragment with a wound of entry often so small as to defy detection caused explosive destruction of tissues underneath the surface. This applied to ocular injuries as well. Though many eyes were struck by minute foreign bodies, the high velocity caused catastrophic injuries to the retina and choroid.

*Multiple Foreign Bodies*

An individual could be sprayed, literally with hundreds of fine fragments from a single exploding shell, bomb, or mine. Both eyes were therefore frequently injured often with multiple foreign bodies in each.

A special type of injury was seen by the writer for the first time. This consisted of a single wound of entry associated with many intraocular foreign bodies. The first soldier who came under my care with this unusual injury was a young sergeant who, while being strafed by low flying enemy aircraft stood behind his 50 caliber machine gun mounted in a half track until he was severely wounded. One eye was completely destroyed. The other had a single 3 millimeter wound of the cornea, but scattered over the entire surface of the iris and lens were myriads of particles which had the appearance of lead. Though the eye remained relatively quiet for a period of two weeks, it sud-

CONSOLIDATED REPORT

Battle Casualties, Injuries, and Diseases — Summary of 4 "Spot Checks"

| | March 20 1944 | | June 25 1944 | | Sept. 25 1944 | | Feb. 25 1945 | | Average | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Actual Number | % | Actual Number | % | Actual Number | % | Actual Number | % | Actual Number | % |
| Total Hospital Census | 33,861 | | 32,668 | | 25,767 | | 22,275 | | 28,643 | |
| Less — PW and Allied Troops | ..... | | 4,762 | | 2,202 | | 1,960 | | 2,231 | |
| Total Hospital Census (US) | 33,861 | 100. | 27,906 | 100. | 23,565 | 100. | 20,315 | 100. | 26,412 | 100. |
| Battle Casualties | 9,361 | 28. | 11,509 | 41. | 7,924 | 34. | 2,670 | 13. | 7,866 | 29. |
| Injuries | 4,282 | 13. | 4,226 | 15. | 3,412 | 14. | 3,103 | 15. | 3,756 | 14. |
| Diseases | 20,218 | 59. | 12,171 | 44. | 12,229 | 52. | 14,542 | 72. | 14,790 | 57. |
| Total Eye Cases | 672 | 1.98 | 604 | 2.16 | 553 | 2.35 | 352 | 1.73 | 545 | 2.06 |
| Eye Battle Casualties | 244 | | 248 | | 224 | | 99 | | 204 | |
| % of Eye Cases | | 36. | | 41. | | 41. | | 28. | | 37. |
| % of Census | | .71 | | .89 | | .99 | | .49 | | .77 |
| % of Battle Casualties | | 2.60 | | 2.15 | | 2.82 | | 3.70 | | 2.57 |
| Eye Injuries | 88 | | 94 | | 68 | | 45 | | 74 | |
| % of Eye Cases | | 13. | | 16. | | 12. | | 13. | | 13. |
| % of Census | | .26 | | .33 | | .25 | | .22 | | .28 |
| % of Injuries | | 2.05 | | 2.22 | | 1.99 | | 1.45 | | 1.96 |
| Eye Diseases | 340 | | 262 | | 261 | | 208 | | 267 | |
| % of Eye Cases | | 51. | | 43. | | 47. | | 59. | | 50. |
| % of Census | | 1.01 | | .93 | | 1.10 | | 1.02 | | 1.01 |
| % of Disease | | 1.68 | | 2.15 | | 2.13 | | 1.43 | | 1.80 |

TABLE II

denly developed a purulent enophthalmitis which went on to complete destruction of the globe. A roentgenologic examination of the enucleated globe showed more than 50 individual particles. My only explanation of this phenomenon is that the eye was perforated by molten metal which scattered through the aqueous as it solidified. I subsequently observed this type of injury four times.

## INSTRUMENT AND APPARATUS

### *Foreign Body Locators*

Perhaps the most useful tool developed during the war years for the diagnosis and accurate localization of foreign bodies is the foreign body locator. It was used with success at Pearl Harbor by Moorhead in December, 1941, subsequently by Minsky and many others. At Valley Forge General Hospital, the Army's largest eye center, it was received and used with great enthusiasm by Lt. Col. M. E. Randolph and his staff including the writer.

The locator operates on the same principle as the mine detector and has especially refined points which can be brought to the operating table under sterile conditions. The apparatus indicates the presence of a foreign body by both a visible dial and a high pitched sound which becomes a squeal as the foreign body is approached. The best known and probably the most refined instrument has been designed by Berman. A similar locator was built for each hospital in the Mediterranean theater of operations by Lt. Col. Henry Carney, D.C., from mine detectors. Some of them had special appliances for ophthalmic operations which were used with great success in several eye centers.

The detector causes no movement of the foreign body and therefore in contrast to the "magnet test" for foreign body, no harm can be done by using it. Its application is the same when used as a diagnostic test for the presence of a foreign body or for accurate localization at the operating table. The visual and auditory indicators can be tuned according to the size, proximity and nature of the foreign body so that it is an instrument of great sensitivity. Some nonmagnetic foreign bodies can be located in this way, for instance copper and brass; though others, wood and plastic, give no response.

The locators give no information which cannot be obtained by precise x-ray localization and in some instances, for example, insensitive foreign bodies, locators are useless and one must rely on x-rays. However, in most cases of intraocular foreign bodies the locator does indicate accurately the nearest approach to the foreign body, reduces the number of roentgen examinations or may obviate them entirely. In determining the point on the sclera which is nearest to the foreign body and therefore in most instances the site for incision, the locator is of great value.

### *Magnets*

Since the ultimate aim is to remove the foreign body with the least possible trauma to the eye itself, it has always been considered good practice to use the weakest magnetic force which will move the foreign body from the tissue in which it is lodged. An instrument with low magnetic force, even a magnetic probe, will often suffice particularly when it can be applied directly on or very near the foreign body. A very powerful magnet such as the electromagnets of Lancaster and Haab can be reduced in power by increasing the distance between the foreign body and the magnet tip, but this manipulation has the disadvantage of being cumbersome and the force is less delicately controlled. Reducing the magnetic force by reducing the electric flow is also pos-

sible, and often used in large magnets. The magnetic force has more recently been reduced by using magnetic cores of diminishing size, a principle utilized in the Mellinger magnet. This instrument is constructed with large windings turned in an oval shape so as to fit around the patient's entire face. The patient's eye is therefore in the magnetic field and an iron or steel rod becomes a magnet by bringing it into the field. The size of the piece of steel introduced determines the amount of magnetic force.

For extractions by the posterior route a strong magnet is rarely if ever necessary if localization has been properly done. The Lancaster hand magnet which was authorized in every Army hospital, proved to be an exceedingly useful instrument. The fact that each magnet had its own rectifier, and that the entire assembly fitted into a small compact case was especially appreciated by highly mobile hospitals. Giant magnets which find their usefulness in anterior route extractions are cumbersome because of their size and weight. Lancaster's model which was available to especially designated Army hospitals comes equipped with a large motor generator, but unfortunately there is no direct switch or "cut-off" between the converter and magnet so as to give the operator the advantage which comes with the initial inflow of current through the magnet's coil. Such a switch can be added by any competent electrician.

A great many Army magnets were burned out by operators who did not understand that if a foreign body was not attracted instantly the moment the switch was turned on, it would not be attracted by keeping the current flowing through the magnet until the coils became hot. In this connection it is appropriate to repeat a similie used by Lancaster. If it requires 30 pounds pull to extract a cork from a bottle, one might exert 25 pounds pull indefinitely, or repeatedly every day, without success. On the other hand a pull of 30 pounds or more will extract the cork immediately. The Lancaster magnet was not designed to exert a constant magnetic force over a long period of time.

## FACTORS WHICH INFLUENCE THE CHOICE OF OPERATION FOR INTRAOCULAR FOREIGN BODIES

In no field of ocular surgery is surgical judgment more important. A routine procedure should never be adopted, but let each case be judged according to the individual problems it presents. Among those factors to be considered are the following:

1. How much has the eye already been damaged? This cannot be estimated at the time of injury or even for a few days thereafter. Often have I seen records of the initial examination made by competent ophthalmologists showing vision to be nil, only to find definite light perception recorded after a few days. For this reason alone, the operation of immediate enucleation should be abandoned except in extensive injuries where enucleation becomes part of the general debridement. Wounds of the ciliary body have long been known to be the most dangerous ones as far as sympathetic ophthalmia is concerned. No evidence has come forth to refute this teaching and surely this type of wound weighs heavily in the scale favoring enucleation. Wounds of the lens, per se, do not justify enucleation though they strongly influence one to enucleate an eye if other injuries preclude the possibility of clear media to a normal macula. Intraocular hemorrhages make examinations difficult and prognostication uncertain. It is important to determine their extent and exact location. They may be from the retinal or uveal circulation and occur in the anterior chamber, vitreous, retina or uveal tract. Anterior chamber hemorrhages should

be promptly evacuated if associated with glaucoma. Hemorrhages into the vitreous show great variation in their rapidity and degree of absorption and should be given every opportunity for spontaneous absorption. Choroidal hemorrhages are usually extensive, quickly cause irreparable damage to the overlying retina and therefore indicate a poor prospect for return of useful vision. Since the more anteriorly situated hemorrhages obscure deeper ones from view, the examiner must rely on subjective examinations to determine the extent and location of deeper ones. It is especially important to differentiate those in the vitreous from those in the subchoroidal space since, as indicated above, the prognosis is quite different. The differentiation can often be made by examining the visual field with a point source of light of low intensity. An ideal test object is the May ophthalmoscope after the condenser has been removed from the bulb.

2. What is the nature of the foreign body? Throughout civilian experiences with the subject, we have been accustomed to expect intraocular foreign bodies to be single chips of metal from chisels, hammers, rivets, nails and the like with the occasional variation of a nonmagnetic bird shot or the fragment of a copper detonation cap. The war taught us to expect many other types of foreign bodies and if we did not realize what was passing before us, I am sure we have been surprised by Mrs. Wilder's analysis of foreign bodies removed from eyes sent to the Army Medical Museum. Multiple foreign bodies were seen by all military ophthalmologists but I am sure that few were aware of the number of eyes which contained both magnetic and nonmagnetic particles. Furthermore, I am even more sure that none of us realize how many eyes contained secondary foreign bodies such as paint, vegetable matter, skin and cilia carried in by the primary missile. Preoperative knowledge of this would surely have hastened our decision to enucleate many of these eyes.

That foreign bodies vary greatly in their toxicity to tissues is well known. Generally speaking those of high atomic weight are inert. Gold, silver and platinum are well tolerated; lead and zinc less well so. On the other hand, iron, especially soft iron, and copper oxidize rapidly and cause the well known clinical pictures of siderosis and chalcosis. The war brought to our attention a group of less well known materials. Plexiglass and other plastic materials were often found causing no intraocular inflammation whatever. I recall a pilot whom I saw in consultation with Dr. Karl Sandt of the 26th General Hospital whose anterior chamber was transfixed by a spicule of plexiglass. Even after several weeks this patient had normal vision and no evidence of intraocular inflammation. Minerals were blasted into eyes especially by mines, but also by shells exploding in the ground. When dozens of bits of rock were found embedded in the corneas, at all different levels, the relative toxicity of different ones could be studied with the slit lamp. Limestone particles invariably caused edema and softening of the surrounding cornea and attracted blood vessels rapidly. The tracts of entry remained soft and unhealed. Crystalline rocks such as granite, quartz, etc., caused almost no reaction and were therefore well tolerated. Since an analogous tissue reaction occurs when such foreign bodies are intraocular, it is understandable that there is tremendous variation in the degree of endophthalmitis caused by different types of rock and minerals.

3. Can the foreign body or foreign bodies be removed, and if so, with how much additional damage to the eye? There are probably no magnetic foreign bodies which defy removal with modern

magnets and with both anterior and posterior approaches available to the surgeon. A colleague told me with some pride that he had removed 125 intraocular foreign bodies from as many patients in various casualty clearing stations. Certainly this indicated a tremendous surgical effort and application to duty, but knowing the diagnostic and surgical equipment this medical officer had, the hardships under which he worked, and the immediate evacuation policy enforced in his sector, I wondered if the first surgical principle, *primum non nocere*, had been followed. The surgeon should always ask himself, "Was the foreign body removed with the least possible additional trauma?" Nonmagnetic foreign bodies naturally present more difficulties and can be removed only under direct vision or under indirect vision by some ingenious device like the Thorpe endoscope. Indeed, localization must be "pinpoint" in accuracy to avoid failure or calamity.

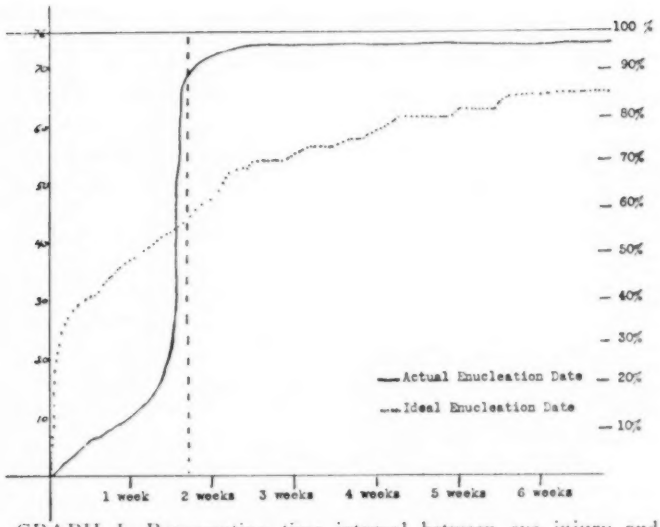
4. Are intraocular foreign bodies emergencies? The vast majority of individuals who are struck in the eye by flying bits of metal experience enough pain or disability to report for medical care promptly. These comprise a large group of the relatively few ophthalmologic emergencies. In hospitals which are staffed and equipped to care for ophthalmic problems it has been customary to extract such foreign bodies as soon as possible, either after x-ray localization has been done, or even before, if such an examination seemed superfluous. The accent was placed on speedy removal, usually at the expense of poor localization. Hirshberg was a great exponent of speed and I have been told by Lancaster that foreign bodies were often removed within a few minutes after the patients arrived in the Hirshberg Clinic. This certainly reduced the likelihood of infection and gave the foreign body little chance to become fastened in the tissue except by fibrin. In institutions where anterior extractions were the rule, a "magnet test" was usually done, and if the patient experienced pain or a "pull" when the large magnet was applied, the diagnosis was confirmed and the extraction performed under more complete anesthesia. But the rapidity with which foreign bodies become firmly embedded in tissues is not accurately known and of course varies enormously with several factors, among others the tissue concerned, the size and shape of the foreign body and with the amount of hemorrhage surrounding the foreign body. Were the rapidity of embedding plotted against time, it is probable that the curve would rise very fast during the period of clotting and slowly thereafter. Therefore, disregarding infection, it would matter less whether the foreign body were removed between the second or sixth day than between the second or sixth hour after injury. Two factors have altered the need for speed. Sulfonamides and penicillin have reduced the incidence of infection to a small fraction of what it was. During the early months of the North African Campaign and until the summer of 1943, sulfanilamide was carried by each soldier and taken by him immediately after injury. Suppurative endophthalmitis was rare. It was my observation and that of several colleagues that when penicillin therapy partially replaced sulfanilamide as the routine treatment after wounding, the incidence of endophthalmitis rose perceptibly. Even then, frank intraocular suppuration was rare, and probably occurred only in those eyes which were heavily inoculated with virulent organisms. It is doubtful in my mind that early removal of the intraocular foreign body would have saved many of these eyes. Considering these factors, my plea before this Academy is that emphasis be changed from speed of extraction to accuracy of localization.

5. By which route should intraocular foreign bodies be extracted? An obvious change has occurred in the surgical attack on foreign bodies in recent years. Since the days of Haab, the pendulum has swung so far from the anterior towards the posterior approach that many surgeons use the latter exclusively, and are practically unfamiliar with the anterior extraction. Others are unwilling to admit that the anterior route has any place in ophthalmic surgery. Perhaps the intimacy we have gained with the posterior segment of the globe through operations for detached retina has given some surgeons a feeling of complete security in incising normal retina and choroid to extract an underlying foreign body. But there are other well-known ophthalmic centers where anterior extractions are still routine procedures. Any magnetic particle, regardless of its location is drawn forward into the anterior chamber and extracted through a corneal incision. Only when this procedure fails after repeated attempts on successive days is a posterior sclerotomy done. The pars planum approach advocated by Verhoeff was devised to minimize operative injury to just such patients.

The disadvantages and dangers of each method are quite clear and should be remembered by every operator. When incising the sclera, uvea or retina, there is danger of hemorrhage from these tissues, which although reducible by preliminary diathermy coagulation of the sclera or by incision with the diathermy knife, is nevertheless present. It is surprising how rarely serious intraocular hemorrhage does occur. Detachment of the retina follows in a definite number of patients who have had posterior operations and offers little hope of cure, presumably because normal vitreous actually separates the retina from the choroid. The danger of sympathetic ophthalmia is increased slightly by posterior segment operations; probably more by incisions through the ciliary body than the choroid. Anterior route extractions can cause the retina to be torn or even pulled off when the foreign body is firmly embedded in it or attached to it by inflammatory adhesions. The other danger is that of injury to the clear lens. No surgeon at the Massachusetts Eye and Ear Infirmary could recall such an accident before the war, but I have been told that it occurred in two successive cases. In the first a small (2x2x3 mm.) and in the second a large (4x5x6 mm.) foreign body traversed the clear lens causing cataract. This accident should be avoidable, by using care in planning the path of the fragment. It should be remembered that foreign bodies approaching a magnet develop speed in accordance with the law of gravity and therefore require some distance to cause such an accident. A foreign body directly behind the posterior capsule would have much less chance of developing the necessary speed than one in the posterior vitreous. The anterior route is less desirable for large foreign bodies which cause extensive tearing of the zonula. Danger of sympathetic ophthalmia is not especially increased by the additional keratotomy.

Let us therefore, utilize the advantages of both methods. When a foreign body can be seen in the vitreous with no attachment to the retina a carefully executed anterior extraction is the perfect operation. Even when hemorrhage or cataract obscures the foreign body this is the operation of choice. On the other hand all those which are in, under, or attached to the retina should be removed by the direct scleral incision. It should never be forgotten that each of these patients represents a potential detachment. Let him be treated accordingly.

6. When should enucleation be done? This is frequently the most difficult de-




GRAPH I—Representing time interval between eye injury and enucleation. Based on study of 76 cases at 6th General Hospital.

cision the ophthalmologist has to make. The danger of sympathetic ophthalmia, though it varies greatly in different types of perforating wounds of the globe, is never absent entirely.

The fear of sympathetic ophthalmia must have been greater during the time of World War I than at present, causing enucleations to be done earlier and probably more frequently. I have heard several general surgeons of that war describe enucleations which they did presumably as emergencies to save the other eye. Also there is no proved case of sympathetic uveitis resulting from World War I which indicates that this was true. During World War II, unfortunately, the enucleation of hopelessly damaged and dangerous eyes was frequently delayed too long. The Army Medical Museum has already made the diagnosis of sympathetic uveitis fourteen times, and I have seen three other soldiers with clinically unmistakable disease in both eyes.

During my service in North Africa, 76 soldiers who had had enucleation of one eye were evacuated from our hospital. I plotted the number of individual cases against their respective enucleation dates and compared the resulting curve to what I constructed as the ideal enucleation curve. (Graph I.) If one agrees that twelve days is the absolute limit of safety for sympathetic ophthalmia all eyes that eventually will come to enucleation should be removed before that dead line. Of course, this ideal is not possible since medical judgment is not exact and the future is always uncertain. It is my opinion however that in at least 90 per cent of cases the decision can and should be made before twelve days have elapsed after injury.

After signs of disease have begun in the sympathizing eye, the exciting eye should not be removed unless it is sightless. It has been shown that on numerous occasions when the disease has been finally arrested the originally injured eye has better vision than its fellow.

## SUMMARY AND CONCLUSIONS

1. An analysis has been presented of the incidence of ocular battle casualties, injury and disease in two theaters of operation in World War II. A similar

analysis has been presented of the admissions to one general hospital functioning in those theaters.

2. The extensive use of sulfonamides and antibiotics during the war has conclusively shown that sepsis induced by intraocular foreign bodies can be reduced tremendously by the use of these agents.

3. The removal of intraocular foreign bodies should no longer be considered so urgent that meticulous localization of the foreign body cannot be made.

4. Enucleation of perforated eyes with or without retained foreign bodies should be postponed until a definite estimation can be made of the salvagable vision and this estimation can be weighed against the danger of sympathetic ophthalmia.

5. Precise roentgenologic localization, and the use of foreign body locators have greatly minimized the uncertainty attached to the removal of intraocular foreign bodies.

6. The choice of operation for intraocular foreign bodies should be made according to problems presented by the individual case. The advantages of the anterior and posterior methods should be considered and the proper operation selected.

# PREOPERATIVE RECONSTRUCTION OF THE ORBIT

## (An X-ray Aid to Surgical Restoration)

E. L. Shiflett, M.D.
LOUISVILLE, KY.
BY INVITATION

Many patients were admitted to the eye plastic section of an army general hospital with severe fractures of the orbit who had to have reconstructive surgery for cosmetic reasons and for the fitting of eye prostheses. There are individuals who suffer an expansile type of intra-orbital fracture with marked increase in orbital volume from the explosive force of small shell fragments traveling with great velocity. Often there is no external evidence of this fracture to aid in the detection of this type of injury. Unless this is appreciated and all cases are x-rayed preoperatively, restoration surgery is frequently inadequate.

The surgeon is handicapped in the operating room by having only flat x-ray films or a written report for reference. These facilities usually are inadequate to guide and aid him unless he has retained a mental impression of the orbital deformities from a previous stereoscopic study of the films.

The eye plastic section recounted some of the difficulties to the radiologic service which led to the development of x-ray reconstruction of the orbit through the medium of orbital tracings described below. This process was developed through cumulative experience and the successive stages will be discussed in sequence of development.

Stereoscopic postero-anterior films in the chin-nose position, the chin and nose resting lightly on the cassette holder, and stereoscopic laterals, the injured side down, were made. The films were processed in the usual way. After they were processed they were studied stereoscopically for the type, depth, and location of fractures. After this was completed, and while looking stereoscopically, the normal and abnormal were sketched on the film with one hand while the films remained in the stereoscope. The depth of injuries, and areas where tissue had been lost completely were indicated by using different colored wax pencils so that the dimensions could be appreciated and recorded when the final tracing was made. Technic varied little and always represented an actual outlining of normal and abnormal findings in each case.

The final tracing was obtained by placing the sketched film flat on an ordinary view box in the horizontal position. A sheet of transparent blueprint tracing paper was then superimposed on this film, and by transmitted light, the outlined orbits on the film just described were easily and accurately transferred to the transparent paper by a retracing process. This was done with an ordinary lettering pen and india ink, and with different colored pencils to indicate the depth, location, and the amount of injured or lost tissue. Enough of the bony silhouette of the face was traced for appreciation of facial symmetry.

The vertical, horizontal, and other meridians of the normal and abnormal orbit were measured, and any increased or decreased dimensions of the involved orbit were set down in comparative

Formerly at Crile General Hospital, Cleveland, Ohio. Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

measurements of the normal orbit so that an accurate comparison could be made. The finished tracing with explanatory legends, and a written report, were incorporated in the patient's record. These could be studied before the patient was taken to the operating room and the approximate volume and shape of tissue or material needed for restoration could be prepared in advance. The accurate localization also enabled the operator to proceed quickly to the immediate problem of restoration.

The procedure described above proved to be quite helpful but had limitations. The reconstruction failed to show the relationship of the normal position of the globe and orbital injuries. We believed that if the normal position of the globe could be shown in its relation to fractures and other defects, it might aid materially in planning operative procedure, providing that the following could be established: the size and shape of the orbit, the normal position of the globe in the orbit, the relative position of the globe to the site of injury, and the depth of periglobal tissue. If these could be determined and proved to be of value, a more accurate and purposeful preoperative planning was promthe normal position of the globe in the ised. This study for reconstruction of the globe in the orbit was undertaken. The material selected consisted of films on a series of 50 cases on whom Sweet localizations had been made for foreign bodies in the eye. Cases with severely deformed orbits and shrunken globes were not used. We assumed that since the Sweet localizations were accurate enough to justify entering the globe for removal of the foreign bodies, a reconstruction of the relationship of the globe in the orbit, based on two known planes, would be of sufficient accuracy to be of practical surgical aid. The two known planes were the distance of the summit of the cornea from the ball of the Sweet localizer and the horizontal axis of the globe. The globe was reconstructed in the orbit by constructing a sphere 25 millimeters in diameter on the plane of the horizontal axis, and the summit of the cornea was established by constructing a tangent to the sphere perpendicular to the horizontal axis exactly 10 mm. from the ball of the Sweet localizer. This gave a practical relationship of globe to orbit as existed before injury. It established the relationship of globe to orbit in position and volume, and the position of the globe in reference to injuries of the orbit and facial bones. This study indicated that there are three general types of globes and orbits; namely, the enophthalmic eye where the vertical plane of the summit of the cornea lies posterior to the anterior margin of the orbital floor and constitutes 42 per cent, the exophthalmic type where the summit of the cornea lies anterior to the anterior margin of the orbital floor and constitutes 32 per cent, and, a third type where the summit of the cornea and the anterior margin of the orbital floor are flush, constituting 26 per cent of the cases. There is a considerable individual variable in each of the types. We found by this study that although there are orbital averages, the variable is so great that the only reliable control in any individual is the contralateral normal orbit. Anatomical features were established which should be definitely helpful in restoration of the destroyed globe and the injured orbit. The relation of globe to sinuses varies considerably. The dorso-ventral position of the globe is inconstant but falls into three large groups; namely, the enophthalmic, exophthalmic and the neutral types. The enophthalmic constitute nearly half of the individuals. The cephalocaudad position also varies greatly, and although the average globe lies 7.3 mm. below the orbital roof, the individual globe may practically lie

against the orbital roof or on the orbital floor. The average eye is poorly protected against trauma from the side. After accomplishing this reconstruction in the lateral plane, we desired to reconstruct the globe in the orbit in its normal position as viewed from the front.

We reviewed a series of scout films which had been made as preliminary studies to Sweet localizations. The vertical and horizontal meridians of the orbit were measured on films made in the chin-nose position. The point of intersection of these meridians was used as a center of a sphere 25 mm. in diameter so that the similar meridians of the sphere and the orbit superimposed, letting the horizontal and vertical meridians represent the vertical and horizontal planes of the cornea. Foreign bodies were then charted above or below the constructed horizontal plane of the cornea and to the nasal or temporal side. These positions were then checked against the position of foreign bodies as reported at the actual time of Sweet localization. The positions varied from zero to several millimeters. We therefore decided that a reconstruction in this plane was of sufficient accuracy to be used as a helpful surgical guide and added this procedure to those described above.

This completed our studies on the preoperative x-ray reconstruction of the globe and orbit.

Orbital cases were submitted for this study before and after operation. The surgeon received written reports, and orbital tracings with explanatory legends with suggestions by the radiologist as to the probable importance of certain mechanical factors. The globe-orbit-injury triad was portrayed in different colors when it was necessary to differentiate between displaced and lost tissue and to indicate the depth of injuries. Increased or decreased orbital diameters were measured in millimeters and compared with similar planes of the normal orbit. Facial asymmetry could be appreciated by referring to the tracings in the front and side views. When both orbits were badly shattered the complete reconstruction could not be done and tracings only of bone deformities were submitted.

We had adequate opportunity to confirm the accuracy of these reconstructions. Tracings were made before and after operation where tantalum, which is opaque to x-ray, was used to correct deformities within the orbit. Postoperative tracings showed that the tantalum filled the bony defects which had been demonstrated and measured by this method prior to operation. The postoperative tracings made without reference to the original duplicated the original with much accuracy. We also had cases where the opaque prosthetic ball was used as an indicator of the site and of the amount of restoration when cartilage instead of tantalum was used.

Our studies of reconstruction subjected to surgical control caused us to draw certain general conclusions with which the surgeon may or may not agree. The cephalocaudad position of the globe is important in that it might be used as an indicator for conservative or radical elevation of the orbital floor. It would certainly indicate that the same elevation is not applicable to both the high and low lying globe. Restoration of lost tissue, without a preoperative method of measurement is usually too conservative. The most evident deformity is not always the chief offender as the cause of an unsightly prosthesis or malposition of the eye. Correction of the lesser of several injuries will often give a good cosmetic result which could not be obtained by correcting only the most evident injury, and a decision as to which one to correct cannot always be determined by inspection and palpation alone. Unrecognized expansile intra-

orbital fractures are frequently the cause of the sunken and otherwise misplaced prosthesis. The size of the orbital floor defect is not necessarily compatible with global malposition or displaced prosthesis, as the prolapse of orbital tissue through a small defect will cause malposition of both. It is possible that the opaque prosthetic ball position has some influence on the position of the artificial eye. Difficulty in attaching the superior paddle of the implant eye can be anticipated and prepared for by the simple procedure of orbital reconstruction. This procedure will aid the surgeon to measure and to shape with reasonable accuracy the tissue or material necessary for restoration before the patient is brought to the operating room.

Practical applications of this method were discovered and applied to the surgical restoration of the wounded orbit. We believe that persistent study and consistent use of this method, or some other suitable method of preoperative reconstruction, will aid the surgeon and benefit the patient. We offer this method only as a practical aid to the surgeon, believing that it has a high degree of clinical and surgical accuracy, but do not offer it as representing in any way a physical or anatomical formula of complete accuracy. The method was developed by mutual assistance and co-operation of the ophthalmology and x-ray services, and neither service could have developed it independently.

## DISCUSSION

GILBERT C. STRUBLE, M.D., West Los Angeles, Calif.: This method of orbital tracings and preoperative reconstruction of the orbits devised by Lt. Colonel Shiflett has proved to be of extreme value in our work on orbital reconstruction. Since its development it has been performed routinely in all of our cases prior to operation. I have chosen the term, orbital "blow-outs," as being most descriptive of many of these injuries. They occur as a result of the terrific impact produced by the modern high velocity missiles. We are also seeing cases of a somewhat similar nature in accidents incurred in civilian life. Enophthalmos may be so marked that at times only the up-turned cornea of the globe is visible. In the majority of these cases, however, the eye has been destroyed and has been removed before the patient reaches us.

Correction of these cases requires elevation and restoration of the orbital contents to their normal position and filling of the defect with either preserved cartilage or some inert material such as tantalum mesh as devised and developed by Dr. A. D. Ruedemann. With tracings available the required thickness of the cartilage, tantalum, or other type of packing utilized can be estimated to an accuracy of one or two millimeters.

In many instances ordinary examination, including routine x-ray procedures and palpation of the orbital margins, have proved misleading in our attempt to evaluate the extent of injury present. Such an accurate analysis of the true extent of injury is important since certain patients in whom a cosmetic operation might result in fatality, may be eliminated from surgical consideration entirely. This was well demonstrated in 2 cases which at first appeared well suited to the usual surgical procedure. Preoperative orbital tracings of both patients, however, indicated a severe bony loss of the posterior orbital walls with the probability of brain herniation underneath the conjunctiva, a condition which was later confirmed by the neurosurgeon in both instances. (A case of this nature was presented at this time.)

A surgical approach through the conjunctiva is advocated. It leaves no external scar and in our hands has proved more satisfactory than the old incision through the skin.

We are indebted to Dr. A. D. Ruedemann, the civilian ophthalmic consultant to Crile General Hospital, for this technic. We wish at this time to again express our sincere appreciation for the hours he spent with us each week and for the help and suggestions given us during the development of these procedures.

(Three case reports were then presented showing pre- and postoperative photographs. The orbital tracings on these patients made before and after surgery were reviewed.) One patient had incurred a severe fracture involving the orbital floor, entire ethmoid capsule and cribriform plate resulting in a complete loss of the sense of smell, bilateral. At the time of the original orbital reconstruction the depressed area in the posterior superior aspect of the ethmoid labyrinth was not

corrected because of the possibility of opening up the old fracture lines in this area extending into the anterior fossa of the skull. In spite of this precaution this patient, however, did develop some drainage of cerebral spinal fluid from the orbital incision along with rather severe headaches for the first three or four days following operation. Following penicillin and sulfadiazine in adequate doses the headaches disappeared and the operative orbital approach healed without further complication with complete cessation of drainage of cerebral spinal fluid. The ultimate cosmetic correction in this case was not quite satisfactory because of the incomplete correction mentioned above. This resulted in some residual depression remaining in the superior nasal aspect of the upper lid. This mild deformity was subsequently corrected by a second stage procedure which consisted of placing a curved acrylic contour stent between the tarsal orbital fascia and the orbicularis muscle as advocated by Sugar and Forestner[1]. (A short 16 mm. Kodachrome movie was shown demonstrating the use of orbital tracings and the operative procedure.)

Lt. Colonel Shiflett, in developing this original method of orbital tracings, has made it possible for the ophthalmoplastic surgeon to approach the problem of orbital restoration with much more assurance and peace of mind than was formerly possible. The true extent and location of orbital injuries and deformities can now be accurately determined and charted prior to operation. This three dimensional tracing may be made a part of the patient's permanent record and is thus available in the operating room for reference at the time of surgery. By knowing in advance the location and amount of correction required the surgeon can proceed with the reconstruction with greater promise of success than has been heretofore possible, and, by reference to such a diagram, dangerous areas may be avoided. In some instances certain patients in whom a cosmetic operation might result in fatality may be eliminated from surgical consideration entirely.

## REFERENCES

1. Sugar, H. S. and Forestner, H. J.: Methacrylic resin implant, for sunken upper lid following enucleation, Am. J. Ophth., 29: 993-1000 (Aug.) 1946.

# SUGGESTIONS ON THE SURGICAL REPAIR OF FACIAL PALSIES OF OTITIC ORIGIN

KARSTEN KETTEL, M.D.
HILLEROD, DENMARK
BY INVITATION

IT is with a feeling of deep gratitude and pleasure that I can speak today to American otologists on the surgical repair of the facial nerve. In Denmark we are impressed by the results obtained over here; especially by the work done by Bunnell and by Ballance and Duel, who have created the foundation on which the treatment rests.

In 1939 I paid my first visit to the United States and, thanks to what I learned from Dr. T. G. Tickle, on my return I was able to introduce the modern surgical treatment of facial palsies into my country. During the last seven years I have operated on 150 patients after the methods of Bunnell and Ballance and Duel comprising 100 cases of otitic palsies and 50 patients suffering from Bell's palsy. The time today only permits me to forward a few suggestions on the surgical treatment of otitic palsies, but a detailed report of my results will soon appear in the *Archives of Otolaryngology*.

Regarding both sorts of palsies, however, the capital question is: Which cases should be treated conservatively and which should be operated on? Ballance and Duel wrote, "It has been said recently that the difficulty in deciding on operation is that we do not know whether the nerve is crushed, divided or merely involved in inflammation. We do not want to know. The question of operation has little to do with how the paralysis was produced. The reason for operation is that the patient has facial palsy. In all early mastoid cases, whether inflammatory or caused by direct injury, an operation should be done immediately; the sooner it is done, the easier the operation, the less the damage to the nerve and the better the condition of the muscles." They continue, "The time to operate is—NOW."

Tickle relies upon the faradic reaction, saying that the operation should be done in cases where the response is negative, and English surgeons have adopted this line. My opinion differs from that of these physicians. I believe that the faradic reaction is not a reliable indicator. I have seen several cases of otitic palsies, as well as Bell's palsies, recover completely and spontaneously under a conservative treatment even though they had negative responses to faradic stimulation. This apparent discrepancy is easy to understand. The faradic current is a high frequency alternating current, the length of phase and frequency of which cannot be varied. When a muscle is paralyzed a transitory hyperirritability sets in (diminished chronaxia) immediately superseded by a reduced irritability (increased chronaxia). It is simply impossible to obtain contractions by means of the faradic current, as the duration of the irritation (the length of phase measured in milliseconds) is too short to influence the muscle, the chronaxia of which is increased. To rely upon the faradic response as an indicator for operations means to perform a great number of superfluous operations, be-

Presented at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 13-18, 1946, Chicago, Ill.

cause to do a decompression in all cases of otitic palsy is highly incorrect. Every otologist of experience knows that in many cases, facial palsy will disappear under adequate treatment of the disease of the ear.

I have tried accordingly to solve empirically the problem of indications for surgery in the otitic palsies by re-examination of 264 cases treated without operation as regards the nerve. Upon the basis of this study, I would suggest the following procedure:

A. PREOPERATIVE PALSIES

1. *A preoperative palsy in association with acute otitis media* is by no means rare (½ per cent) and when it occurs at an early stage of the otitis it is in no way a symptom of mastoiditis. The ear disease should be treated as though no facial palsy were present. In itself, facial palsy does not indicate the need of a mastoidectomy. If the palsy is steadily increasing however, or if it does not decrease after observation for a reasonable time and in spite of free drainage through the drum membrane, a mastoidectomy should be done. When other considerations indicate the need of mastoidectomy, a facial palsy adds additional justification for operation. If, however, facial palsy appears two or three weeks after the beginning of the otitis, it does suggest the need of mastoidectomy which as a rule will be indicated by other signs of surgical mastoiditis.

In all the 264 cases which I reviewed the palsy disappeared completely under the treatment outlined above. Just as facial palsy in association with acute otitis media is not a symptom of a mastoiditis, and consequently in itself does not indicate a mastoidectomy, when facial palsy complicates acute mastoiditis, there is no reason to do a decompression of the facial nerve in order to secure the future normal function of the nerve.

If a surgical mastoiditis is present, a complete simple mastoidectomy should of course be done. In 16 out of 18 cases in which only a complete simple mastoidectomy was done, the palsy disappeared, and consequently it is evident that simultaneous mastoidectomy and decompression is not indicated. In the cases in which the palsy does not disappear following mastoidectomy, a decompression should be considered after observation for two months. This time limit has been chosen because my re-examinations showed that in all cases of otitic palsies, in which the patient regained full mobility of the facial musculature, the first signs of returning function were noted before this limit was passed.

2. *Preoperative palsy in association with chronic otitis media* occurs in 1.7 per cent of cases and always indicates the need of radical mastoidectomy. Should a decompression be made simultaneously? Ballance and Duel consider it foolish not to deal with the palsy at the end of the operation by making a decompression of the facial nerve.

In my opinion this depends upon whether or not there is a fistula of the facial canal. In 9 out of 11 cases in which a fistula was demonstrated but no decompression was made, a permanent facial palsy was the result. In only 2 cases did the facial palsy disappear after radical mastoidectomy alone. I believe therefore that a decompression should be done in these cases. I have been further confirmed in this opinion, as subsequently during decompression in several cases I discovered that the osteitis from the fistula had made its way well into the facial canal where the nerve too had been involved in the inflammatory process.

Consequently, when a fistula in the facial canal is demonstrated during radical mastoidectomy, it should be widened till every trace of osteitis in the wall of

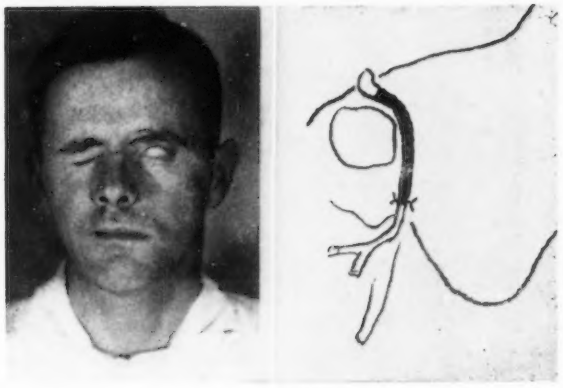


FIG. 1.—Nerve-grafting 14 mm.

the fallopian canal is removed. Then the nerve sheath should be split. Complete removal of infection in the fallopian canal may be obtained in some cases by a partial decompression, in others it requires opening of the facial canal from the ganglion geniculi to the stylomastoid foramen.

If at the operation no fistula of the facial canal can be discovered, the patient is observed for two months. If at this time there is no sign of returning function, a decompression should be done.

B. POSTOPERATIVE PALSIES

3. *A postoperative palsy in immediate association with the operation* has a very grave prognosis. Of 111 cases where the palsy was noted directly after a simple or a radical mastoidectomy about 60 per cent had a massive palsy on re-examination years after, and if one makes a thorough analysis of these cases it is found that 100 per cent had a permanent palsy when the nerve had been severed.

Thanks to American surgeons most of these patients can now be cured. I have done 50 nerve graftings or nerve sutures. I cannot tell you the results of 10 cases yet, as these operations have been done recently, but 36 out of 40 patients or 90 per cent regained mobility. It must, of course, be understood that not even the most successful grafting or suturing can restore full mobility. The results, however, ought not to be judged by what is lacking in a 100 per cent function, but must be compared to the situation as this would have been if the nerve had not been repaired. In none of the 36 successful cases can the palsy be noted when the face is at rest, all of the patients can smile and laugh, most of them can close and screw the eye up, but only 3 patients can wrinkle their foreheads (figs. 1-6).

The reason why the operation in 4 cases proved a failure is probably that the muscles had partly atrophied in 2 patients at the time of operation and accordingly the repair should not have been tried in spite of the patients' urgent wishes. In the other 2 cases the patients were more than 60 years old, and the power of regeneration lowered. The age in itself, however, is no contraindication, as in one case, a man 73 years old, I obtained a beautiful result (a graft of 30 mm.).

Speaking of nerve graftings and nerve sutures I want to stress certain points:

a) When the palsy appears in direct association with the operation, the cause should immediately be established.

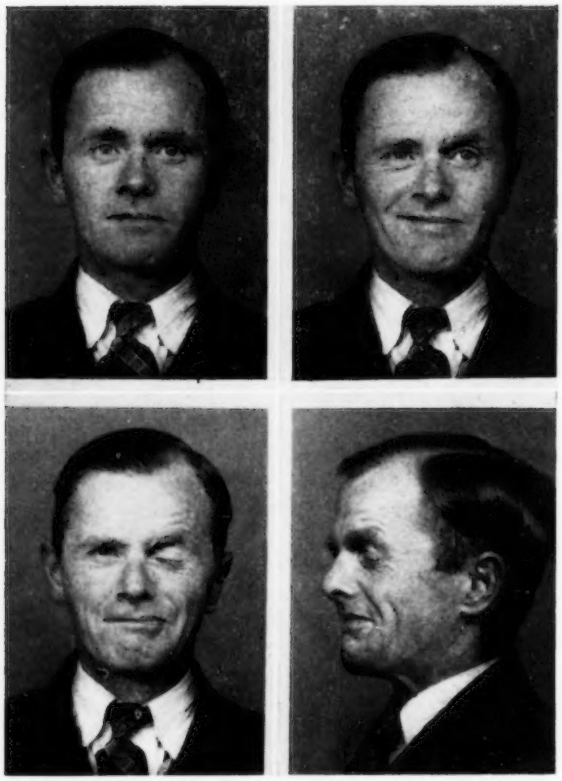


FIG. 2.—Same as figure 1. Twenty-one months after the operation. Note that the patient can wrinkle his forehead.

There is no excuse for waiting if the state of the patient permits exploration. What is the use of waiting? As most of the palsies are of traumatic origin, the repair should be made immediately. If you wait, it is much more difficult to find the nerve stumps, embedded as they are in granulations or cicatricial tissues. I can speak of this, as most of my patients have been referred to me after months or years of observation in vain. The earlier the operation is done, the easier it is, the better the condition of the muscles and, last but not least, one can tell the prognosis and put his mind and that of the patient at rest. I do not hesitate to predict that if the repair is made immediately and properly the patient has almost a 100 per cent chance of regaining his emotional expressions.

b) It has been proposed that repair be delayed until the infection is gone and the field clean. I strongly object to this. In almost all my cases the field was infected, but in one only, and for quite different reasons, was the graft expelled. If the operation is done immediately, the graft covered with gold foil, and the cavity filled with strips of gauze soaked in penicillin, and the dressing left alone for seven days, when it is removed, the cavity is clean.

c) Should the nerve graft or a nerve suture be preferred? Bunnell insists on suture, saying that in this way the neurofibrils, in growing downwards, only will have to pass one hindrance whereas if a nerve graft is inserted, they

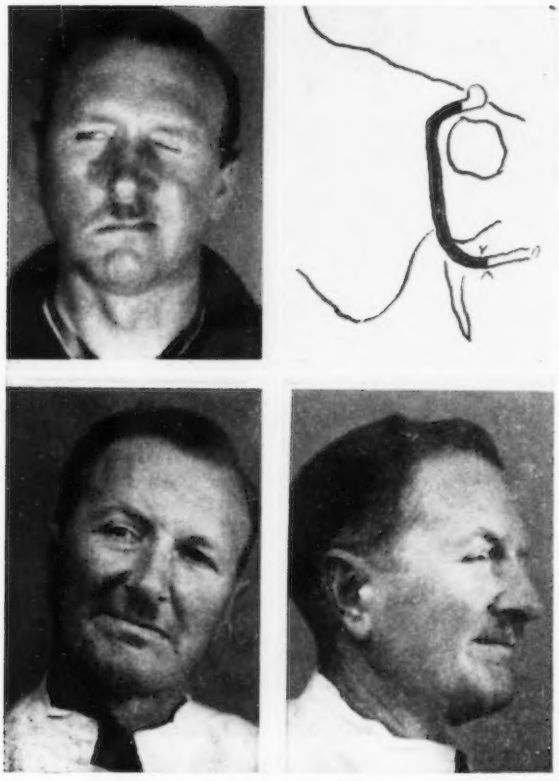


FIG. 3—Nerve-grafting 30 mm. Before and two years after operation.

will have to pass two. Ballance and Duel, on the other hand, prefer grafting because the nerve remains in its bed with an undisturbed blood supply.

My results are equally good with both methods. If the nerve can be sutured without removing it from its bed, that should be done. I personally prefer grafting to re-routing and I would stress the fact that if in doing re-routing it proves necessary to sacrifice the contents of a sound middle ear, this operation is strongly contraindicated.

d) Which nerve should be used as a graft? Most authors recommend the lateral cutaneous femoral nerve, but very often it is difficult to find and I much prefer to use n. ilioinguinalis, as suggested by Bergström.

e) Should the graft be held in place by means of sutures? No. A suture is only required if the reunion takes place in the soft tissues, and ther a tantalum wire should be used, as suggested by Lathrop. Yesterday, however, I heard a most interesting paper read by Dr. J. A. Sullivan, who united the stumps in the soft tissues by means of fibrin, and I certainly shall try this.

4. *A postoperative palsy occurring after a free interval* generally has a very good prognosis. When due to pressure of tamponade the palsy generally disappears after its removal.

If, in a very few cases, after two months observation no signs of returning mobility can be observed, a decompression should be done.

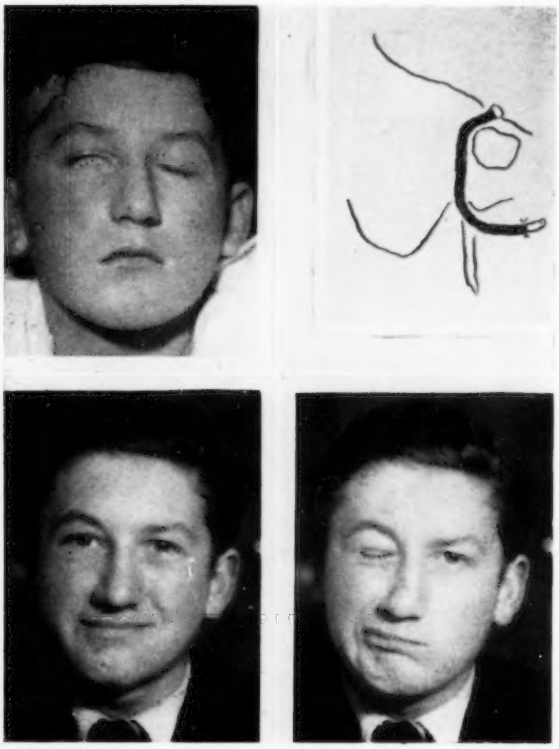


FIG. 4—Nerve-grafting 35 mm. Before and two years after operation.

If, in spite of a first operation there is a progressive osteitis, a revision is, in most cases, sufficient to make the palsy disappear. Should a fistula of the facial canal now be discovered, a partial or complete decompression according to circumstances should be done. If no fistula is found the patient is observed for two months before decompression is done.

If finally a hemorrhage in the facial canal is suspected a decompression may be considered.

## DISCUSSION

J. A. SULLIVAN, M.D., Toronto, Ontario, Can.: It is a great privilege and honor for me to discuss this presentation of Dr. Kettel's, an outstanding otologist of Denmark. It was in 1938 that I had the privilege of visiting his country, and I am well aware of the work that they are doing over there. Dr. Kettel is to be congratulated not only on the results that he has obtained, but on some of the points that he has brought out in an attempt to clarify problems that have confronted us particularly in regard to the concept of Bell's palsy and also in the return of the functions of the occipitofrontalis muscle.

Dr. Kettel may be quite correct in his idea about the use of the faradic current. However, my experience is different. By means of a special faradic applicator I obtain useful clinical information from stimulation of the nerve any time from a period of seventy-two hours to fourteen days after injury. A nerve in a dog can be cut and a faradic current directly applied to it and faradic response will be present up to a period of fourteen days. After that period the reaction to stimulation with faradic current disappears. Therefore, in cases of Bell's palsy, with complete paralysis as far as the neurogenic impulse is concerned, I believe that the direct exposure of the nerve at the stylomastoid foramen is indicated, with

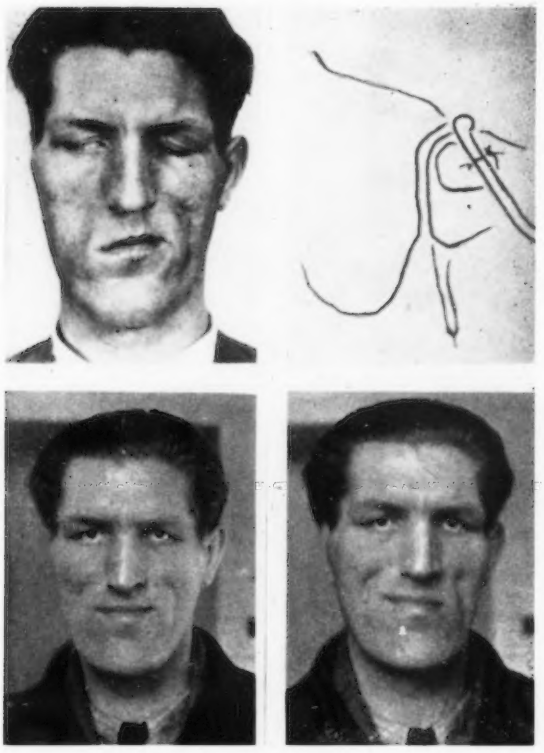


FIG. 5.—Nerve-suture (re-routing). Before and two years after operation.

the exact application of the electrode to the nerve. In that way we are not doing the patient any harm and we are getting the fullest information we possibly can.

He may be right as to his suggestion that one should wait two months to see if spontaneous regeneration takes place before advising decompression. Every case of facial paralysis does not warrant decompression. In mastoiditis, we usually have evidence of what has occurred during the mastoid operation. In Bell's palsy, we know the anatomical pathway, and we must utilize our knowledge of the function of the chorda tympani nerve and the greater superficial petrosal nerve to determine the site of the lesion. In acute surgical mastoiditis, accompanied by facial paralysis, complete exenteration of the mastoid process alleviates the paralysis in most of the cases. This is similar to the results obtained in apical petrositis where complete exenteration of the mastoidal portion of the temporal bone will in many instances obviate the necessity for surgical intervention on the petrosa.

In the chronically discharging ear with facial paralysis, I believe that we are dealing with a different problem. The onset of paralysis in chronic suppurative otitis media is a most important sign and one that warrants a radical operation, but does not necessarily at the moment indicate the necessity of a decompression of the facial nerve. It is my experience that a facial paralysis that has come on in the course of a chronic suppuration of the ear will require, when that type of ear quiets down, a decompression in order to give the best results.

Emotional expression is usually regained in cases which have been subjected early either to a decompression operation, an end-to-end union, or a nerve graft. However, where a nerve graft has been done any time after three to six months following injury, it is most unusual for true emotional expression to return. Return of function of the facial nerve is partly an educational response and it depends on the relative intelligence of the patient.

FIG. 6—Nerve-suture (re-routing). Before and two years after operation.

The return of function of the occipitofrontalis usually occurs in cases which have had early operation. Why this does not occur in late cases has been a question that has not been completely settled. It is my opinion that it is owing to the fact that the facial muscles except the more distal part of the facial musculature, are not covered with fascia, and, therefore, the muscle as it is inserted into the skin becomes denervated faster than ordinary muscle which has a fascial covering. This is particularly true in the case of the frontalis and superciliary muscles.

Nerve graft versus suture. I am wholeheartedly in support of the use of a nerve graft, and I believe that Dr. Kettel's results show that that procedure is an established practice. I do not believe that the facial nerve at any time should be dislocated from the fallopian aqueduct, as it has its own nerve bed and it has its own blood bed, and in preserving these we are offering the best possibility for return of function.

Bell's palsy is a big subject and, as I have previously stated, we must make the differential diagnosis between the peripheral and the central types of Bell's palsy. Eighty-five per cent of the cases of Bell's palsy recover, as Dr. Kettel has stated. It is the 15 per cent that do not recover that we have our trouble with, and I repeat that where we have a complete neurogenic block, that case must be subjected to a decompression operation.

I would say one more word about nerve grafts, and that is a long graft is not a contraindication to its utilization in the mastoid region, because now they are using grafts in peripheral nerve surgery anywhere from two to three inches long. Two things have given the greatest trouble in nerve grafting—the formation of endoneural scar at the distal end of the graft and inaccurate coaptation of the nerve ends in the use of suture. With the new plasma technic, I believe that a greater degree of accurate approximation of nerve can be brought about.

I should like again to congratulate Dr. Kettel on his excellent paper and results and the

beautiful English that he used to express them to us over here in this country.

Thank you.

EDGAR P. CARDWELL, M.D., Newark, N. J.: I had the opportunity at one time to operate on a type of case of facial palsy that has not been discussed, unless I missed it. That is a long nerve graft directly from the nerve stump into the musculature. This was first done experimentally probably fifteen or twenty years ago. In this particular case, a long section of the trunk and all the branches of the facial nerve were lost in the removal of a tumor. In the repair, five nerve grafts, all of which were around 10 centimeters long, were fanned out from the stump into the facial muscles.[2]

Since that time, in this war, Dr. Frank Lathrop, according to reports from Dr. Norton Canfield,[1] has used some long grafts directly into the muscle, and good results are being obtained in cases that one might otherwise fail to do anything for.

My reason for working on this type of case was that I felt that there was nothing to lose. In my opinion, you can all take heart in the worst type of paralysis cases because the facial nerve has a tremendous faculty for regenerating.

I want to congratulate Dr. Kettel. I hope to talk to him and visit Denmark to see some of the work that he is doing.

Thank you.

## REFERENCES

1. Canfield, N.: Otolaryngology in the European Theatre of World War II, Ann. Otol., Rhin. & Laryng., 55:108-120 (March) 1946.
2. Cardwell, E. P.: Direct implantation of free nerve grafts between facial musculature and facial trunk, Arch. Otolaryng., 27:469-471 (April) 1938.

KARSTEN KETTEL, M.D., Hillerod, Denmark: Thank you, gentlemen, for your kind words. You doubtless are much more experienced in this field. I am just a beginner, and, therefore, I am very glad to learn that we are agreed as to the treatment.

Much more could be said about Bell's palsy, but I just want to stress that Bell's palsy is not an etiologic entity. It is a diagnosis. We do not know to what the paralyses really are due. I only wanted to show you that at least 20 per cent of the cases of so-called Bell's palsy are masked otitic infections. Many of the patients have been exposed to colds. I think it was in that way that the resistance of the organism was lowered and that is the way they got the condition.

Of course, I know that in the future the indications for operation on Bell's palsy will have to be revised. I only tried to outline a treatment so we can do it today.

Thank you very much.

# AN ACRYLIC OBTURATOR EMPLOYED IN THE REPAIR OF AN OBSTRUCTED FRONTONASAL DUCT

JOHN B. ERICH, M.D.
*and*
GORDON B. NEW, M.D.
ROCHESTER, MINN.

OTOLARYNGOLOGISTS have encountered fewer difficulties in preparing than in maintaining an artificially created passage from the frontal sinus to the nasal cavity in patients with an occluded infundibulum. Extranasal operations devised to create a frontonasal duct for adequate dependent drainage of the frontal sinus have become standard and reliable technics. But surgical procedures intended to prevent or overcome the tendency toward partial or complete cicatricial stenosis of this newly constructed channel have been decidedly unsuccessful in a great many cases. Before making a plastic closure of an external frontal sinus fistula, it is essential that the frontonasal duct be open sufficiently to maintain drainage. If free and permanent drainage into the nose is not present, the fistula may recur. It was in making a frontonasal duct for this purpose that the present method was worked out. In a search for something to obviate this difficulty, we have found that an acrylic obturator (fig. 1), if properly constructed and if worn for a sufficient length of time, will avert such a stenosis.

As is well known, any cavity or tract artificially prepared in the body becomes quickly occluded by scar tissue unless it is held open by some mechanical means. After a time this tendency toward contracture gradually diminishes until finally the cavity will stay open without mechanical aid. In treating strictures of the larynx, nostrils, external auditory canals and so forth, we have found that an obturator must be worn for approximately six months before the danger of contraction of the scar tissue in the walls of these structures has passed. Similarly, following external operations to re-establish the patency of a stenosed frontonasal duct, we have discovered that a corresponding length of time is necessary before an obturator can be removed from the duct without the prospect of subsequent cicatricial occlusion. The usual plan of mechanically maintaining the patency of the canal for a few days or weeks or even for three months, as described by Harmer and Hogg,[1] seems inadequate in the average case.

Obturators prepared to keep open a frontonasal duct have been constructed from different substances. Most surgeons employ either metal tubes or more frequently rubber tubing for this purpose. While obturators of such materials do maintain patency of the duct, both have disadvantages. Metal tubes are difficult to keep in position over a long period, while rubber tubing tends to elongate and change shape on exposure to the secretions in the nose. When a rubber tube has been displaced, it often is difficult to reinsert it into the frontonasal canal. Metal tubes do not inhibit the formation of granulation tis-

From the Section on Laryngology, Oral and Plastic Surgery, Mayo Clinic, Rochester, Minn.

Presented before the Academy Section on Otolaryngology and the Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 18, 1946, Chicago, Ill.

FIG. 1a. Acrylic obturator employed to maintain the patency of an artificially created passage from the frontal sinus to the nasal cavity in patients with an obstructed frontonasal duct. It is necessary to use such an obturator for about six months, at the end of which time there is no longer any tendency for this newly constructed channel to contract. *b*. Obturator being held external to the nose to show approximate position which the appliance would assume in the nasal cavity.

sue in and around the duct, and rubber tubes actually stimulate the growth of granulations. Moreover, acting as a sort of irritating foreign body, metal and rubber tubes are likely to cause a discharge, often with a foul odor. These, then, are among the difficulties encountered when the usual metal or rubber obturators are employed.

We have found, as was previously stated, that an obturator composed of one of the dental acrylic materials (we use veronite) overcomes these difficulties. Such acrylic substances are employed by dentists in the preparation of artificial dentures. Acrylic obturators of proper contour have the following advantages: 1. They are worn without discomfort. 2. They never change shape. 3. After being worn for two or three weeks, if they should become displaced or are removed, they can easily be reinserted even by the patient himself. 4. They are so smooth, nonporous and unirritating that they inhibit the formation of granulation tissue. As a matter of fact, granulations which may be present at the time that the obturator is inserted quickly disappear. 5. They produce no discharge and consequently no odor.

An acrylic obturator (fig. 1) for maintaining the patency of a surgically constructed frontonasal duct is prepared as follows: (We shall omit the details in the technic of constructing a passage from the frontal sinus to the nasal cavity in patients with an occluded infundibulum except to say that an external approach to the frontal sinus is employed.) As soon as an adequate frontonasal canal has been established and while the cavity of the frontal sinus is still exposed through the exter-

FIG. 2. Rubber tube in the reconstructed frontonasal duct. This type of roentgenogram is helpful in the construction of an acrylic obturator; see text.

FIG. 3. External frontal sinus fistula of long duration and secondary to osteomyelitis of the wall of the frontal sinus cavity. Since the normal frontonasal duct was occluded, a new channel into the nose was created and held open for six months with an acrylic obturator. See figures 4 and 5.

nal incision, a rubber tube of proper size is inserted through the nostril and through the newly prepared duct into the cavity of the frontal sinus. This piece of rubber tubing should be sufficiently long so that it protrudes out of the nostril for a short distance. It is held in position with adhesive tape.

After the inflammatory reaction of the operation has subsided, usually a period of about one week, a roentgenogram of the patient's skull is obtained; such a film (fig. 2) reveals a contour and length of the rubber tube as it passes through the nasal cavity and the frontonasal duct into the frontal sinus. A sheet of pink dental base-plate wax then is heated and rolled into a tubelike structure approximately the same size as the rubber tube in the patient's nose. While still warm, it can be molded to a contour simulating that which the rubber tube assumes in the nose as is exhibited on the roentgenogram (fig. 2). The upper end of the wax obturator, which is to enter the frontal sinus, should be tapered slightly while the lower end, which will protrude through the nose externally, should be made to extend 2 or 3 cm. beyond the opening of the nostril.

We think that it is advisable at this stage to remove the rubber tube from the nasal cavity and insert the wax pattern through the nose into the frontal sinus. If it cannot be inserted with ease, its contour should be changed as required; such alterations can be easily accomplished by warming the wax and molding it manually. After it is certain that the wax obturator is of proper size and shape, the rubber tube is reinserted into the nose and the wax pattern is sent to a dental laboratory for processing in one of the dental acrylic materials. When the acrylic appliance is ready, the rubber tube is removed from the patient's nose and is replaced by the obturator. That portion of the obturator

FIG. 4. See figure 3. *a* Dotted line indicates the position of an acrylic obturator in the right nasal cavity, the reconstructed frontonasal duct and the frontal sinus. *b*. First step in plastic repair of external frontal sinus fistula. Flap of skin adjacent to defect is elevated and sutured back in position. This delay in transference of the skin flap insures its blood supply from the pedicle along the mesial aspect of the defect. *c*. Two weeks later. Pedicle flap of skin re-elevated, turned over and into the defect, and sutured in this position; this flap makes an epithelial covering on the frontal sinus side of the repaired fistula. A flap of skin (under the arrow) is elevated and slid across the defect to make an external epithelial covering. The triangular shaded area represents a fold of skin which must be excised to prevent buckling of the skin in this region. *d*. Completed plastic repair of fistula.

FIG. 5. See figures 3 and 4. External frontal sinus fistula closed.

FIG. 6. See figures 3, 4 and 5. Six months later a roentgenogram shows normal frontal sinus and wide open frontonasal duct into which a probe can be passed readily.

which protrudes out of the nose can be cut off flush with the lower border of the nostril. Usually no fixation is required, although a piece of adhesive tape across the nostril may be necessary in some instances. The obturator should not be made hollow, since a hollow obturator would permit nasal secretions to cake within; secretions of the frontal sinus work out easily around the obturator. Should an external frontal sinus fistula be present (fig. 3), no attempt should be made to close such a tract until the acrylic obturator can be permanently removed. Such an external fistula permits visual inspection of the upper end of the obturator, which should project into the frontal sinus cavity. Following removal of the obturator, a plastic closure of the frontal sinus fistula can be accomplished as illustrated in figures 4, 5 and 6.

The obturator should be kept in position for approximately six months if the patency of the newly constructed frontonasal duct is to be assured. However,

the obturator can be removed as an office procedure at any time should the physician wish to inspect the passage that it occupies.

## REFERENCES

1. Harmer, W. D. and Hogg, J. C.: Operations on the nose and pharynx. In Turner, G. G.: Modern operative surgery, Ed. 3, London, Cassell and Company, Ltd., 1943, vol. 2, pp. 1380-1437.

## DISCUSSION

GORDON B. NEW, M.D., Rochester, Minn.: I have always felt that the usual method of postoperative drainage of the frontal sinus by means of a rubber tube which is left in the seven to thirty days allows frequently the stricture of the nasofrontal duct, and these strictures are usually very difficult to take care of. This method that Dr. Erich has presented has given excellent results and I believe should be used in these cases.

Dr. Erich's presentation was excellent and I wish to congratulate him.

# CONSERVATIVE SURGERY OF CANCER OF THE LARYNX

JUSTO M. ALONSO, M.D.
MONTEVIDEO, URUGUAY

WHEN we have to treat a patient suffering from cancer of the larynx we feel that the two principal objectives to be striven for are, first, the preservation of the life of the patient and, second, the preservation, if possible, of the function of the organ of speech.

Two methods of attempting to secure these ends are available: surgery and radiation therapy. Surgery provides a greater percentage of lasting cures, but it often causes mutilation. Radiation therapy preserves the function of the organ, but is not so successful in achieving lasting "cures" and the results are sometimes misleading and contradictory. The roentgenologists, however, are constantly trying to better their apparatus and technic.

It is possible to secure a better result by combining both types of therapy. I shall not review the indication for these procedures or their combination. This has already been done in the official report on "Tratamiento del Cáncer Laríngeo," presented during the second South American Congress of Otolaryngology (Montevideo, November 1944).

In varieties of laryngeal cancers other than those in which the tumor was limited to a vocal cord, surgeons of almost all countries have made attempts at total extirpation of the tumor without impairing the function of the larynx. I shall not discuss the technics used to remove a tumor from a vocal cord nor the classical hemilaryngectomy (Glück-Sorensen) nor the Hautant hemilaryngectomy, nor the simple removal of the larynx.

I shall make a summary of the technics that I use in two principal types of extrinsic cancer of the larynx.

1. Those cancers which involve the pyriform sinus in which a partial vertical laryngectomy is done.
2. Those cases in which the cancer involves the vestibule of the larynx, the epiglottis and/or the vestibular bands in which a partial horizontal laryngectomy is done.

1. The cartilages of the thyroid are similar to two covers of a book which is open at an angle of 70°: they protect the larynx and the hypopharynx as would a shield. The front part of the thyroid cartilages is in direct relation with the anterior attachment of the vocal cords.

The external and back part of the thyroid cartilages protects the prolongation of the hypopharynx that extends between the back part of the larynx and the thyroid cartilage on each side (fig. 1).

The constrictor muscles of the pharynx that close from behind the triangular prism whose two outside and front sides are constituted by the thyroid cartilages are inserted in the vicinity of the back edge of the thyroid cartilage.

When one sees a tumor of the pyriform sinus whose development is early and has not infiltrated widely, one should determine especially if it infiltrates towards the outside or inside wall. In the latter case one ought to try to determine the area of the larynx invaded

Presented before the Academy Section on Otolaryngology and the Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 18, 1946, Chicago, Ill.

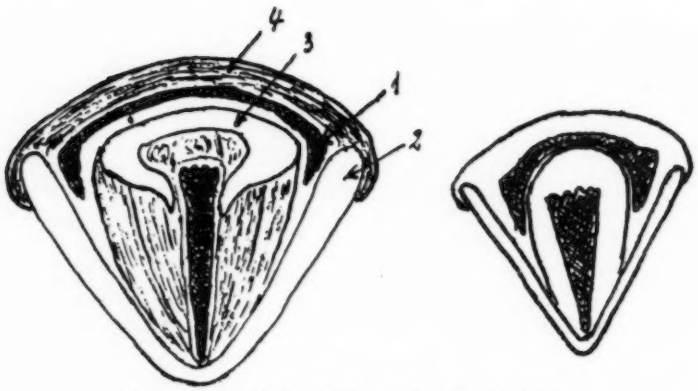



FIG. 1—The relation of the vocal cords and larynx to the thyroid cartilage.

and the extent to which the cancer has spread. In some cases the tumor is seen to have originated in the anterior dihedral angle of the pyriform sinus and developed in both an inwards and outwards direction invading both walls as it grew.

Cancer of the hypopharynx can appear in any part of the pharynx, but originates most frequently in the pyriform sinus. At first there are very few symptoms, but later difficulties in swallowing and hoarseness indicate invasion and stenosis of the larynx, and signs of involvement of the regional lymph nodes are present. The roentgen-ray findings made after swallowing opaque media and the tomograph help us to make the diagnosis. Direct laryngoscopy must also be done. Past surgical technics generally have had little success in eradicating this type of lesion. Trotter made a valuable addition to our technic by cutting the hyoid bone and part of the thyroid cartilage, and pushing the entire flap laterally so as to give easy access to the pyriform sinus. Henry Orton made some modifications of this technic.

I have published an article on this subject in the *Revista Brasileira de Otorinolaringologia*, 1940, in which I have tried to systematize the technics of extirpation of this type of cancer. I use the following flap: a vertical incision is made from the hyoid bone to the top edge of the cricoid cartilage one or two centimeters towards the diseased side from the center line. From the top end of the first cut an incision is made, first horizontally and towards the back following the hyoid bone and then in a backward and upward arch behind the angle of the lower jaw so as to reach the inferior part of the meatus auditorium externus. From the lower end of the vertical incision another incision is made in an arch backwards and downwards until it reaches the front edge of the sternomastoid muscle as it passes

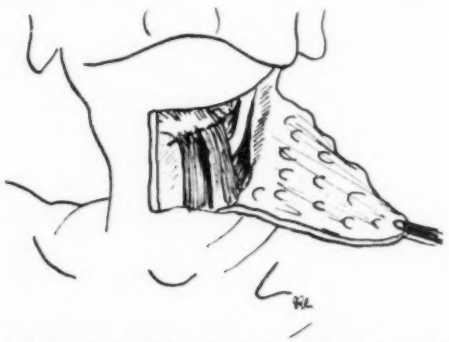


FIG. 2—Our incision for tumors originating in the pyriform sinus. This shows the excellent exposure of the lateral aspects of the larynx and pharynx. If the incisions are extended backward to include the sternomastoid muscle, the carotid sheath may be explored and the jugular vein extirpated.

downward to insert in the sternum. The flap formed by these incisions includes skin, platysma, and superficial fascia. If the incision is carried further back inferiorly, it may include the sternomastoid muscle and allow us to explore the region of the jugular vein (fig. 2).

It depends upon what is found whether or not the sternomastoid muscle or the internal jugular vein is extirpated. The omohyoid and the deep cervical fascia should be cut to expose the lower portion of the internal jugular vein for exploration or extirpation. Dividing the posterior belly of the digastric muscle gives access to the upper portion of the carotid region.

In the majority of the cases when the lymph nodes are extirpated the facial thyrolingual trunk and the facial vein are cut as they come out from beneath the submaxillary gland; the infrahyoid muscles are extirpated on the involved side (or they are divided if the tumor is not malignant), the superior artery of the larynx is tied, the superior nerve of the larynx is cut, and the superior horn of the thyroid cartilage is removed. In this way we have ample room to reach the pharynx. The outer part of the thyrohyoid space is opened or cut (that is to say, around the epiglottis) and the space is explored in all its extension with both the finger and the sight, and in all directions, but especially downwards and inwards (towards the larynx). We must study it carefully before extirpating it (fig. 3).

Regarding the lateral and horizontal extension of cancer of the pyriform sinus, three principal varieties are observed. In the first, only the outside or external wall is invaded by the tumor. In the second, the internal wall is invaded superficially. This superficial invasion of the internal wall may be the primary site of the tumor or a tumor of the external wall may have secondarily invaded the internal wall. In the third variety, the anatomical elements of the internal wall of the pyriform sinus may be deeply invaded by the tumor, and also therefore the hemilarynx on the same side.

FIG. 3.—The exposure of the pharynx obtainable by the author's approach for lateral pharyngotomy.

If the disease involves only the outer wall of the pyriform sinus (first type), an incision is made in the homolateral thyroid lamina that begins at its superior edge about one centimeter from the medial and superior angle and continues downwards and outwards until it reaches the back and lower angle, in the vicinity of the inferior horn of the thyroid cartilage. Thus we reach directly to the bottom of the pyriform sinus whose mucous membrane is cut in the deepest part (that is to say, furthest forward). This incision is guided not only by direct vision but also by the finger in the pyriform sinus. A horizontal incision of the thyrohyoid ligament is completed from the midline to its lateral attachment to the hyoid bone. The resulting flap, made up partly of the lamina of the thyroid cartilage and

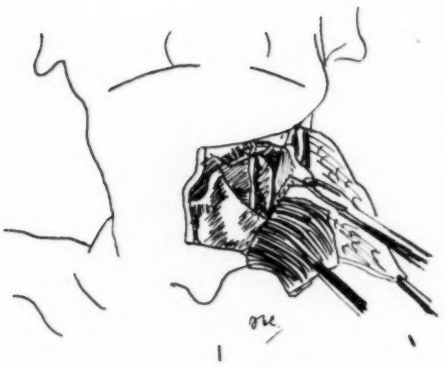


FIG. 4—Demonstrating the flap of the lamina of the thyroid cartilage and the mucosa of the pyriform sinus just before it is cut away.

the mucosa of the outer side of the pyriform sinus is pushed back and extirpated, cutting it well posterior to the tumor (figs. 4 and 5).

If the tumor invades the anterior angle of the pyriform sinus or its medial wall without deeply invading the corresponding hemilarynx (second type) the incision of the thyroid cartilage should begin at the superior angle of the thyroid cartilage, a few millimeters from the middle line and should be directed downwards and slightly outwards, so as to reach the bottom edge of the thyroid lamina at a centimeter or more from the midline of the diseased side. In this way a larger exposure of the mucosa of the pyriform sinus is secured so that the surgeon may push it back, alone if the growth is superficial or together with the outer part of the muscles of the larynx if the infiltration is deeper. With this incision the internal part of the thyro-arytenoid muscle, its anterior tendon of insertion, the arytenoid cartilage, and the mucosa of the endolarynx remain untouched.

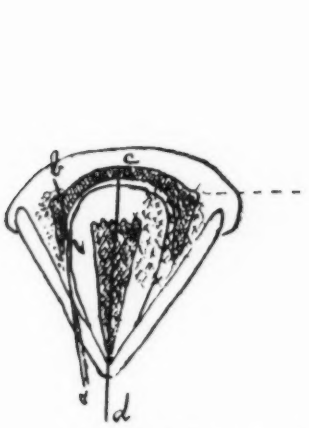
If the tumor extends downwards (third type), the line of incision will not end in the lower edge of the thyroid cartilage, but will extend down through the external part of the cricoid cartilage which is removed without injuring its internal perichondrium, so as not to expose the mucosa of the larynx in the subglottic area. The cricoid cartilage is separated downwards and backwards in order to give better access to the lower part of the hypopharynx and the mouth of the esophagus (fig. 6).

If the tumor extends superiorly, the half of the hyoid bone on the involved side is removed (Trotter). It is not necessary to cut the hyoid bone and make the hemiextirpation of the larynx if the tumor does not extend upward.

In tumors of the first and second types, all the flap is extirpated and the borders of the mucosa are sewn to the edges of the skin. The outer skin flap is sewn to the mucosa of the lateral or back wall of the pharynx, depending on the size of the extirpated zone. A lateral stoma of the pharynx is left; its side edges are formed by the skin of the flap, and the rest of the circumference by the suture of the external skin to the internal mucous membrane. Under the external base of the skin flap a small tube is left for a couple of days. The stoma is left open and free, so that saliva can come through.

Nutrition is given by a nasal tube and a loose, flat bandage should be put around the neck; if there is much saliva, a tube is used in the stoma connected to a continuous suction apparatus for four to seven days. A month and a half to three months later, the stoma becomes smaller, and at this time may be closed plastically in two planes. Rarely does it close of its own accord.

If the tumor invades deeply into the hemilarynx (third type), the pyriform sinus should be removed together with the larynx or the hemilarynx of the corresponding side. Using the flap described above, a classical hemilaryngectomy is done, together with the extirpation of the zone of the pharynx attacked, also leaving the stoma. In these cases we can frequently save the



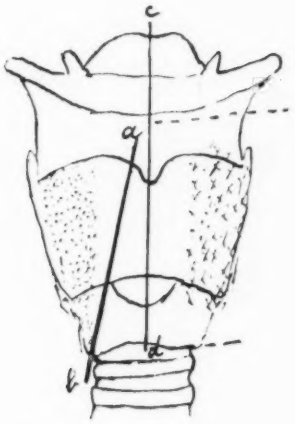




FIG. 5—View from above of the incision through the lamina of the thyroid cartilage.
FIG. 6—Demonstrating the lines of incision for exposure of the pyriform sinus, the hypopharynx, and the mouth of the esophagus.

wall of mucous membrane between the pharynx and larynx (Trotter), that is to say, the mucous membrane that is behind the cricoid cartilage.

In cases of benign tumors or of other benign formations (laryngoceles or cysts) one of the following procedures can be done: the outer and top part of the corresponding wing of the thyroid is extirpated (we have done this in some mucoceles and perilaryngeal cysts); or that part of the cartilage that must be separated is pushed backwards and outwards and after the mucocele or cyst has been removed, the cartilage is replaced. This can generally be done without opening the laryngeal mucosa (cyst) or closing the existing opening (laryngocele). I have done this several times.

Operations for benign or malignant tumors of the external wall of the pyriform sinus do not always require tracheotomy. One of my cases of malignant tumor of the external wall of the pyriform sinus was operated on successfully without a tracheotomy being done.

2. *Vestibular tumors* (tumors of the lower part of the epiglottis and of the ventricular bands—partial horizontal laryngectomy). Examination of larynges extirpated for cancer reveals three important facts.

First, cancer of the lower part of the epiglottis or cancer of the ventricular bands does not exist alone; the cancer that attacks one of these zones invades the other side also.

Second, cancer originating in this part of the larynx invades the preepiglottic space at an early stage. (Justo M. Alonso, "Anotaciones sobre el espacio pre-epiglótico," *Anales de Otorinolaringología del Uruguay*, 1945, page 81). In its development, it then extends upward and frequently invades the lymphatic nodes of the neck situated between the diagastric muscle and the omohyoid muscle, and exceptionally the higher ones.

Third, invasion of the larynx by cancer further down is almost always limited to its anterior aspect by the anterior commissure, by the insertion of the tendon of the vocal cords and further back, by the bottom of the ventricle, which is rarely invaded.

As cancer in this location gives few early symptoms, generally it is seen by the physician at an advanced stage of

development. Laryngoscopy can deceive us regarding the extent of its invasion. The roentgenogram will help us to determine whether invasion of the preepiglottic space has taken place, but invasion occurs in this location long before it throws abnormal shadows on the roentgenogram. Generally the tomograph allows us to see that the anterior commissure is not invaded while showing shadows in the ventricles, even though these are unaffected. The voice is normal except for decreased volume.

Cancer of this region has been treated generally by deep roentgen therapy or by total laryngectomy. The deep roentgen therapy is successful in early cancer but not when the lesion has ulcerated or when there are palpable metastatic nodes.

Surgeons have found that simple total laryngectomy is often not enough. Many of us for a long time have practiced extirpation of the larynx together with the preepiglottic space. This operation has lately been described by Ricardo Bisi (Argentina) under the name of "Laringectomía con extirpación del espacio pre-epiglótico."

I consider that if the vocal cords and the subglottic region of the larynx are intact, their extirpation is not a guarantee that a cancer situated above there will not recur. It is much more important to remove the upward and forward infiltration of the cancer into the base of the tongue and the preepiglottic space, at the same time removing the diseased regional nodes or the ones that are suspected of being involved.

Numerous surgeons, operating on isolated cases, and with an inadequate background of clinical experience, have tried to cure these extrinsic laryngeal cancers by transverse pharyngotomy or laryngofissure. This latter procedure for the treatment of vestibular cancer is contraindicated because it presents three serious drawbacks: first, the incision generally passes through tissues invaded by the cancer; second, as it modifies fundamentally the function of deglutition and does not leave a pharyngostomy, there is a danger of inspiration pneumonia caused by attempts at swallowing; third, I consider that when the vocal cords are healthy it is unnecessary and even injurious to open the glottis. Besides, in a laryngofissure, the regional lymph nodes are not removed at the primary operation but after an interval by a secondary dissection of the neck.

An interesting essay has been written by Huet (Societé de Laryngology des Hospitaux de Paris, 20:VI, 1938) in which, in an early case of ventricular cancer, he has advised extirpation of the preepiglottic space and the tumor by means of an incision that includes the central part of the hyoid bone and the upper anterior part of the thyroid cartilage.

I have tried to develop a technic that would allow me to extirpate the cancer completely and yet preserve the airway and the voice. This technic allows a proper exploration of the malignancy, its complete removal together with the regional lymph nodes, and the formation of a pharyngostomy which will prevent swallowed material reaching the lungs.

### SURGICAL TECHNIC

I commence by making a flap similar to the one used in a cancer of the lower part of the pharynx (fig. 7). We do this on the side which we consider to be most affected. The internal jugular nodes are extirpated, cutting the posterior belly of the digastric muscle and the stylohyoid muscle so as to allow removal of the uppermost lymph nodes. To explore the lymph nodes of the opposite side of the neck one can proceed in one of the following ways: first, the superior line of the incision may be continued across

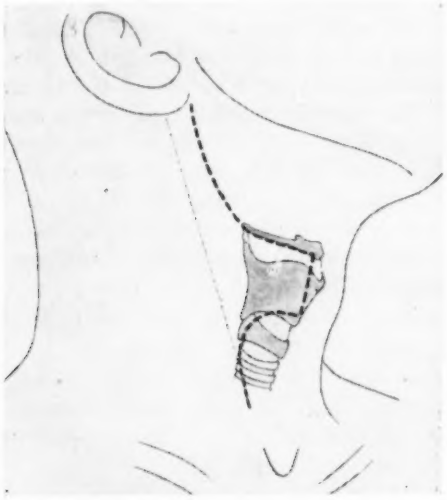


FIG. 7.—Outline of the incision used in the technic for conservative removal of extrinsic cancer of the larynx.

to the opposite side of the neck making an incision similar to that on the first side. This line of incision would go from one external auditory meatus to the other passing by way of the hyoid bone. At the level of the junction of the incision with the anterior border of the sternomastoid muscle, the superficial layers of the deep cervical fascia are cut, allowing exploration of the regional lymph nodes. If necessary the skin incision is continued downward following the anterior edge of the sternomastoid muscle exposing the carotid sheath and the great vessels of the neck. To expose the upper part of the carotid sheath, the posterior belly of the digastric and the stylohyoid muscles are divided. Second, a separate incision exposing the carotid sheath can be made on the least affected side, along the anterior edge of the sternomastoid muscle. When the regional nodes have been dissected out, the way to the pharynx is prepared by finding and cutting the facial vein at the edge of the submaxillary gland. The infrahyoid muscles on the affected side are next removed by dividing them superiorly next to the hyoid and inferiorly at their insertion into the wing of the thyroid, or at the level of the thyroid when they are inserted lower down. The corresponding half of the hyoid bone is freed and cut through just beyond the midline. If invasion of the tissues at the base of the tongue is suspected, the lingual artery is found and tied above the great horn of the hyoid. Then the superior laryngeal vessels are ligated and the superior laryngeal nerves are cut. The upper third of the wing of the thyroid is dissected free, together with the most central and upper part of the opposite wing. The superior insertions of the constrictor muscles of the pharynx are cut at the level of the edge of the wing of the thyroid cartilage. The greater horn of the thyroid cartilage is dissected free and cut on the same side. These procedures give sufficient exposure to reach the pharynx posterior and lateral to the epiglottis. An incision from 2 to 3 centimeters long is then made in the pharynx in that area. This allows us to examine visually and palpate the tumor and the surrounding area. The incision is enlarged horizontally and anteriorly (at the level of the hyoid bone or further up, as the case may be) and then continued downwards to allow palpation and visualization of the tumor, the pharynx and the larynx. If the top part of the tumor does not pass beyond the epiglottis the upper incision can follow the line occupied by the half hyoid extracted. If it invades the base of the tongue, the incision should be made as high as possible. If this is done the epiglottis will be plainly visible; a thick thread of silk passed through it will serve to move it about. Laterally the pyriform sinus and the aryepiglottic fold are exposed.

Let us suppose that the aryepiglottic fold is half invaded in a front to back direction. We then pull on the silk that

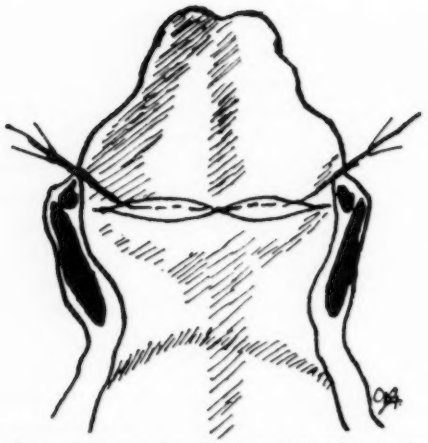


FIG. 8—Demonstrating the line of incision passing below the laryngeal ventricles.

goes through the epiglottis so that the top part of the larynx makes a bending movement allowing us better visualization of the tumor.

The aryepiglottic fold is cut between the most posterior extension of the invaded zone and the arytenoid cartilage. The incision is continued towards the inner part of the larynx, passing below the ventricle and is then continued forward so as to cut just above the tendon at the anterior commissure (fig. 8). On the outside of the larynx the incision passes along the junction of the upper third with the middle third of the corresponding wing of the thyroid cartilage from behind forward and slightly downward so as to reach the depression in the midline a centimeter below the anterior angle of the thyroid cartilage (fig. 9).

FIG. 9—Demonstrating the line of incision made along the line of junction of the upper and middle third of the thyroid cartilage.

When the tumor is very big and the internal part of the larynx can not be seen well, the incision in the thyroid cartilage may be made first, thus allowing mobilization of the affected part of the larynx (fig. 10).

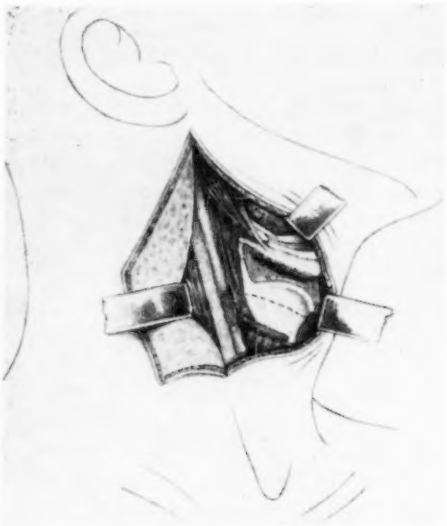


FIG. 10—Demonstrating the exposure of the larynx secured after removal of a portion of the thyroid alae.

To extirpate the affected area on the opposite side of the larynx, the infrahyoid muscles are lifted with a separator and the involved area excised along with the upper and medial part of the wing of the thyroid cartilage. This enables us to avoid the superior nerve of the larynx on that side. The cut should always be made one centimeter or more

beyond the apparent limits of the infiltrated area spreading out from the tumor. If the invasion reaches the floor of the ventricle, the mucous membrane that covers the superior surface of the vocal cord together with the upper muscular fibers must be excised.

If there still remains a small area in which infiltration by the cancer is suspected, it should be electrocoagulated. Small bleeding vessels are also electrocoagulated. Electrocoagulation must not be too extensive as this leads to the formation of edema of the larynx. Following removal of the involved areas, the mucous membrane is sutured to the skin around the opening; superiorly, the skin is sutured to the mucosa of the pharynx. The lateral flap is pushed down into the wound bending it a little downwards so that the upper line of the incision that followed the hyoid bone will take a vertical direction in its medial half. Inferiorly the suturing is done with much care. The mucosa of the pyriform sinus is grasped with two forceps, the one holding the mucosa of the internal wall and the other one holding the mucosa of the external wall. The mucosa of the internal wall of the pyriform sinus is sutured to the mucosa of the superior face of the vocal cord with a fine suture. The mucosa of the external wall of the pyriform sinus is sutured to the skin edge of the vertical portion of the skin flap.

At the bottom, the suturing is completed by sewing skin to skin, leaving only a small hole for the exit of a rubber tube placed deep to the skin flap for drainage. This tube is left in place for two days. Feeding is given by nasal tubes. A plain dressing is used together with a tube to the larynx that is connected to some mechanism for continued aspiration. The postoperative stage is generally without complications. In two months' time the stoma has become much smaller and may be closed in two layers. Frequently, swallowing may be difficult for months, even though the superior nerve of the larynx has been kept intact on one side. These difficulties generally disappear with regeneration of the nerve. On the other hand, as the voice is good the patient bears these difficulties with optimism. After the tracheal tube is removed the patient recovers nasal breathing.

Our patient is now able to carry out any active function. The danger of a recurrence is the same as would be the case following total laryngectomy.

This operation tends to preserve the life of the patient, without destroying the normal functions of the larynx.

## DISCUSSION

GORDON B. NEW, M.D., Rochester, Minn.: Dr. Alonso has presented his method of surgical removal of two types of malignancy of the hypopharynx and larynx. His paper terms this, "conservative surgery," although it seems to me that the term, "adequate and rational surgery," would be the better because his technic calls for more extensive excision than that which we usually term, "conservative." It is, obviously, quite necessary surgery. The first method he mentions is for those lesions of the pyriform fossa or pyriform sinus, in which he does his method of partial vertical laryngectomy. The second type of operation which he recommends is for vestibular growths for which he does a partial horizontal laryngectomy.

Both of these methods seem to me to be very ingenious and, quoting Dr. Alonso, "the operation tends to save the life of the patient without loss of the normal functions of the larynx." He uses surgical diathermy or electrocoagulation when necessary, which I feel is very important.

He mentions that "the excision should be made one centimeter or more from the limit of the zone which is suspected of being malignant." I should like to ask him if he uses fresh, frozen sections, making an immediate microscopic diagnosis in order to determine the activity of the growth and just about where the margin is, because we know the more active growths are always far beyond their apparent limits.

He does not stress the necessity of opening the trachea in these cases for a short time, nor the question of intranasal feeding with a Rehfuss tube.

Dr. Alonso's work, of course, is very well known in this country, as we have followed his writings for years. I feel that I have been honored to be asked to discuss his paper and I wish to congratulate him on his very original methods of treatment.

VALMORE LATRAVERSE, M.D., Montreal, Can.: Do the voice and respiratory function of the patient remain good following operation?

F. E. LEJEUNE, M.D., New Orleans, La.: I should like to ask Dr. Alonso to elucidate a little more on the indications for this particular type of operation. It is not clear in my mind whether it is possible for him to do this conservative surgery which he advocates if there is a fixation of the arytenoid on one side or the other.

JUSTO M. ALONSO, M.D., Montevideo, Uruguay: Dr. New asks whether I do rapid biopsy of the margins of the excised specimen. I have followed this practice whenever possible.

Dr. Latraverse asks if the voice and respiratory function remain good following operation. After a month or two, they are good. In some cases of cancer of the pyriform sinus, it is not necessary to do a tracheotomy.

Dr. LeJeune asks whether, in the event that there is fixation of the arytenoid, I do a partial horizontal laryngectomy. When the arytenoid is involved, I do a total laryngectomy.

# OSTEOPLASTIC OPERATION ON THE LARGE FRONTAL SINUS IN CHRONIC SUPPURATIVE SINUSITIS: END RESULTS

RAUL BERGARA, M.D.
BUENOS AIRES, ARGENTINA
BY INVITATION

IN spite of valuable therapeutic drugs such as sulfonamides and penicillin, surgery still plays an important part in the treatment of septic infections of the paranasal air cavities. Owing to various reasons, an acute inflammation may enter the chronic stage, thus rendering chemotherapy sometimes quite ineffective and surgery the sole means of treatment. This explains why we specialists are constantly endeavoring to discover better surgical procedures in the hope of attaining definite final results, which are sometimes difficult to achieve.

This is particularly true with reference to large frontal sinuses, partially divided by septa and containing retention cells (stasigenic elements of van der Hoeven Leonhard). This is the type of sinuses in which suppuration frequently becomes chronic, with the added complication that not only sulfonamides and penicillin are of no avail, but even surgery often fails in achieving permanent relief.

It is with this in view that we wish to present before this meeting the results obtained in cases of chronic fronto-ethmoidal-maxillary suppuration with a personal osteoplastic technic successfully carried out by us since 1934.

Our procedure has been the subject of several papers, though slight modifications have been introduced as our experience has become greater.

Presented before the Academy Section on Otolaryngology and the Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 18, 1946, Chicago, Ill.

An operation that attempts to cure a chronic frontal suppuration by the external route should obey the following principles:

a) Give a good exposure of and access to the sinus cavity, its diverticuli and the frontal ostium. This is made possible only through wide resections of the outer wall so as to enable the surgeon to appreciate the depth of the cavity and discover any fronto-orbital extensions or deep ethmoidal sinuses.

b) Provide an ample and *permanent* drainage of the cavity.

The technic we use consists of a large flap of skin and bone in which is included, as far as possible, most of the outer wall of the sinus. Such a flap offers a wide opening into the cavity and its extensions, and also gives afterwards an excellent cosmetic result.

An ample and persistent drainage—as we shall see later on—is provided by the removal of the frontal process of the maxilla, the lacrimal bone and the anterior part of the ethmoid bone.

A chronic frontal sinusitis is nearly always due to an extension of a maxillary sinusitis through the ethmoid and therefore the infections of both air cavities become one: the fronto-ethmoidal-maxillary sinusitis.

Some years ago we used to operate first on the antrum (Caldwell-Luc technic), this being followed a fortnight later by the osteoplastic operation on the frontal bone. Today we perform both operations simultaneously: a trans-

maxillary ethmoidectomy (Ermiro de Lima's procedure) first, followed immediately by the frontal opening. As the latter is a very strenuous operation, each of the operations should be performed by a different surgeon.

Leaving aside the preoperative stage, we shall now describe some points of our procedure.

The eyebrow is shaved and the corresponding nostril packed with a strip of gauze soaked in a solution of cocaine-adrenalin. Regional and hypodermic local anesthesia is secured with a 2 per cent solution of novocain and adrenalin. The incision of the skin is made above the eyebrow and extended towards its outer end (length proportionate to the size of the sinus) while the nasal portion will descend from the head of the eyebrow, down the middle of the corresponding side of the nose as far as 1 cm. below the inner canthus. This incision will only include skin and soft tissues. The upper flap is then undermined a little beyond the actual diameter of the diseased sinus.

*Search for the frontomaxillary suture.* A 1½ cm. incision of the periosteum is made in the inner canthus; this membrane is elevated only to an extent that will allow the exposure of the frontomaxillary and frontolacrimal sutures. Then it is prolonged upwards to the site selected for trephining the frontal sinus, which, in our opinion, should be made 3 mm. above the frontomaxillary suture.

*Trephining of the flap.* An exploratory opening with a round 3 mm. electric burr is made in the outer aspect of the frontal sinus at the previously mentioned site. As soon as the burr is set into action, continuous warm saline irrigation and suction must be started. The inner aspect of the sinus can now be examined by means of a fine curved-end probe passed through the opening which will also act as a guide for determining the most convenient spot near its boundaries for the second perforation. The intervening periosteum between the exploratory opening and the place chosen for the second is incised and elevated sufficiently to allow for the new perforation and postoperative suture. Smaller burrs may be used for this and further perforations. A series of perforations at 5 to 6 mm. distance from each other are made as near as possible to the upper margin of the sinus, until the upper orbital margin is reached, and then these are joined by a 1 mm. burr lineal trephining, which must be made in bevel-edge fashion, so as to allow for firm apposition when the flap is returned and thus avoid its falling back into the sinus. The burr makes a clear-cut section and consequently there is no morcellation of the diploe as when a rongeur is used. Many cases of postoperative osteomyelitis are due, in our opinion, to displaced or nonviable chips of partially adherent, but insufficiently vascularized diploe.

*Shaping of the osteoperiosteal flap.* The lower or orbital portion of the frontal bone is the thinnest and therefore most fragile, so that by inserting a couple of retractors through the bone incision it is quite easy to fracture the orbital wall. This provides us with an osteoperiosteal flap hinged on its lower side, which renders an ample view of the frontal sinus when brought down. The air cavity is cleansed by profuse saline irrigation and suction of exudates. The use of gauze swabs—so useful at other times—must be abandoned here as the pressure necessary for detersion would cause the spread of the infection through the veins that enter the endocranium. *No gauze* therefore—plenty of saline irrigation and suction.

*The mucosa of the sinus.* A frontal sinusitis that requires an external surgical means of access for its treatment

implies that the mucosa is diseased and therefore should be totally excised, even though it may appear to be sound: it must be borne in mind that it can often conceal diverticuli and foci of osteitis. Besides, we advocate the reduction of a large proportion of the cavity, through fibrous obliteration, and this demands the total elimination of its lining. No vigorous scraping but a careful and total removal of all intracavum tissues by means of small adequately curved curets. Any ethmoidal air cell which may protrude into the orbital wall must be excised, the mucosa removed and its cavity made to form one with the frontal sinus.

*The nasolacrimal groove.* Having secured a total clearance of the sinus and its diverticuli, the next step is to obtain an easy and *permanent* drainage. One of two methods is selected, depending on whether the patient has previously undergone a simple Caldwell-Luc operation for his maxillary sinusitis or whether transantral ethmoidectomy for ethmoidal-maxillary sinusitis was the treatment applied (Ermiro de Lima's technic).

In the *first case* a permanent and sufficient drainage may be obtained by simply widening the nasolacrimal groove through excision of the anterior ethmoidal sinuses, part of the frontal process of the maxilla and the lacrimal bone. This is the method we followed in our first cases after introducing a soft metal probe into the nasal cavity to act as support and protection to the cribriform plate of the ethmoid bone. With large round burrs and proceeding in an anteroposterior direction, we then resected all the structures situated in front and a little towards the outer side of the probe. This was started in the highest part of the frontal process of the maxilla, continued with the lacrimal bone until the level of its crest was reached (great care was taken not to injure the lacrimal sac, the lacrimal portion of the orbicularis and Horner's muscle) and ending with the "agger nasi" cells and the ethmoidal sinuses surrounding the lacrimal groove.

When a partial resection of the lacrimal bone is carried out, there is no danger of opening the orbital cavity and thus risking the possibility of an infection; the reason for this is due to the presence of the orbital septum which is attached to the edge of the orbit, at the level of the crest of the lacrimal bone and just behind the above-stated muscular insertions. The pulley of the obliquus superior always remains untouched.

It is obvious that these resections provide a wide access to the rest of the ethmoid and to the sphenoid bone.

In the *second case* a careful clearance of the antrum will enable the surgeon to see exactly what results were achieved by the ethmoidectomy, and will suggest the best course to follow. Were it considered that the ethmoidectomy has provided sufficient drainage, then it will only be necessary to complete what ethmoidectomy was unable to do, that is, to resect whatever cells surround the groove. But if the ethmoidectomy was found to provide insufficient drainage, the resection of cells will have to be pursued further.

*Rubber tube drainage.* When a satisfactory outlet to the sinus has been secured, a rubber tube is placed in situ, large enough to fit in exactly. This is left for a period of thirty days and is usually well tolerated by the patient.

*Replacement of the flap and suture.* The operation is now practically finished. The flap is firmly secured against the frontal bone, there being no fear of its falling into the sinus owing to the bevelled edges. Several stitches in the periosteum will hold it in place. The eyebrow is closed by subcuticular skin suture and interrupted stitches close the

rest of the incision. A capillary drain is left in the inner angle of the wound, which can be removed the second day. A sterile gauze pack and bandage will be placed as dressing.

This technic has given us excellent results in the thirty-two cases treated, not only by providing a complete cure of the sinusitis, but also from the cosmetic point of view. However, on two occasions we met with slight complications. In the first case, in a rather restless patient, the capillary drain slipped out and she therefore developed a hematoma; the patient did not complain about the wound, so that her temperature was attributed to an infection of her sinus, but on removing the dressing on the fifth day, we found an infected hematoma that had to be opened in the inner end of the incision and forthwith healed completely without damage to the flap.

In the second case and nine months after the operation, the patient developed a subperiosteal abscess localized at the outer end of the eyebrow. Having opened the abscess, instrumental examinations revealed a denuded bony surface near the site that had been trephined. The patient was re-operated on and the whole of the bony part of the flap resected; the sinus was found to be completely obliterated by fibrous tissue, with the exception of the most outward corner where a small diverticulum had not been properly freed of mucosa and therefore became the source of the abscess.

Of the 32 cases referred to in this lecture, 28 were operated for fronto-ethmoidal-maxillary chronic suppuration and four on account of intractable frontal neuralgia.

## DISCUSSION

HENRY L. WILLIAMS, M.D., Rochester, Minn.: I read Dr. Bergara's paper with a great deal of interest and attention. I think that all of us have found that in sinus surgery the problem of cure of chronic frontal sinusitis is one of the major difficulties that we have to deal with. It has been obvious for a long time that the obliterating type of operation is the one which has been the most successful in securing permanent relief for the patient. Unfortunately this often does produce what might be called a poor cosmetic result and for this reason many patients object to this type of surgery.

Dr. Bergara has solved this problem and has devised an obliterating type of operation with good cosmetic results. I believe that his operation will find a very definite place in surgery of the frontal sinus. I think it is a very definite improvement in the technic of such surgery.

He rightfully emphasizes the importance of complete removal of the mucosa of the sinus, pointing out that if any of the mucosa is left, cyst formation will ensue with subsequent difficulties which would be expected, of course, in an obliterating type of procedure.

He also well emphasizes the importance of getting rid of the orbital extensions of the ethmoid cells in this procedure. The only question on which I have any tendency to differ with Dr. Bergara is in regard to the surgery of the nasofrontal opening. I have felt for a long time that occlusive scars in the nasofrontal opening have come from the orbital periosteum. I also feel that one ought to retain the lacrimal process, the lamina papyracea, and the frontal process of the superior maxilla, and produce an infundibuliform opening from the frontal sinus into the nose. This should prevent the collapse of soft tissue into the nasofrontal opening and development of scar tissue either of which might occlude the nasofrontal opening. Of course, Dr. Bergara advises leaving a rubber tube in situ for thirty days; after this length of time I believe he hopes that hyperplastic tissue will have occluded the frontal sinus, so that eventually the intranasal opening will be entirely unnecessary except for the inner aspect of the sinus. I feel, however that leaving the rubber tube in for only thirty days would possibly not be long enough. I have had difficulty with leaving rubber in situ in frontal nasal openings because of its irritating tendency, and in many cases have used the old Ingalls tube.

Dr. New has recently delighted us with the invention of an acrylic obturator which I believe will be a distinct advantage over the rubber tube in operations of this sort. I believe he is to present his invention later on in this session. I understand that it can be

left in for six months resulting in epithelization of the tract and a very good postoperative result.

The combination of this operation with Dr. de Lima's transmaxillary ethmoidectomy can be advantageous, because by this procedure transmaxillary ethmoidectomy will preserve the bony structures of the nasofrontal opening and in this way prevent the collapse of the soft tissues into this area which often causes trouble.

JUAN M. TATO, M.D., Buenos Aires, Argentina: (*Translated and summarized*) Dr. Tato feels that the operation that Dr. Bergara suggests for large frontal sinuses, in cases of chronic suppurative frontal sinusitis, has given better results, because the opening is made in the superior part of the anterior wall of the sinus cavity. With the other type of operation circulation was completely cut off.

Dr. Tato mentions five cases on which he has operated, using this technic, with no adverse after-effects.

He thinks that better results will be obtained with an acrylic obturator, which can be kept in situ for a longer time, without any fear of resulting irritation, in order to avoid any failure.

Like Dr. Williams and Dr. Bergara, he thinks that this operation should be done in conjunction with that of de Lima (transmaxillary ethmoidectomy) in cases of pansinusitis.

RAUL BERGARA, M.D., Buenos Aires, Argentina: Many thanks, Dr. Williams and Dr. Tato, for discussing my paper.

The closure of the drainage in the frontal sinusitis is really a serious problem. We leave the rubber tube in for the time that the progress of the healing requires it.

# ADVANTAGES IN NOT TAMPONING AFTER SUBMUCOUS RESECTION: PERSONAL TECHNIC

ROGER A. LANZA CASTELLI, M.D.
CORDOBA, ARGENTINA
BY INVITATION

*The following is an English translation of the summary of the paper presented in Spanish by Dr. Lanza Castelli.—Ed.*

THE following are the necessary requisites for operating on the nasal septum without any need for tampons. Before operating, clinical and hematological conditions must be normal. In order to avoid hemorrhage on the fifth or eighth day after operating, clotting must be surgically favorable, the ideal time being five minutes and never more than eight. Bleeding time should be one and a half minutes but never more than two. It is also necessary that the mucosa be free from severe inflammatory conditions. Should the inflammatory condition be of a chronic nature, previous desensibilization with a 5 per cent nitrate of silver solution is suggested. This may be done by swabbing, and helps to prepare the mucosa for slight trauma, avoiding secretory and vasomotor reactions after surgery. Should there have been a general or local infection, fifteen or thirty days must be allowed to lapse before operating.

As to the surgical technic: for infiltration use adrenalin and privine; for surface anesthesia be cautious with the amount of adrenalin used. The patient should be on his back, slightly lateralized towards the surgeon. Such a position is very convenient for neat and detailed surgery.

When operating, keep in mind:

1. *Cauterize along line of incision,* from the roof downward and slightly extending along the floor of the nose.
2. *Perichondrium and periosteum on both sides are to be kept intact.*
3. Special care must be taken not to tear or traumatize the mucosa.
4. Just enough perichondrium and periosteum must be taken up with the mucosa to lead down to the bone deformation.
5. All bleeding spots must be cauterized till bleeding stops. For insignificant or surface bleeding, swab with privine. Pay particular attention to the point where the palatal artery emerges, low down at the front. If found bleeding, as it is apt to do, cauterize till it stops. Should there be no bleeding, leave a tampon wet with privine in place for complete hemostasia.
6. Wash out the sac continually with liquid paraffin and pump during the operation to obtain complete cleansing. After operating repeat the process to secure adherence of both mucosae and permit capillary phenomenon to take place.
7. A small hole is surgically punctured into the mucosa. This favors adherence of mucosa eliminating serous secretion by capillarity, also acting as drainage in case of complicating hematoma.

Presented before the Academy Section on Otolaryngology and the Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 18, 1946, Chicago, Ill.

8. Both sides are gently pressed against each other introducing Killian's long speculum alternately into the nasal fossae till both mucosae adhere perfectly. Care must be taken to powder with sulfamide (sodium salt) before doing so. Thus capillary phenomenon becomes established.
9. Suture of incision is not necessary but there is no objection to doing so if desired.

Postoperative care is very simple. Just remember that the patient must not be touched if trauma is to be avoided. After the seventh or eighth day, swab with privine, look out for synechias, and so on. Hematoma is one of the complications to expect, but give it time and the artificial outlet made in the sac will take charge of draining it. The only postoperative trouble that the patient may complain about is a breathing difficulty that disappears after twenty or thirty days when everything becomes normal. Trying to eliminate or explore the clot by surgical means only makes the complication worse.

By eliminating the tampons, I've done away with postoperative hemorrhage. Out of more than 600 cases operated on and carefully followed up, only two such accidents have been observed. One was due to inadequate compression while effecting puncture in the mucosa. This was happily detained without any need for tampons. The other hemorrhage occurred after an associated intervention that had nothing to do with the septum in itself.

The advantages are great, whether with regard to postoperative complications or postoperative care, as not any of the patients complained of trouble in their ears, paranasal sinuses, pharynges, or of headaches, fever, etc.

As long as tampons are not used and all the instructions for preoperative, surgical and postoperative measures are well carried out, this operation becomes simple for the surgeon and is not troublesome to the patient. It furthermore helps in the associated surgical procedures advocated by Professors J. M. Tato and Albert Haedo in their work put before the second South American Congress of Oto - Rhino - Laryngology and put into practice for some time back in our country.

## DISCUSSION

WALTER REINKING, M.D., Mexico City, Mexico: I want to congratulate Dr. Lanza Castelli for the work which he has presented to us, which was very well developed, logically written, and presented in easy and understandable language.

In my opinion there are four principal points which have to be emphasized. First of all is the hemostasis which he carries out very carefully. He does not let any bleeding persist inside the cavity after the bone has been removed. He uses cautery which I think is a good thing when you don't use it too hard, because I believe that a cautery also irritates the mucous membrane.

The second thing is that he depends on capillarity to keep the mucous membrane together. I think the use which he makes of this phenomenon is very good indeed.

The third part and a very important part of his surgical procedure is drainage of the pouch which remains. He does it in the center part of the septum whereas we use it in the lower part.

He presents the results of 600 cases and he has had very few hematomas. Almost every one of us who operates has hematomas. We usually make an incision in the lower part in order to get into the chamber.

The thing by which I am struck is the very few complications which Dr. Lanza Castelli has had. After submucous resections we all see tonsillitis, sometimes infected sinuses, and sometimes pharyngitis, and he apparently has no complications of that kind. He has had hematomas in 9 cases out of the 600, which I think is very good, and I want to congratulate him on that result.

We usually put the gauze inside the nose. We have our reason for that. In Mexico, as a general rule, we operate on our patients in our offices and send them home. I don't know whether Dr. Lanza Castelli's patients

were operated on in his office or in a hospital, but if you operate in your office and you send the patient home, you want to be sure that their noses are plugged up, so they won't call you out between two and three o'clock in the morning. We usually use a special gauze which I got some years ago from Germany, and I am using a Swiss gauze now which is very soft. It is impregnated with grease and it has a special kind of talcum which is antiseptic.

We make a little cushion. We put some cotton inside the gauze and we use four of these tampons, two in each side. They are very soft and as the nose swells and the mucous membrane becomes a little thicker, they adapt themselves very well to the contours of the nose. The compression of these tampons should be very slight so there is no leakage through the mucous membrane. Thus a flapping septum will not be formed, avoiding in this way the possibility of adhesions or synechias.

We also think that if we compress the mucous membranes a little, the veins are compressed. If there is no dressing in the nose, the nose will bleed not only from the arterial blood vessels but also from the venous blood vessels, which I consider very important. Also, we use a tampon for the coaptation of the flaps. Sometimes we have a very large deflection of the septum, and you know perfectly well that in such cases after you have taken out all the bones and all the cartilage the septum still is deflected.

As far as trouble after the operation is concerned, we have very little in our patients. We used these tampons that are very soft, not too tightly pressed in. We give the patient medicine to relieve the pain, aspirin or whatever is used in the various countries. We find that sometimes patients come back the next day to have the tampons taken out and they say that they have not taken any medicine to kill the pain. In order to insure that the patient has a good night we give him nembutal.

Removing the gauze the next day is very easy because it does not stick. Patients are not frightened at having the tampons taken out. We never leave the tampons in more than twenty-four hours. In order to minimize discomfort for the patient, we usually operate in the afternoon and take out the tampons early the next morning. Some surgeons leave the tampons in for forty-eight hours, but I think this is hard on the patient.

As far as the drainage of the hematoma is concerned, which may occur with any surgeon, we do not believe in waiting. We go at it. We open up the incision. We take a little piece out so that the opening is wide. We see the patient every day, and go in with a little probe to get the liquid out. If the liquid does not come out, infection will occur and you will have pus under the mucous membrane. I think it is not hematoma and lack of nutrition of the cartilage which makes a saddle nose or a depressed nose. It is the infection that develops if the hematoma is not drained.

We have never had a suppurating nose that I can remember, unless our patients go to some other doctor and complain about us, but usually they complain to the surgeon if their noses are deformed.

We also use a tampon because in many cases we do a retouching of the lower turbinates, which are vascular organs and may bleed.

Our postoperative treatment differs a little from that of Dr. Lanza Castelli. We see our patient every day and clean the nose out with a spray. We have the patient blow his nose and he feels a lot better if the nose is clean. If the nose is clean there will not be much reaction of the mucous membrane. If you leave the nose alone or the patient does not come back to see you and you see him two or three years later, his nose will be full of crusts. Fibrous membranes are forming. You have to clean the nose in our opinion.

Another point on which we differ with Dr. Lanza Castelli is that we sometimes operate when there is an infection present. Sometimes we have a sinusitis, and in a very narrow nose, the nose has to be ventilated first in order to treat the sinus.

The hemorrhages which come after two or three or five days come just the same whether you put a piece of gauze inside the nose or not.

I want to congratulate Dr. Lanza Castelli again on his nice work.

H. C. Ballenger, M.D., Winnetka, Ill.: In his effort to minimize bleeding during and after the submucous resection, I am very happy to have Dr. Lanza Castelli emphasize a normal clotting and bleeding time and most important in my opinion, a complete absence of any acute inflammation of the nose or throat.

Most cases will do well without packing or with a minimum of packing, thereby avoiding the secondary discomfort and irritation of the pack. In those cases in which a pack is used, it is seldom necessary for a period longer

than twenty-four or forty-eight hours. In most instances, a unilateral pack on the side of the greater convexity is all that is needed. I frequently use nothing in the form of a pack other than a shaped piece of flat dental compound. This is easily inserted and removed and seems to have little or no irritating qualities. If additional compression is needed, vaseline gauze strips may be inserted externally to the wax compound on one or both sides, as indicated.

In my experience, the essential needs for nasal packing after a submucous resection are two: first, to control excessive or persistent bleeding. This rarely occurs, but is possible in a few cases. If present, adequate packing on the affected side or sides should be done until the bleeding has been controlled. The normal serosanguineous discharge which follows this operation, with or without packing, should not be considered abnormal. The second indication would be to retain as much of the deviated septal flaps as possible in the midline, in those few instances where a complete removal of the deviated portion of the septum is not possible or feasible. In this event a unilateral packing on the convex side for one or two days may be all that is necessary. If complete removal of all deviated portions of the septum is done, the remaining mucoperichondrial and mucoperiosteal flaps tend to seek the midline position. In most instances, I use a unilateral pack rather than the bilateral type. A third possible indication for nasal packing might be as a preventive of a hematoma between the flaps. However, this mishap is rare and can be prevented in most instances, as pointed out by Dr. Castelli, by puncturing a hole or holes in the inferior portion of the septal sac or by extending the primary incision for a short distance through the inferior portion of the mucoperichondrial or mucoperiosteal flaps parallel to the floor of the nose.

Dr. Lanza Castelli is to be commended for reporting his long list of successful operations for deviated septum, without the use of a nasal pack.

RICARDO BOMPET, M.D., Buenos Aires, Argentina: (*Translated*) I congratulate Dr. Lanza Castelli, and I would like to add my few words to the discussion of his paper. Like Dr. Castelli, I do not believe that the omission of tamponage after submucous resection, using the subperiosteal and subperichondrial technic, predisposes to hemorrhage. I have measured the amount of blood lost by patients operated on both with and without subsequent tamponage, and have proved that there is no appreciable difference.

If care is taken to assure that the mucosa is well approximated and is not everted, capillarity is sufficient to maintain it in situ and at the end of eight hours, cicatrization will be seen to have begun.

For more than ten years I have followed this principle of not tamponing, in my private practice and also in hospital work; on the service of otolaryngology of the Hospital Ramos Mejía, in Buenos Aires, of which Dr. Antonio Zambrini is chief, and where approximately 600 submucous resections are done per year, this technic has been the usual one since 1934.

LYLE S. POWELL M.D., Lawrence, Kan.: I am sorry that I haven't had a chance to read the English translation of the doctor's paper, but I should like to corroborate in general his theory and his practice of not using tampons after submucous resections. For the past fifteen years I have not packed a single nose following a submucous resection. In thinking the thing over prior to that time, it seemed to me that it was contrary to some of the surgical principles that we are taught to observe, and I proceeded to try it with very good results.

There are one or two things that are necessary though, when a submucous resection is done. When the operation is done, it is necessary to cut loose the flap along the floor of the nose. If that is done, you won't have any hematomas. You won't have any collection of tissue fluid. Furthermore, it seems to me that the more simply we can do any surgical procedure, the better it is, and the more we can give nature a chance to do things in her way the better it is. As far as I am concerned, nature knows a great deal better than we do how to pack a nose, and after a submucous resection, without any packing, the nose swells shut perfectly and stays that way for about twelve hours. You couldn't ask for any better packing than nature provides. The drainage is down. Gravity carries any fluid or any blood downward, and there are no complications due to retention between the flaps.

Another thing that I should like to point out is—and I am not sure that the doctor does not in his paper—that Dr. Roberts some years ago gave me an idea that has been very valuable to me. After doing some of these submucous resections, the patients complained

of flapping septums. They will complain that when lying on one side, the side of the nose that is down stops up. Upon turning over, the other side stops up. If you will take a pointed hammer angle forceps when you finish the operation and go along the floor of the nose and make a deliberate perforation about half way back on the floor of the nose, you will get enough scar tissue reaction to hold that scar tissue in place and you won't have any flapping. Furthermore, you can do that in patients who have already had submucous resections and give them relief from this flapping that disturbs them so much.

WENCESLAO MEDRANO, M.D., Ciudad Trujillo, Dominican Republic: (*Translated and summarized*) Dr. Medrano disagrees with Dr. Lanza Castelli's technic because he feels that without tamponing, there is danger of more postoperative hemorrhage. He also feels that if tamponing is done, the mucosa heals better and more rapidly and there is less risk of postoperative complications.

ROGER A. LANZA CASTELLI, M.D., Cordoba, Argentina: (*Translated and summarized*) Dr. Lanza Castelli first thanked all those who constructively criticized his paper, stating that he believed criticism to be necessary to any scientific work.

In reply to Dr. Reinking, Dr. Lanza Castelli said that, in his opinion, the main reason for continuing tamponage after submucous resection is fear; the fear of postoperative complication. Actually this fear is unfounded, for the postoperative complications *without* tamponage are reduced.

He thanked Dr. Bompet for pointing out that a series of Dr. Zambrini's cases over a period of time show the same results as his (Dr. Lanza Castelli's) own.

Replying to Dr. Medrano's criticism, Dr. Lanza Castelli reiterated that postoperative complications are reduced rather than increased, and that there is no more danger of severe postoperative hemorrhage without tamponage than with it.

As far as Dr. Powell's technic of operation is concerned, Dr. Lanza Castelli stated that he did not use this procedure because he felt that there was definitely more danger of postoperative complication, and recovery was much slower.

# CANCER OF THE LARYNX: LARYNGECTOMY WITH SYSTEMATIC EXTIRPATION OF THE CONNECTIVE TISSUE AND CERVICAL LYMPH NODES AS A ROUTINE PROCEDURE (LARYNGEAL CANCER AND LYMPHATIC METASTASIS)

JAIME DEL SEL, M.D., *and* ALEJANDRO AGRA, M.D.
BUENOS AIRES, ARGENTINA
BY INVITATION

*The following is an English translation of the summary of the paper presented in Spanish by Drs. Del Sel and Agra.—Ed.*

IN the present paper, the authors undertake to demonstrate that laryngectomy should be accompanied by systematic removal of the lymph nodes.

They state some considerations with reference to the orientation of general surgery of cancer, which have not been applied in our specialty. By means of the technic described herein, it is believed that the rate of cure in cases of cancer of the larynx will be improved.

The authors comment on the characteristic features of cancer of the larynx in Argentina which differ in histology and location from those of the northern countries.

They add some remarks about lymph node cancer and go on to conclude that these should be taken into consideration in conservative surgical procedures in surgery of cancer of the larynx.

They reserve conservative surgery for cancers in which the vocal cord is freely movable, or those of the commissure. Except in these cases, they prefer the radical operation as the only surgical means by which one is able to increase the possibilities of a cure.

They make some comments on pre- and postoperative care without forgetting their preference for local anesthesia, and describe the technic of the removal of the regional lymph nodes which is carried out simultaneously with laryngectomy. This technic was devised by the Argentine physician, Dr. Sylvestre Begnis, and the authors consider it the method best adapted to modern requirements, since the extirpation includes the larynx, the lymph nodes and above all the collecting channels, thus satisfying one of the most fundamental principles of cancer surgery.

By means of chemotherapy and meals rich in calories, the postoperative period has been made much more favorable.

The authors refer next to the opinions of renowned authors which do not coincide with their views. Though all accept the danger of the encroachment of the lesions on the lymph nodes, not all of them believe in intervention when the nodes are affected.

The authors add that the most favorable cancer for surgical treatment is that which develops on a removable organ, the efferent lymphatic channels of which form a series of trunks close to each other united in ganglionary groups that can be removed in a single block. The larynx offers exceptional conditions for realizing this.

They do not approve of the classification of lymph nodes into "palpable" and "nonpalpable" as a criterion for operative procedure. Along side of a simply

Presented before the Academy Section on Otolaryngology and the Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology at the Fifty-First Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 18, 1946, Chicago, Ill.

inflammatory hypertrophied node there may be a neoplastic microadenopathy. They mention cases, in order to corroborate this point, in which the examination of the excised block from the neck made it possible to prove this.

The palpation of lymph nodes is not always an easy matter because of individual differences in the development of the neck.

The laryngeal growths in Argentina are supraglottic in a portion of 1:9, an area which is a potential source of metastasis, and because of the relative absence of symptoms, patients consult the specialist when the lesion has grown beyond the larynx. The surgeon must anticipate the tumor, and increase the possibilities of cure.

The curative and prophylactic removal of lymph nodes, ideal theoretically and possible technically, which the authors propose as a systematic procedure, is, in their opinion, the operation which will be used in the future.

## DISCUSSION

GORDON B. NEW, M.D., Rochester, Minn.: Dr. Del Sel's beautiful presentation has recommended bilateral prophylactic dissection of the upper cervical fascia and lymph glands in all cases where a laryngectomy is done. He advises the removal of the glands on both sides of the larynx and upper cervical region in one specimen or one piece so that the whole group is taken out at that time. He feels that a dissection of the lymphatics in the treatment of extensive cancer of the larynx is just as essential as the removal of the axillary glands in cancer of the breast or removal of the submental submaxillary gland as a prophylactic measure in cancer of the lip.

Of course, this is a very logical method to follow. So far, however, he has not been able to make a comparison as to the results in five-year cures in a group of cases of his where glands have not been removed and a similar group in which this particular dissection has been done, so that he has not been able to make a comparison to show the additional value as to the end result for the patient by this gland dissection.

In our cases of low grade tumors with gland involvement a block dissection is done, but if a gland is involved not only the upper cervical glands are taken out but also a complete block dissection to the clavicle along with the removal of the sternomastoid muscle is done. That is, we feel that at the time of operation a fresh frozen section on microscopic examination will tell us whether the gland is involved microscopically. If this gland is involved microscopically, it does little good to take out that particular group of glands alone. One must take out the complete group of glands in the neck in order to block the trouble.

Dr. Del Sel's presentation is outstanding and I wish to congratulate him on his excellent piece of work.

PLINIO BARRETTO, M.D., Sao Paulo, Brazil: I will do my best to summarize the few most important points of my discussion in English. First of all, with the modern methods that we have to decrease the mortality of laryngectomy or of any other surgery of the neck, especially using penicillin or other means at our disposal, we have increased very greatly the good results of any operative method, but we should always remember that we are taking a life in our hands. Although we think that our surgery is almost perfect we should always keep in mind that we should try not only to save the patient's life but if possible preserve function.

I should like also to point out the value of conservative methods because not always does the patient agree to permit such a large intervention. It is easier to persuade the patient to submit to conservative surgical intervention than to the removal of his larynx.

I mentioned surgery and radiotherapy, because I always say that we should use every method to treat the patient and that we should not try to select our cases or to say that one method is better than another, but, if possible, try to work with both methods.

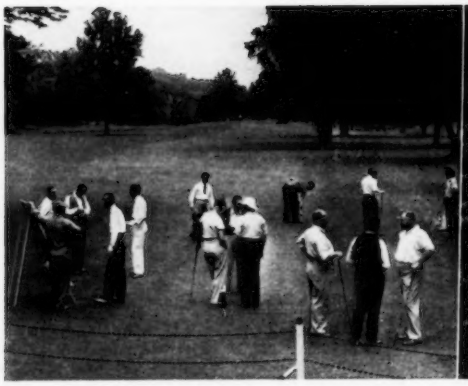
I called Dr. Del Sel's attention to the fact that he is dealing with a very interesting group of patients because the great majority have cancer of the upper part of the larynx. In Brazil we don't have such a great number of cases of the epiglottic lesions as compared with the subglottic. The lymph nodes that become involved first are in the area of this operation. So there is no doubt that his operation can remove at least in a few cases glands that we are not able to detect on clinical examination and that are invaded.

JAIME DEL SEL, M.D., Buenos Aires, Argentina: (*Translated and summarized*) Dr. Del Sel feels that cancer of the larynx should be treated in the same way as the cancers of other organs, for example, cancer of the uterus. Therefore the nodes should be removed at the same time that the laryngectomy is done, because microscopic biopsy shows that even in cases where the nodes are not yet palpable, they may be involved.

With reference to the duration of the operation and the loss of voice which results, these are in fact, not so important as they may seem, because, in the first place, there is no shock following this operation and, in the second place, although it may be difficult to convince the patient of the advantages of a laryngectomy, because he loses his voice, all attempts should be made to convince him, because the operation is being done for his own good.

# 1947 POST CONVENTION VACATION

*Aerial view of French Lick Springs Hotel*



*First tee on lower golf course*

*A view of formal gardens*

## A WORD ABOUT THE DIVERSION CLUB

For a great many years the Academy sponsored golf tournaments as well as other types of sports, but it became so occupied with educational activities that it seemed best to discontinue them. Then the late Dr. Wherry conceived the idea of having a Diversion Club of the Academy, and that it have a short vacation following the convention each fall. This was done for a few years but during the war it had to be discontinued. It was decided that the activities should be revived this year and French Lick Springs Hotel was selected as the most logical place to have the outing.

French Lick Springs Hotel is located in the Cumberland foothills of southern Indiana. The grounds comprise some 2,000 rolling, wooded acres, in which there are two very challenging golf courses, an excellent archery range, riding stables (which boast of some fine Kentucky thoroughbreds), a very fine formal garden and many beautiful winding paths. There are excellent tennis courts, badminton courts two swimming pools, a four-lane bowling alley and billiard and ping pong tables.

Meals are regularly served in the main dining room; the Monon Room is open to those who are unable to have their meals at regular hours, and the Demon's Den is quite attractive to those who wish to sharpen their appetites and meet their friends.

Music by an excellent orchestra is provided for dancing in the evening.

The atmosphere is very informal.

The guest rooms are attractively furnished and arranged in a variety of combinations.

As stated in the last issue of the Transactions, accommodations can be had for only one hundred persons in this year's post convention vacation trip. The cost of the entire trip will be $82.50 per person. This will include round trip railroad fare (with Pullman reservations), French Lick Springs Hotel room (American Plan), prizes and golf fees.

The train to French Lick will leave the Dearborn Street Station over the Monon Railroad at 10:05 p.m. (c.s.t.) Friday, October 17, arriving in French Lick the next morning at 7:45, and will leave French Lick at 9:30 p.m. Monday, October 20, arriving in Chicago at 7:40 a.m. Tuesday.

### POST CONVENTION VACATION

C. H. McCaskey, M.D., Chairman, Diversion Club
608 GUARANTY BLDG., INDIANAPOLIS 4, IND.

1. I wish to make reservations for the post Academy convention vacation at French Lick Springs for_____persons.
2. I (we) shall travel by train_____.
3. I (we) shall travel by automobile_____.

NAME_____

ADDRESS_____

President 1947

BALTIMORE, MARYLAND

TRANSACTIONS
American Academy of Ophthalmology and Otolaryngology
*Official Publication of the American Academy of Ophthalmology and Otolaryngology*
Published under the direction of the Editorial Board
WILLIAM L. BENEDICT, Rochester, Minnesota, *Editor*
W. HOWARD MORRISON, Omaha, Nebraska, *Associate Editor*
HENRY L. WILLIAMS, Rochester, Minnesota, *Associate Editor*
Printed by Douglas Printing Company, 109 North 18th Street, Omaha 2, Nebraska

All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, the American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

# Editorial

## Home Study Courses

*The Seventh Year.* The seventh year of the Home Study Courses has just been completed. The total registration was 544 — 353 in Ophthalmology and 191 in Otolaryngology. This is a 35 per cent increase over the total enrollment for the previous year.

A new lesson on Pharmacology was added to the 1946-1947 Course in Otolaryngology, and a new lesson on External Diseases has been planned for the Course in Ophthalmology and will be included in 1947-1948.

The following quotations are from a few of the expressions of appreciation that have been received from many who have taken the Courses the past year:

> "I have greatly enjoyed and profited from the Home Study Course during the past nine months. It has been a great stimulus to organized reading in the basic sciences."
>
> "This letter accompanies the last of my examination papers in the 1946-1947 Home Study Course. I feel that I want to say 'Thank you' to those of you who have made this course possible. Especially do I appreciate the work of those doctors who have written valuable comments while grading the papers. The course has been most useful to me and I shall continue to employ it as an outline of study."
>
> "I would like to take this opportunity to thank you and your associates for the trouble you have taken in putting on this fine course. It has been and will continue to be of the greatest assistance to me."
>
> "Please allow me to express my thanks to you and everyone else concerned in the Home Study Course. I personally feel that the course has been of a very great benefit. In no other way could one have been able to cover the basic sciences in a systematic manner."
>
> "May I express the interest I have had in this course, and state that the reading assignments and questions have been interesting, stimulating and very valuable."
>
> "I wish to thank those who corrected my papers. The criticisms

have stimulated me to read more thoroughly the prescribed assignments."

*Exhibit at the Academy Meeting.* There will be an exhibit of the Home Study Courses work at the Academy meeting in October. It will include brief historical data on the accomplishments under the program since its inception in 1940, a complete roster of the present Faculties, copies of the 1946-1947 lessons, and samples of some of the unusually good work of several of the physicians who have taken the Courses in the past year.

*Scope of the Program.* The Home Study Courses program has become one of the largest functions of the Academy. Most of the physicians who are in training for the specialties of Ophthalmology and Otolaryngology in the United States, and many in Canada, now register for this basic study.

**NOTICE**

The Atlases of Ophthalmic and Otolaryngic Pathology may be obtained by ordering directly from the Registry Office, Army Institute of Pathology, Washington 25, D. C. The price for Atlases is $5.00 each, and checks should be made payable to the National Academy of Sciences.

## FINANCIAL REPORT

### Annual Audit for Calendar Year — 1946

May 20, 1947

The American Academy of
Ophthalmology and Otolaryngology
Rochester, Minnesota

Gentlemen:

As requested, we have examined the accounts and records of The American Academy of Ophthalmology and Otolaryngology, an unincorporated association, for the calendar year ended December 31, 1946, and have prepared therefrom the accompanying balance sheet, statement of operations, analysis of net worth, analysis of decrease in market value of securities and schedules showing securities owned on December 31, 1946.

We have also reviewed the system of internal control and accounting procedures, and without making a detailed audit of cash receipts and disbursements and other transactions, have examined or tested accounting records and other supporting evidence, by methods and to the extent we deemed appropriate.

Cash on deposit at the various banks was verified by communicating with the depositories, and the petty cash fund was verified by actual count.

Membership dues receivable, amounting to $1,995.00, represents delinquent dues for 1946 and prior years, which were verified from the records only.

Investments in stocks and bonds, which are described in Schedule No. 1, had a market value of $195,471.50 on December 31, 1946 as compared to $150,052.70 on December 31, 1945.

Securities in the Research Fund, which are held for safe-keeping by the Trust Department of the First National Bank, Minneapolis, Minnesota, were verified by communicating with the custodian of the securities. U. S. Savings Bonds in the Wherry Memorial Fund are held by Dr. W. L. Benedict, Executive Secretary-Treasurer and were inspected by us. Interest earned on bonds since the dates on which the last payments became due, has not been accrued. An analysis of decrease in market value during the year is shown in Exhibit "D".

Furniture, fixtures and equipment amounting to $4,428.26 represents equipment acquired by the Academy over a period of years. Equipment acquired in years prior to September 1, 1942, which had been charged to operating expenses as purchased, was inventoried

and appraised at a market value of $1,420.28 on January 1, 1944, and set up on the books for that amount. Equipment purchased since September 1, 1942, amounting to $3,007.98 was for the most part verified through an inspection of supporting vouchers, and of the latter amount, $728.37 was purchased during the current year. Depreciation has been deducted and charged to operations at a rate that appears to be adequate.

Unexpired insurance premiums totaling $259.13 were computed by us. Prepaid postage deposit of $45.13 represents the balance of cash advanced to the U. S. Post Office at Omaha, Nebr., to be applied as postage on subsequent mailing of the "Transactions" by the publisher at Omaha.

Current liabilities of $1,572.41 consist of accrued expenses totaling $772.81 for unpaid December printing and other items, federal withholding taxes totaling $549.60, and membership dues paid in advance totaling $250.00. The committee of the Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology has made expenditures, the amount of which is not known, and which is not included in the above total of accrued expenses.

An analysis of the net worth of the Academy is shown in Exhibit "C", which reflects an increase in net worth from $159,712.74 a year ago to $204,099.80 on December 31, 1946.

Upon examining the insurance policies in force, we found Owners' and Tenants' Public Liability insurance in the amount of $5,000.00/$10,000.00 coverage, and Workmen's Compensation subject to statutory amounts. The Executive Secretary-Treasurer and Chairman of the Finance Committee are bonded for $25,000.00 each, while the principal office employees are bonded for $1,000.00 each. Furniture, fixtures and equipment are insured for $4,000.00 fire and extended coverage.

In our opinion, the accompanying balance sheet and related statement of operations and net worth present fairly the position of the Association at December 31, 1946, and the results of its operations for the year, in conformity with generally accepted accounting principles applied on a basis consistent with that of the preceding year.

Respectfully submitted,

SHANNON & BYERS
Certified Public Accountants
By Edw. A. Wobschall, C.P.A.

*EXHIBIT "A"*

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

*Balance Sheet as at December 31, 1946*

ASSETS

| | | |
|---|---|---|
| *Current Assets:* | | |
| Cash, First National Bank, Rochester, Minn. | $2,504.01 | |
| Cash, First National Bank, Minneapolis, Minn. | 84.46 | |
| Cash, Bank of Montreal, Toronto, Ont. | 1,767.52 | |
| Cash, petty cash fund | 25.00 | |
| Membership dues receivable, (delinquent) | 1,995.00 | |
| Other receivables | 231.11 | |
| Total current assets | | $ 6,607.10 |
| *Investments:* (Schedule No. 1) | | |
| Stocks and bonds (at market value) | | 195,471.50 |
| *Fixed Assets:* | | |
| Furniture, fixtures and equipment | 4,428.26 | |
| Less reserve for depreciation | 1,138.91 | 3,289.35 |
| *Deferred Charges:* | | |
| Prepaid insurance | 259.13 | |
| Prepaid postage deposit (Transactions) | 45.13 | 304.26 |
| Total Assets | | $205,672.21 |

LIABILITIES AND NET WORTH

| | | |
|---|---|---|
| *Current Liabilities:* | | |
| Accrued expenses | $ 772.81 | |
| Accrued withholding taxes | 549.60 | |
| Deferred income, membership dues | 250.00 | |
| Total current liabilities | | $ 1,572.41 |
| *Net Worth* (Exhibit "C") | | 204,099.80 |
| Total Liabilities and Net Worth | | $205,672.21 |

*EXHIBIT "B"*

AMERICAN ACADEMY OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY

*Statement of Operations*
*Year Ended December 31, 1946*

| | | |
|---|---|---|
| *Revenues:* | | |
| Membership dues | | $ 20,203.00 |
| Membership applications | | 4,794.00 |
| Atlases | | 3,796.13 |
| Certificates | | 1,184.00 |
| Transactions | | 20,363.05 |
| Home Study Courses | | 5,476.81 |
| Convention | | 57,122.60 |
| Contribution to Research Fund | | 112.75 |
| Income from securities: | | |
| Interest, Research Fund | $ 2,185.75 | |
| Dividends, Research Fund | 1,602.50 | |
| Profit on securities sold, Research Fund | 1,104.01 | |
| | 4,892.26 | |
| Interest, Wherry Memorial Fund | 115.00 | 5,007.26 |
| Sundry receipts | | 127.03 |
| Total Revenues | | $118,186.63 |
| *Operating Expenses:* | | |
| General administrative | 18,609.18 | |
| Transactions | 14,071.15 | |
| Home Study Courses | 4,555.26 | |
| Research | 159.00 | |
| Conventions and meetings | 34,890.87 | |
| Total Operating Expenses | | 72,285.46 |
| *Net Income* | | $ 45,901.17 |

*EXHIBIT "C"*

AMERICAN ACADEMY OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY

*Analysis of Net Worth Account*
*For Year Ended December 31, 1946*

| | | |
|---|---|---|
| *Net Worth, December 31, 1945* | | $159,712.74 |
| *Additions:* | | |
| Net income, year ended December 31, 1946 | 45,901.17 | |
| | | 205,613.91 |
| *Deductions* | | |
| Decrease in market value of securities during year (Exhibit "D") | 1,514.11 | |
| *Net Worth, December 31, 1946* | | $204,099.80 |

*EXHIBIT "D"*

*Analysis of Decrease in Market Value of Securities*

| | *Cost* | *Market* | *Decrease* |
|---|---|---|---|
| 12/31/45 | $154,523.62 | $150,052.70 | $4,470.92 |
| 12/31/46 (Schedule 1) | 201,456.53 | 195,471.50 | 5,985.03 |
| Difference | $ 46,932.91 | $ 45,418.80 | $1,514.11 |

SCHEDULE No 1

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

## Securities Owned, December 31, 1946

RESEARCH FUND

| BONDS | RATE | MATURITY | PAR VALUE | COST | MARKET VALUE |
|---|---|---|---|---|---|
| Brooklyn Edison Co., Inc. Cons. | 3¼ | 5/15/66 | $ 3,000.00 | $ 3,045.00 | $ 3,112.50 |
| Citizens Building Co. of Cleveland—1st Leasehold Sinking Fund | 6 | 1/1/46 | 5,000.00 | 5,000.00 (1) | |
| Dominion of Canada Conv. Loan Ser. R8 | 4½ | 11/1/58 | 5,000.00 | 4,950.00 (2) | 5,075.00 |
| Dominion of Canada, 8th Victory Loan Ser. P5 | 3 | 10/1/63 | 2,500.00 | 2,500.00 (2) | 2,462.50 |
| Van Sweringen Co., Certificate of Indebtedness, Series 1925 | 6 | 12/31/48 | 6,000.00 | 6,031.33 | 1,800.00 |
| U.S.A. Treasury 1967-72 | 2½ | 2/15/72 | 10,000.00 | 10,000.00 | 10,287.50 |
| U.S.A. Savings, Series "D" | ..... | 4/1/49 | 10,000.00 | 7,500.00 | 9,000.00 |
| U.S.A. Savings, Series "D" | ..... | 3/1/50 | 10,000.00 | 7,500.00 | 8,700.00 |
| U.S.A. Savings, Series "F" | ..... | 12/1/55 | 13,500.00 | 9,990.00 | 10,260.00 |
| U.S.A. Savings, Series "G" | 2½ | 8/1/53 | 10,000.00 | 10,000.00 | 10,000.00 |
| U.S.A. Savings, Series "G" | 2½ | 6/1/55 | 4,000.00 | 4,000.00 | 4,000.00 |
| U.S.A. Savings, Series "G" | 2½ | 3/1/56 | 6,000.00 | 6,000.00 | 6,000.00 |
| U.S.A. Savings, Series "G" | 2½ | 5/1/56 | 8,000.00 | 8,000.00 | 8,000.00 |
| U.S.A. Savings, Series "G" | 2½ | 7/1/56 | 2,000.00 | 2,000.00 | 2,000.00 |
| U.S.A. Savings, Series "G" | 2½ | 11/1/56 | 25,000.00 | 25,000.00 | 25,000.00 |
| U.S.A. Savings, Series "G" | 2½ | 1/1/57 | 2,000.00 | 2,000.00 | 2,000.00 |
| U.S.A. Savings, Series "G" | 2½ | 7/1/58 | 30,000.00 | 30,000.00 | 30,000.00 |
| U.S.A. Savings, Series "G" | 2½ | 12/1/58 | 25,000.00 | 25,000.00 | 25,000.00 |
| Total Bonds | | | | $168,516.33 | $162,697.50 |

| STOCKS | KIND | PAR VALUE | SHARES | | |
|---|---|---|---|---|---|
| Citizens Building Co., Cleveland | Shares | No Par | 50 | None (1) | ..... |
| Commonwealth Edison Co. | Common | $25.00 | 100 | $ 2,619.00 | $ 3,375.00 |
| E. I. duPont de Nemours & Co. | Common | 20.00 | 15 | 2,347.31 | 2,861.25 |
| Louisville Gas & Electric Co. | 5% Pfd. | 25.00 | 200 | 5,450.00 | 6,050.00 |
| Massachusetts Investors Trust | Shares | 1.00 | 617 | 17,923.89 | 15,887.75 |
| Ophthalmic Publishing Co. | Capital | 20.00 | 5 | None (1) | ..... |
| Total Stocks | | | | $ 28,340.20 | $ 28,174.00 |
| Total Research Fund | | | | $196,856.53 (3) | $190,871.50 |

WHERRY MEMORIAL FUND

| | RATE | MATURITY | PAR VALUE | | |
|---|---|---|---|---|---|
| U. S. A. Savings, Series "G" | 2½ | 1955 | $4,600.00 | $ 4,600.00 (4) | $ 4,600.00 |
| Total Investments | | | | $201,456.53 | $195,471.50 |

(1) No market available
(2) U. S. Dollars
(3) Held for safe-keeping by Trust Dept. of First National Bank, Minneapolis, Minnesota.
(4) Held by Dr. W. L. Benedict, Executive Secretary-Treasurer, Rochester, Minnesota.

# OCCUPATIONAL OPHTHALMOLOGY

## INTRODUCTORY STATEMENT

*Romaine's paper is an excellent statement, couched in simple language, explaining the relationship of extraocular muscle imbalances to industrial eye problems. There is not enough appreciation of the elements of binocular vision and accommodation as they apply to industrial vision. For example, forced plus correction in the face of a convergence insufficiency, or forced minus correction in the face of a convergence excess can follow an improper adjustment of working lenses.*

*The "closed session" held last October during the meeting of the National Safety Council was based solidly on a previous extensive survey. The all-day get-together of every type of individual who enters the picture of industrial eye problems, plano eye protection, prescription lenses, safety glasses, filter lens, etc., brought rich results. It would pay anyone specifically interested in the discussion of eye protection practices to send for a copy or reprint of the original article on "Industrial Eye Protection Practice" which appeared in the July issue of National Safety News. An immense amount of work was put into this session and there are plans for a continuance of the work with another get-together scheduled for October, 1948. The paper included here was written by John Roche, the spark plug of the project.*

*Fleischer's contribution on welding radiation deals with first hand observations. He had a tremendous experience with industrial medical problems on the east coast where he was responsible for all phases of this work in the Maritime Commission shipyards. It is a very important study for those of us working in industry.*

*The Joint Committee has often raised the question in its discussions as to the advisability of some type of simple test added to the visual tests in industry which could detect at least the gross field defects found in either brain tumor or glaucoma cases. While it is true from the ophthalmologist's point of view that there is no good substitute even for a very rough test for the regulation form field, nevertheless, there is probably something to be gained by developing and introducing in industry a test that would catch the gross field defects. It was with this thought in mind that McClure's work at Purdue was conducted and presented in this issue.*

*Now that the war is over, it is important that industry and their staff of medical consultants remember the excellent record of the blind or partially blind in war production. A summary of this splendid step forward in industrial placement is included to bring the Academy membership up to date on this particular phase of industrial eye work. The diversity of employment is significant of the progress made. The old stereotyped basket-weaving concept of blind usefulness is now obsolete.*

HEDWIG S. KUHN, M.D.
*Secretary, Joint Committee*
*Occupational Ophthalmology*

# THE EYE MUSCLES IN OCCUPATIONAL OPHTHALMOLOGY

HUNTER ROMAINE, M.D.
NEW YORK, N. Y.

OF the many problems facing the ophthalmologist in the handling of large industrial groups, those conditions concerned with the ocular muscle play a very large part. The position of the ophthalmologist is usually that of a consultant to the medical group in charge of the particular industry involved. His primary job is to organize and set up satisfactory visual tests and to help the permanent staff in the eye division in their analyses of the visual tests. It is also his job to straighten out any unusual problems that present themselves.

The muscles with which we are most concerned can be divided into the internal and external groups. The internal muscles with which we shall have to deal are the dilator and sphincter muscles of the iris and the ciliary muscles which govern the amount of light entering the eye and the necessary changes in accommodation that must take place during work. The external muscles concerned are the extraocular muscles which govern the ability of the patient to see a singular binocular object. They govern the ability of the eyes to fuse, and the degree to which fusion may be obtained. In conjunction with the latter muscles, we must also consider the elevator of the lid—the levator palpebrae—because of its close relation to the superior rectus muscle and its importance in carrying out normal opening of the eye.

There are many jobs in industry which require certain essentials of the eyes—those jobs in which there is a moving object requiring a constant readjustment of accommodation (that is, the eye must adjust from one point to another in rapid manner). In addition the objects seen sometimes must be viewed with concentration to pick up fine details, while in other cases the concentration is not as important. Then again there are those jobs which require rapid movement of the eyes from object to object, such as inspection jobs in which the operator must examine the articles on a moving belt. The articles are constantly moving towards the examiner and at the same time require movement of the fixing point of the eyes. On some occasions these objects may be on alternating belts coming from either side. It is obvious that jobs of this nature require the utmost in accommodation and fusional capacity. Then again there are those jobs which require exceedingly accurate depth perception as in the manipulation of a crane or similar apparatus; also dangerous jobs which require the adjustment of articles in pressure instruments.

Let us now consider how the ocular muscles enter this picture. One of the major purposes of the pupillary muscles—the dilator and sphincter—is the control of the amount of light entering the eye. It is obvious that looking incessantly into a very bright light will make an excessive demand on these muscles. Looking alternately in quick succession from a bright to a relatively dark surface is a particularly severe condition. The operators of computing and tabulating machines experience this strain looking rapidly from white paper to black machine. Methods of adjusting the type of illumination and the direction of this illumination will help to eliminate some of the distressing symptoms brought about by improper lighting. If it is found necessary for the worker to be under such conditions, the use of lenses having absorbent qualities will help to reduce the fatigue factor

Presented at the New York University College of Medicine as a course on Occupational Aspects of Ophthalmology, March, 1947, New York, N. Y.

induced by the conditions. Rearrangements in lighting apparatus and reduction of glare by special paint may help. In addition the personnel department may aid the medical staff by assigning workers who have minimal disturbances from light to jobs in which requirements of this sort are necessary.

The ciliary muscle comes into play in the constant use of the eyes for exacting close work, particularly in individuals with hyperopia and/or astigmatism. Uncorrected conditions of this sort lead to rapid tiring and inefficiency on the part of the worker. The nearsighted eye though fully corrected for distance is frequently unsatisfactorily corrected for close work. Quite often the fact is overlooked that a worker who has held a job for a long period of time will become presbyopic and need correction for near where exact working distances must be carefully taken into consideration.

The function of the extraocular muscles is that of maintenance of singular binocular vision. Interference with this function can be due to poor muscle tone which may be associated with poor muscular tone in the entire body; individual muscular weakness of a congenital nature or as the result of an acquired paralysis; and lastly, rapid fatigue factors resulting from poor general physical condition. We must consider the results of an inability of the worker to maintain fusion satisfactorily. One of the first complaints noted is that of blurring of the vision which frequently is the result of loss of fusion. The first stage of double vision is one of blur rather than an actual diplopia. If this condition continues the worker will be troubled with intermittent double vision and will shortly reach a point where he is unable to continue with his job. In addition to this a vertical imbalance existing in the eyes may be markedly increased to the point where the worker may no longer carry out his job as a result of a constant vertical rotating motion of the eyes necessary in doing his work. The existence of a ptosis may reduce the ability of the employee to carry out certain jobs where his requirements include looking upwards, whereas a job which required only looking into a lower field would be within his qualifications and one in which he could well justify his employment.

Too frequently in industry it is noted that actual working lenses which are not properly adjusted may produce muscular weaknesses which will result in unsatisfactory work. This again is too frequently blamed on the inefficiency of the worker rather than on the unsatisfactory adjustment of the lenses. As an example of this I mention the increased amount of convergence insufficiency resulting from a fully corrected hyperopia in the face of a small degree of convergence insufficiency. Likewise in the myope full correction of the minus in the face of a convergence excess will only exaggerate the symptom and bring the worker to the point where he is unable to carry out his job to satisfaction. In addition, lenses which are so ground that the adjustment for distance or near is not correct may result in prismatic effects producing convergence insufficiency or convergence excess. In addition, if the lenses are not properly adjusted there may result a definite difference in size of the objects seen, particularly in those patients suffering from anisometropia.

A question of the approach to the satisfactory handling of these problems lines up as follows: The medical division should have a consulting ophthalmologist, one who knows the factors necessary to outline a satisfactory ocular division of the plant. His tests should be so designed that they detect such conditions as rapid fatigue of accommodation, marked light sensitivity, tendencies

towards convergence excess or insufficiency, divergence excess or insufficiency, unsatisfactory near point, poor vertical, improper function of the levator muscle. In addition there should be a satisfactory optical department in which lenses can be ground and adjusted, where there exists full knowledge of design and use of special bifocal and trifocal lenses, equipment to handle hook-over adjustments, an understanding of the necessity for decentering lenses and the methods of doing this to the benefit of the worker.

The medical division finds it advisable to work hand-in-hand with the personnel department in the adjustment of employees so that the most suitable work within the ocular limits of the workers' capabilities are put to adequate use. It is of extreme importance that the employees should be examined ocularly from time to time to ascertain whether their ocular condition is satisfactory to maintain a satisfactory output of work. It is unfair to give a worker unsatisfactory efficiency rating because he is ocularly in no position to carry out the assignment given to him.

The medical division of any large industry with the aid of a consulting ophthalmologist should be able to establish a division which will not only benefit the worker markedly but tremendously increase the efficiency of the plant.

## OPHTHALMIC EFFECTS OF WELDING RADIATIONS

WALTER E. FLEISCHER, M.D.
BALTIMORE, MD.

IN the fall of 1942, the U. S. Navy and the U. S. Maritime Commission appointed a group of industrial health and safety consultants to investigate and make recommendations concerning health and safety in all Maritime Commission and Navy contract shipyards. Later on the War Shipping Administration yards were added. Our particular area covers about 100 shipyards in the Great Lakes, Gulf and Atlantic Coast Regions. Briefly, we were charged with the elimination of industrial health hazards and the prevention of occupational disease.

With the onset of the war welding emerged from comparative obscurity to perhaps the most important single process in the construction of ships. In our yards, at the peak of production, there were over 200,000 welders and probably at least as many again in Navy yards and other war industries. At the Bethlehem-Fairfield yard in Baltimore a contract for 384 Liberty ships was recently finished. This type of ship is easy to build, can be launched and transport cargo in a few months, and contains far less welding than a destroyer, submarine, cruiser or battleship. Some enterprising individual at the yard calculated that all the welding done on these Liberty ships would reach seven times between Baltimore and San Francisco. And that is only one yard.

Because of its numerous advantages, welding will play an important role in many industries in the future. It, therefore, behooves us to incorporate into the training of industrial physicians as much knowledge as we possess on the occupational hazards involved. There are various hazards in welding in addition to those resulting in eye injuries—electric shock, inhalation of fumes from lead, cadmium, zinc, chromium and fluorine, explosions from welding in tanks or on pipes containing inflammable gases, etc. However, eye injuries are far more numerous than any other type.

Medical Director, Rustless Iron and Steel Division, American Rolling Mill Company.
Presented at the New York University College of Medicine as a course on Occupational Aspects of Ophthalmology, March, 1947, New York, N. Y.

*Incidence of Flash Injuries*

Available statistics on the incidence of welding flash are not accurate for unfortunately many shipyard dispensaries have treated and recorded as welding flashes, some cases in which the worker was only momentarily exposed to a welding arc and was afraid that he would develop actinic ophthalmia. As I will explain later, the term welding flash is a misnomer, for a specific amount of exposure is required to develop this syndrome. However, the term is well known to industrial physicians and workers, and is widely used today, regardless of its inaccuracy.

At the onset of the tremendous increase in shipbuilding in 1942, at least 40 per cent of all new injuries in shipyards were eye injuries, and a very large part of these were caused by exposure to the ultraviolet radiations from welding. Despite the fact that the usual case of flash involves no lost time nor permanent disability, there were in 1943 about 3,000 lost-time cases due to welding flash; and in 1944, there were 1,300. According to the Bureau of Labor Standards of the U. S. Department of Labor, a lost-time case is one in which the employee is unable to work at his next regular shift following injury or is permanently disabled. Records covering all injuries indicate that for each lost-time case, there were 50 non-lost-time cases that required medical treatment. Since so few flash cases involve lost-time, I believe that this ratio is much higher for welding flashes—at least 100 to 1. This indicates that in shipyards alone, we had, as late as 1944, when eye protection programs were well established, over 100,000 cases of welding flash.

*Welding Process*

Today, it is essential for the industrial physician to understand the fundamentals involved in any industrial process if he is to treat intelligently patients exposed to its hazards. In general, all modern welding processes are designed for rapid production of intense heat within a small area. In electric arc welding, the intense heat generated by the passage of electric current across the arc melts both the welding rod and the edges of the plates to be welded, thereby filling the gap with a fused layer of metal derived from both the welding electrode and the plates. Coated electrodes have gradually displaced the uncoated, as it is possible with the coated rods consistently to make better and stronger welds at much higher speeds. The so-called shielded arc is produced by these special coatings baked on the welding electrodes. Some of this coating burns to form a protecting gas shield around the molten metal as the metal passes across the arc, while the mineral portion of the coating forms a protective slag on top of the molten metal. Thus, there is less oxidation of metal, less formation of nitrides; the metal cools at the proper rate, and the work is done faster than in the case with bare electrodes.

There are various types of welding, each using specific materials and designed for a particular purpose. Electric arc welding is by far the most common; gas welding second in importance. The great bulk of shipyard electric arc welding on black iron and mild steel is done over a range of 25 to 50 volts and 100 to 300 amperes. The higher voltages and amperages are used with large electrodes or where deep penetration of the base metal is desired, while the lower voltages and amperages are used for small electrodes and mild penetration of the base metal. The remainder of this discussion pertains solely to electric arc welding since it is the type most generally used.

RADIATION FROM ELECTRIC ARC WELDING

The biologic response to radiation emitted by the welding arc follows the laws of light in that the intensity of the eye reaction varies directly with the length of exposure and inversely with the square of the distance. Within a 24 hour period, the effects of repeated exposures are cumulative.

*Ultraviolet Radiation*

The limits of the band of welding radiations and of the cut-off for various tissues of the eye vary somewhat in the literature on this subject, for considerable work has been done on it. However, the differences are insignificant. (A chart of the spectrum was shown to which was added the cut off wave lengths in the ultraviolet region of the crystalline lens at 336 mμ, of the cornea at 300 mμ and of the vitreous humor at 230 mμ. That is, these are the shortest wave lengths that the tissues will transmit.—Ed.)

The toxic ultraviolet rays are those below 300 mμ and unfortunately the welding arc is rich in the short as well as the long ultraviolet rays.

Sufficient exposure to short ultraviolet rays produces the following picture of welding flash: The pathologic changes are confined to the superficial structures of the eye. Examination with a slit lamp reveals some superficial devitalization and minute edematous blebs on and in the epithelium of the cornea which is quickly repaired in 12 to 18 hours after onset of symptoms. The cornea, iris and lens absorb the ultraviolet rays and prevent further penetration, thereby protecting the deeper structures of the eye. We have found no evidence that this temporary injury to the corneal epithelium either permits ready bacterial invasion or contributes to permanent injury of the cornea even though frequently repeated. There is deep-seated pain in the eyes which leads to blurring. Accommodation is temporarily affected, and the attempt to accommodate may lead to eye pain and headache. In some cases apparently the abnormal sensitivity to ultraviolet radiation may persist indefinitely.

Kinsey, Cogan and Drinker measured the amount of ultraviolet necessary to produce a welding flash or actinic ophthalmia, as it is more correctly called. They found that 150 foot candle minutes was the threshold and that 200 foot candle minutes of exposure consistently produced a flash. To illustrate—if a worker watches an arc for 1.5 minutes from such a distance that the intensity of the arc exceeds 100 foot candles, then there is danger of getting a flash. The intensity and wave length distribution of the ultraviolet radiation from the welding arc varies with many factors, such as voltage, amperage, size of rod, length of arc, composition of base metal, composition of welding rod and composition of welding rod coating. With all these variables, it is not possible to set absolute standards of length of exposure and distance from the welding arc that are necessary to prevent eye injury. However, with most black iron welding in shipyards, we have found the threshold for nearby workers is about 20 seconds at 7 feet and for more distant workers, 17 minutes at 50 feet. In view of these facts, it is obvious that the term welding flash is a misnomer.

*Treatment of Flash*

The treatment of arc welding flash is simple and symptomatic:

1. Examine the eyes to rule out presence of a foreign body, as the patient may have both a flash and a foreign body. Since the vast majority of welding flashes are bilateral, the presence of a foreign body must be ruled out before making the diagnosis of a unilateral flash.

2. Instil a few drops of a local anesthetic agent such as holocain, butyn, pontocaine, metycaine, etc.
3. Follow with a few drops of adrenalin and cold compresses.
4. Advise dark glasses if the case is severe.

Some physicians have advocated the use of the infra-red lamp in treating welding flash. It has been tried in some shipyard dispensaries but without much success. In the absence of experimental proof of its value, we do not recommend the use of infra-red for treating welding flash in shipyards.

*Protection Against Radiation From Arc Welding*

The primary purpose of the dark glass in welding helmets is to decrease the volume of light to within the range of the retina to get depth perception and to be able to follow the flow of metal from the welding rod. It is most essential that the puddling of metal be always under control. Too much deposit requires grinding and too little makes a weak joint.

In the American Standard Safety Code are given the densities for visible radiation and per cent of transmission for total visible, maximum total infra-red and maximum ultraviolet at 4 wave lengths: 313, 334, 365 and 405 mμ. There is also a description of the type of welding for which each shade is designed. It will be noted that the amounts of ultraviolet and infra-red transmitted are very small. We know from sad experience that the welder rarely gets a flash from his own arc, but rather from a nearby arc when he flips up his hood to inspect or clean his work. Ultraviolet rays reflected from bulkheads, walls, screens, etc., undoubtedly contribute to the accumulation of sufficient exposure to result in a flash.

Workers such as welders' helpers, shipfitters, and pipefitters in the vicinity of welding arcs should also have eye protection. Obviously a shade 1.5, 2.0 or 2.5 will filter out more ultraviolet, visible and infra-red rays than will a clear safety lens. However, visible acuity is greater with clear lenses, thereby reducing accidents from tripping and stumbling, particularly at night. Moreover, we are convinced that clear lenses will filter out enough of the ultraviolet to protect the worker who is only occasionally exposed to the welding arc. It is also easier to get shipyard workers to wear clear lenses. In our yards, therefore, we recommend that all workers occasionally exposed to welding arcs wear clear safety lenses, a practice which has the added advantage of protecting the eyes from flying particles. Of course, if a worker prefers, he may wear a darker shade of lens.

For some time now we have been advocating the wearing of clear safety lenses under a welder's hood. They protect the eyes both from nearby arcs and flying particles and yet do not interfere with visual perception through the dark welding glass. Prescription lenses, which have been discussed by a previous speaker, have their place in welding.

Eye protection is afforded nearby workers through isolation of the welder in a screened booth or behind a portable screen. The portable screen affords adequate protection but it is difficult to get the welder to carry it with him on board ship.

REFERENCES ON RADIATION

1. Laurens, Henry: The physiological effects of radiant energy, Chemical Catalog Company, Inc., 330 West 42nd St., New York, N. Y.
2. Heyroth, Francis F.: The chemical action of ultraviolet rays, Reinhold Publishing Corp., 330 West 42nd St., New York, N. Y.
3. Kuhn, Hedwig S.: Industrial ophthalmology, Chapter VI, 1944, C. V. Mosby Company, St. Louis, Missouri.

4. Kinsey, V. E., Cogan, D. G. and Drinker, Philip: Measuring eye flash from arc welding, J.A.M.A., 123:403-404 (Oct. 16) 1943.

5. Rieke, F. E.: Arc flash conjunctivitis, J.A.M.A. 122:734-736 (July 10) 1943.

6. Cranch, A. G. and Vosburgh, B. L.: Health aspects of welding, 31st National Safety Congress Transactions, 1942, Nation Safety Council, Inc., Chicago, Ill.

7. Drinker, Philip: Measurement and prevention of eye flash, National Safety News, Vol. 49, No. 6, June, 1944.

8. Standards for the protection of workers in gas and electric welding, Division of Labor Standards, U. S. Department of Labor, Washington, D. C.

9. Health protection in welding, Metropolitan Life Insurance Company, New York, N. Y.

10. American Standard Safety Code for the Protection of Heads, Eyes and Respiratory Organs, National Bureau of Standards Handbook H24, 1938, Washington, D. C.

11. Federal Standard Stock Catalog, Section IV, Part 5, "Federal Specification for Goggles: Welders—GGG-G-511" Superintendent of Documents, Washington, D. C.

12. Britton, J. A. and Walsh, E. I.: Health hazards of electric and gas welding, J.I.H. & T., Vol. 22, No. 4, April, 1940 (Contains bibliography of 122 references.)

13. Verhoeff, F. H. and Bell, Louis: The pathological effects of radiant energy on the eye, Proc. Am. Acad. Arts & Sciences, 51:630, 1916.

14. Goggles, Safe Practice Pamphlet No. 14, National Safety Council, Inc., Chicago, Ill.

15. Fleischer, W. E., Nelson, K. W. and Drinker, Philip: Health hazards of welding fumes. J. Maine M. A., Vol. 35, No. 12, Dec., 1944.

16. Control of welding hazards in defense industries, Special Bulletin No. 5, Div. of Labor Standards, U. S. Dept. of Labor, Washington, D. C. 1941.

# THE DEVELOPMENT AND STANDARDIZATION OF A NEW TYPE TEST OF PERIPHERAL VISION

JOHN ALLAN MCCLURE, PH.D.
Newark, N. J.

RECENT studies in industry have proved the value of proper visual skills in relation to job performance, quality of workmanship and accident experience. Results indicate that one of the visual skills, peripheral vision, not previously included in the research, might prove to be an important factor in jobs requiring this skill, such as crane operator, truck driver, and industrial tractor operator. In order that research may be conducted to determine the importance of peripheral vision in industry, the new type perimeter described in this report was developed.

The present study is concerned with the determination of the reliability of the instrument, the relationship of peripheral vision to other visual skills, and the accumulation of standard norms.

## THE APPARATUS AND TEST PROCEDURE

The instrument consists of a base, two swinging arms mounting lamps that contain the test objects, a stationary arm in the center mounting the fixation target and its lamp, protractors for measuring the angle of each swinging arm, enclosures, head rest and the necessary wiring. The light control, an independent unit, consists of a double throw switch that turns the stimulus lamps on steady or into the flash timing circuit, a selector switch that turns the side test object lamps on in various combinations

Formerly of Division of Education and Applied Psychology, Purdue University.
From *Journal of Applied Psychology*, Vol. 30, No. 4, (August) 1946.

with the center lamp and an electronically operated flash timing switch. The experimental model operates only in the horizontal or temporal plane. It was made this way to simplify construction and yet allow for an evaluation of the basic concepts incorporated in the instrument.

All three lamps are lighted with 7 watt 110 volt candelabra bulbs. In the side or test object lamps the light is filtered and diffused through a dark filter and opal glass. On each side of the opal glass is a black opaque paper diaphragm with a centrally located 3/16 inch diameter hole. These apertures on the opal glass serve as the test object when the light is turned on behind them. The opal glass is 17½ inches from the eye. The test object size, given as a visual angle, is 37 minutes. This light source of low intensity is directed toward the eyes through one inch diameter tubes that are lined with lampblack to reduce reflection.

The center lamp has a ½ inch diameter aperture. A disc located transversely in the tube in front of the center lamp mounts eight targets. These fixation targets are opaque ¼ inch numerals on tracing paper held between ground glass covers. The disc is notched on its periphery so that the targets index accurately when the disc is rotated by hand. A small beam from a light under the center target illuminates the front of the center target aperture so that the subject can determine where to direct his attention between flashes of the light stimulus.

The headrest is part of the center metal enclosure. The enclosure is shaped so that when the subject's face is pressed slightly into the headrest, outside light is excluded, and the subject's eyes are positioned centrally in relation to each side lamp. The swinging arms are moved by levers on the protractors. The levers and protractors are located at the examiner's position in front of the instrument.

The circuit is wired so that the center lamp always lights. The test object lamps can be turned on with the center lamp in combinations of right-center, left-center, both-center or neither-center. The electronic flash timer consists of a transformer, resistors, capacitors, an electronic tube and a relay. The wiring diagrams of these circuits can be found in a thesis[1] located in the Purdue University Library.

The timer was set for a flash duration of one-tenth of a second. In preliminary trials it was found that this time gave sufficient exposure without allowing the subject time to shift his eyes.

The principal features built into the perimeter are: 1. Both eyes are tested simultaneously but the right eye cannot see the left field nor the the left eye see the right field; 2. The subject must focus his attention on the center fixation target to read the number flashed; 3. The experimenter can determine whether the subject actually sees the test objects or is guessing; 4. The test can be given in less than ten minutes in its present form; 5. The test is not uncomfortable or fatiguing to the subject; 6. The intensity and duration of light stimulus can be carefully controlled; 7. The purpose and procedure is readily understood by the subject; 8. The field of vision is enclosed to reduce the effects of surrounding illumination; and 9. The instrument is readily portable.

The protractors are set to measure the side angle from straight ahead of the subject's eyes. Actually, when the eyes converge on the center fixation target the field angle for each eye is increased four degrees and twenty minutes. When comparing the results of a perimeter that measures one eye at a

1. McClure, John Allan: The development and standardization of a new type test for peripheral vision, Ph.D. Thesis, Purdue University Library, February, 1946.

time with the results of this instrument this fact must be considered.

The instrument was originally made with the center fixation target similar to the side test objects. To insure center fixation of the eyes and prevent falsification of responses, the test objects and center target first were made to flash successively, with the subject required to repeat the order in which the three lamps flashed. A motor driven brush contacting three adjustable contacts gave the sequence. Three tandem selector switches were wired into the contact circuit to give all six combinations of sequence for the three lamps. This method of presenting the stimulus was discarded because, as the test objects approached the peripheral vision threshold, the subject could not remember the sequence of flashes although he could clearly distinguish that a sequence had occurred. The test given in this manner seemed to be more a test of a special type of memory for perceptual experiences than a test of the field of vision. This method of administration was therefore abandoned.

Enclosure of the field of vision was found to be necessary because various external light sources affected the results when the instrument was not enclosed.

*Test Procedure*

The subject is seated on a stool that is adjusted to the correct height with the subject's eyes level with the headrest of the instrument. The subject is asked to remove his glasses if he wears them. The subject is told that the instrument is a perimeter for measuring how far to each side he can distinguish a dim flashing light while looking straight ahead. He is asked to press his face into the headrest so that his eyes are comfortably centered and so that no light enters around his face. The swinging arms are positioned at 45° from straight ahead. All lamps are turned on, after which the subject is asked what number he reads in the center target. He is then asked if he sees a small dim light on each side. Each side lamp is moved slightly while the subject is asked which one is moving. The examiner does not proceed until he is certain that the subject recognizes positively these side test objects. When testing those individuals with extremely narrow fields the arms are moved in closer than 45°. The purpose of the lighted aperture is explained briefly and demonstrated with a flash of light. The lamps are turned into the flash circuit. With each lamp set on 45° the combinations are explained while flashing center-right, center-left, center-both, and center-neither lamps. The subject is asked if he followed the combinations correctly. If necessary, the examiner again demonstrates and explains until this part is thoroughly understood. The subject is told to respond by telling what number he reads in the center target and which of the side lamps, if any, flash.

The selector switch is set to flash both side lamps. The examiner says "Ready," just before he flashes the lights. If the response is correct the lamps are moved to 65° and again flashed. This large initial increase in the angle works well with the average subject in speeding up the testing procedure. During the practice trials both side lamps are flashed except when there is indecision on the part of the subject. In such cases the increments are smaller and more variations in the lamp combinations are given. If the response is correct on the 65° setting, the side lamps are moved to 75°, and then to 85°. From there on the increment is by five degree intervals. Both arms are already set at the same angle from straight ahead. When a setting is reached where the subject starts to give incorrect responses for either eye, or

reports that he fails to see the test objects, three or four extra trials are given to be sure that the subject's threshold, on one or both of his eyes, has been passed. The arms are then brought forward five degrees to a smaller angle and four or five check trials are given. When the subject responds correctly on these practice trials the test trials are begun.

## EXPERIMENTAL PROCEDURE

### *Subjects*

Two hundred and two subjects enrolled in psychology courses were used as subjects. There were 96 males and 106 females. Their ages ranged from 16 to 40 with an average of 21.1 and a standard deviation of 8.37. Two subjects were scheduled for each half hour period.

### *Other Visual Skill Tests*

The Bausch and Lomb Ortho-Rater was used to measure the visual skills of far vertical phoria, far lateral phoria, far acuity of both eyes, far acuity of right eye, far acuity of left eye, color vision, depth perception, near acuity of both eyes, near acuity of right eye, near acuity of left eye, near vertical phoria, and near lateral phoria.

Each subject was given the perimeter test first and then the Ortho-Rater test followed by a retest on the perimeter. There was an approximate lapse of fifteen minutes between the first test and the retest with the perimeter. About one-third of the subjects were tested on days when the weather was cold and clear with bright sunshine on freshly fallen snow. Because of the close scheduling of subjects, only about five minutes could be allowed for light adaptation before the first test. The room lights were on during the testing.

## SUMMARY AND CONCLUSIONS

The perimeter described is adaptable for industrial and laboratory testing of peripheral vision limits because it tests rapidly and objectively, has satisfactory reliability, and can be operated successfully by an examiner without clinical training. The subject cannot falsify his responses because he must fixate both eyes on the center fixation target and the examiner, by controlling test object lamp flashes, can determine when the subject's responses are wrong.

The research has revealed a rather wide range of individual differences in the extent of the visual fields.

Peripheral vision, as measured by this instrument, has been found to be relatively independent of the visual skills of acuity, vertical and lateral phoria, depth perception and color discrimination.

## REFERENCES

1. Burnham, R. S.: A perihemisphere for visual measurements, J. Exper. Psychol. 27:333-336, 1940.
2. Foerster, R.: Vorzeigung des perimeter, Elimische Monatsblätter für Augenheil kund, Stuttgart, 7:411-422, 1869.
3. Low, F. N.: Studies on peripheral visual acuity, Science, 97:586-587, 1943.
4. Mayer, L. L.: Light stimuli of minimal measured duration as a means of perimetry, Arch. Ophth., 14:541-553, 1935.
5. Pascal, J. I.: An improved perimeter-campimeter, Arch. Ophth., 16:103-105, 1937.
6. Purkinje, J.: Beobachtungen und Versuche zur Physiolo. der Sinne, Prag. J. G. Calve, 1823.
7. Thomasson, A. H.: A plea for greater uniformity in methods of field taking, Arch. Ophth., 12:21-32, 1934.
8. Traquair, H. M.: An introduction in clinical perimetry, London, Henry Kimpton, 1942.
9. Von Graefe, A.: Archiv. für Ophth. Berlin, 1855.
10. Young, T.: Mechanism of the eye, Royal Society of London, Philos. Trans. London, 1801.

# INDUSTRIAL EYE PROTECTION PRACTICE

JOHN M. ROCHE
CHICAGO, ILL.
and
HEDWIG S. KUHN, M.D.
HAMMOND, IND.

THE greatly increased use of eye protection devices in industry during the war years produced definite evidence that industrial eye injuries could be controlled. This was dramatically demonstrated in the shipbuilding industry where, after an initial bad experience, eye injuries were reduced nearly 75 per cent by the introduction and use of adequate and proper eye protection devices.

The sharply decreased supply of skilled and unskilled manpower for war industries was one of the principal factors in the suddenly aroused interest of plant managers in making the best possible use of the available labor supply, and in keeping workers on the job through accident prevention and health maintenance programs. The attention given to the prevention of eye injuries and to the correction of visual deficiencies was one of the important by-products of these programs.

Executives had impressed on them by sheer necessity what ophthalmologists, optometrists, and safety engineers had been preaching for years—that protecting good eyes and bringing defective eyes as near to normal as possible with corrective lenses was good business. As a result, many companies adopted the "universal goggle rule" and many others instituted complete eye examination plans with arrangements to supply hardened corrective lenses for those employees needing them. Most companies supplied plano goggles free, some supplied even corrected goggles free. However, in the latter case, a variety of plans were evolved which ranged from giving the employee the eye examination and corrective goggles without charge to making the employee pay all costs.

While the net results were a reduction, or at least holding the line, on the frequency of industrial eye injuries, and the more intangible but self-evident improvement of work efficiency, it was inevitable that mistakes were made. In some cases improper eye protection devices were selected, some methods of enforcing the wearing of goggles, both plano and corrective, were ill advised and aroused the opposition of workers, and there were complaints, too, from ophthalmologists and optometrists that eye examinations were being made and prescriptions written for corrective lenses by unqualified persons.

So that the gains made in the war period could be maintained and improved upon, the National Safety Council in 1942 conducted a Survey on Industrial Eye Protection of its 7,500 members to determine industrial practice with respect to eye protection devices. The results of the survey were studied by a committee of 40, under the chairmanship of Hedwig S. Kuhn, M.D. Industrial users, manufacturers and distributors of eye protection devices, ophthalmologists, optometrists, safety engineers, and organized labor were represented. This committee, representing the best informed opinion on the subject, after a thorough study of the results of the survey, made basic recommendations for the guidance of industry in solving its eye protection problems.

It is important to study some of the data developed by the survey as this determined to a large extent the conclusions of the committee. The data developed by the survey are based on returns received from 730 companies of the 7,500 who were solicited for information. This approximate 10 per cent return is an unusually large one for any

Reprinted from *National Safety News*, July, 1947.

type of survey, and the information gleaned may be considered indicative of average industrial practice. The reports represented the experience of 1,745,633 employees working more than a billion man-hours annually.

Definitely proved by the survey is the fact that *eye injuries are under control.* Figures presented by the 730 companies show that the frequency of lost-time eye injuries was 0.666 accidents per million man-hours as compared to the over-all average frequency for lost-time injuries of 13.63. This indicates that eye injuries are responsible for only about 4.87 per cent of the lost-time accidents.

The highest rate for lost-time eye injuries was reported by the mining industry; foundry operations were next highest; lumbering was third, while the wood-working and leather industries were fourth.

### METHODS OF FURNISHING GOGGLES

Plano or noncorrective hardened lens goggles are almost universally furnished free of charge to employees, 98 per cent stating that employees were given the goggles without charge.

There is a wide variation in practice in furnishing prescription goggles. Five hundred and nineteen companies stated that prescription hardened protective goggles were worn by at least some of their employees. In 27 per cent of these cases, the goggles were furnished free by the company, while 60 per cent of the companies assisted their employees in obtaining the goggles at less than retail price. Sixty-seven of the companies gave no financial help whatever to the employees who needed corrective protective eye wear.

Notable examples of companies furnishing corrective goggles to their employees entirely without cost are the Hawthorne Works of Western Electric Company in Chicago and the Pullman Company, also in Chicago. In both of these companies the eye examinations and goggles are entirely paid for by the company. The almost total elimination of eye injuries in both companies is dramatic evidence to the effect that the investment the company is making in prescription protective eye devices is well worth while.

### *Maintenance Practice—Plano Goggles*

Some of the principal objections raised by employees to wearing protective equipment include: "They don't feel comfortable"; "I can't see through them"; "They're unsanitary"; and "They make me look funny."

The replies of the survey indicate that there is considerable room for improvement in employee relationships involving selling the workers on the necessity and wisdom of wearing goggles. Most of the objections given by employees can easily be rooted out of their minds by a personal interview with a person who is trained to fit goggles and answer the arguments so frequently raised. In only 42 per cent of the cases were trained personnel employed to fit nonprescription goggles. The larger companies, those employing 2,000 or more, employ such personnel in 65 per cent of the cases. This drops to a low of 16 per cent in companies having fewer than 100 employees.

About half of the companies represented place specific responsibility for maintaining the goggles in good condition on their safety department, or on other departments such as first aid. Employees are either required to turn in goggles which have become defective, or the responsible department heads make periodic checks of all goggles and put them in first-class condition. Only 39 per cent of the companies supply goggle cleaning facilities and the majority of these companies are in the class of employing 500 or more employees.

Washing with ordinary soap and water is considered to be satisfactory.

*Universal Goggle Plan And Enforcement*

An unexpectedly large number of companies (20 per cent) stated that they had a rule requiring all employees to use eye protection. There is no marked difference according to the section of the country in connection with this practice; however, surprisingly enough, a greater proportion of small companies have the universal goggle rule than was found true in the larger companies.

About 88 per cent of the companies require that employees wear goggles while performing jobs which are considered to be hazardous to the eyes or while employees are in departments, regardless of whether they are working or not, where eye hazards exist. Industrial plants in the East and far West have the best record in this respect.

The question of enforcing the wearing of goggles is an interesting one. There is a long hard road between putting in a universal goggle rule in the plant and getting all employees to cooperate by wearing the eye protection provided. Most companies successfully applying the universal goggle rule have found that "selling the idea" took several years and required considerable persuasion and some discipline on the part of supervisors and management. Where persuasive persistence was the order of the day, success was usually achieved. Discipline alone did not work; education of the employee fared equally badly but a combination of the two seems to have succeeded.

*Corrected Hardened Eye Protection (Prescription Goggles)*

Supplying corrected hardened lenses for employees requiring them is a logical and desirable step in any industrial eye protection program. Too often, the eye protection problems in a plant are considered settled if each employee is furnished with the proper type of plano hardened lens goggles. Where the worker's vision is not normal, this causes inefficiency in his work, or at the worst, makes it impossible for him to work at all, and the company actually loses money because the optimum type of protection has not been made available.

The most desirable, and in the long run the least expensive, type of eye program is one which includes a complete eye examination for all employees by a professional eye man supplemented by some plan which will make the necessary corrected hardened lens eye protection equipment available for employees. In some cases, companies forego this program and provide "cover-all" goggles for employees who must wear glasses. While cover-all goggles with hardened lenses do protect eyes, the combination of the corrected spectacle plus the cover-all is less efficient than a hardened corrective lens goggle.

The cover-all goggle which is still widely used in industry, should be confined, according to authorities, to occasional and intermittent use by employees whose work would require them to wear several different types of goggles, such as a machinist, who is occasionally called upon to do welding, or employees who do work requiring various focal distances. They are also useful for executives or visitors who are only occasionally, and for a short time, exposed to the hazard of eye injuries. They can be used to good effect while employees are awaiting the delivery of hardened prescription lens eye protection.

Where corrected hardened lenses are used, care should be taken that they are ground to the proper work distance and not to the prescription for the employee's every-day spectacles. A man operating a crane would require a different type of prescription from the

mechanical standpoint of work distance than that of a man operating a lathe or running a milling machine.

In conjunction with prescription goggle programs, 55 per cent of the companies make pre-employment vision tests, in 74 per cent of the cases, the examinations are made by physicians. Plant nurses made eye tests in 14 per cent of the cases, and in 12 per cent the examinations were made by outside consultants.

One of the factors too frequently neglected was that of consideration of near vision requirements for jobs. In 191 cases, companies indicated that they gave no consideration to near vision requirements; however, an analysis by industry showed that companies engaged in machine shop operations and those manufacturing electrical equipment, machinery, and other precision products, did give some consideration to near vision. Since this is one of the most important factors in assuring visual efficiency, it would seem obvious that it should be included in all cases where hardened prescription lenses are furnished.

*Case Histories*

In addition to the factual information obtained from the survey described above, much helpful and valuable information was volunteered by some companies who reported. One company, in stating the value of its eye protection program, said that they furnished all goggles, face shields and prescription goggles free of charge to employees and that "we have not had a serious eye injury since our goggle program was originated in the fall of 1944 and feel that it was money well spent."

Another company employing 600 workers said that their eye accidents have been reduced 75 per cent one year after the installation of the program, while still another company that inaugurated a complete eye protection program that required all factory employees to wear goggles, stated that no accident had been reported since the program was put in effect.

While it is generally recognized that complete eye protection programs are self-liquidating, some companies report discouraging results in attempting to get employees to wear the protective equipment provided.

The following recommendations were developed by the National Safety Council's Committee on Industrial Eye Protection at its meeting on October 8, 1946 at the Stevens Hotel in Chicago, Illinois.

In reviewing the recommended practices, it should be borne in mind that it is assumed that a thorough engineering survey has been made of the plant and that every effort has been made to remove physical eye hazards by guarding the exposures. Not stated in the recommended practices, but highly advisable is the need for periodic re-study of eye accidents reported so the eye protection program may be adjusted to meet current developments.

Plastic face and eye protection was not discussed by the committee as no standards have as yet been set, however, it would seem desirable that its use as supplementary protection to the face be seriously considered. A recent research completed for the American Society of Safety Engineers by the Batelle Memorial Institute indicates there are many jobs where plastic face and eye protection offer adequate and efficient protection.

## RECOMMENDED PRACTICES

*Plano Goggles*:

(1) *That*: A comprehensive analysis of all jobs, operations, and processes be made in each industrial plant to determine the eye hazards that exist and the proper type of eye protection

## COPY OF SURVEY SHEET

### INDUSTRIAL EYE PROTECTION SURVEY

As a result of many requests from members, the National Safety Council is conducting this survey to determine general industrial policy and practice with respect to eye protection programs. Your help is asked in this national survey; please fill out completely this form and return it promptly. All data will be considered confidential. The results of the survey will be made available to all Council members.

Name of company ..... Address .....
(May be left blank if desired)

Number of employees ..... Products manufactured .....

Typical operations .....
(i.e., machine shop, foundry, light or heavy chemical mfg., etc.)

### SECTION I — NON-PRESCRIPTION GOGGLES AND FACE PROTECTION

TYPES OF FACE AND EYE PROTECTION USED (CHECK WHICH)

| *Hardened Glass Lens* | *Plastic Face and Eye Protection* | *Other Types* |
|---|---|---|
| ☐ Spectacle without side shields | ☐ Plastic-lens goggles | ☐ Wire screen goggles |
| ☐ Spectacle with side shields | ☐ "Short" face shields | ☐ Wire screen mask |
| ☐ Goggles over spectacles | ☐ "Short" face shields | ☐ Rubber mask |
| ☐ Cup direct ventilation | ☐ Tinted face shields | ☐ Hand shield |
| ☐ Cup indirect ventilation | ☐ Others | ☐ Welding helmets |
| ☐ Filter lens-welders-burners | | ☐ Others |
| ☐ Filter lens-glare | | |
| ☐ Flat type lens | | |
| ☐ Curved type lens | | |
| ☐ Others | | |

Damaged face shields repaired?..... Replaced?.....Goggles free to employee?.....
Face shields free?.....Welders helmets free?.....If these devices not free, describe your plan. .....

Goggles fitted by trained employee?.....Who trained employee? .....
How do you maintain goggles, welding helmets and similar devices? .....

Goggle cleaning stations provided?.....How often are goggles sterilized?.....
Do you have rule requiring all employees to use eye protection?.....Certain departments?.....
Explain: .....

How do you enforce rule? .....

Comments on phases not covered above .....

**ALL QUESTIONS ON THIS SIDE OF FORM REFER TO NON-PRESCRIPTION EQUIPMENT**

(Over)

## SECTION II — HARDENED GLASS CORRECTIVE VISION GOGGLES

*Note: Questions in Section II pertain to prescription goggles only.*

Prescription goggles furnished free?..... Who pays for examination: Company?.....Employee?..... Pre-employment vision tests made?..... By nurse at plant?..... Doctor at time of physical examination?..... Outside consultant?..... Does employee choose examiner? ..... Check which are tested and recorded: Distant vision?..... Near vision?..... Color perception?..... Depth perception? .....Binocular coordination?..... Are individual visual tests made?..... Or are battery tests used?..... Telebinocular?..... Orthorator?..... Sight screener?..... Snellen chart?..... Projector chart?..... Are following groups retested periodically: Cranemen?..... Chauffeurs?..... Electric truck operators?..... Others? ..... How often?.....

Any special attention given to near vision operations as close inspection, small part assembly, etc.? .....

Is prescription copy of that for personal dress glasses or is work distance taken into account? .....

If employee pays all or part of cost of goggles, describe your plan in detail. .....

.....

.....

.....

.....

.....

.....

Goggles purchased from manufacturer?..... Distributor?..... Other?.....

Employee pay for normal replacement?..... Pay for goggles if broken?.....

Employee retain goggles on termination employment? .....

## SECTION III — OTHER DATA

Man hours worked 1945?..... Total lost-time accidents 1945?.....

Number eye accidents 1945: First aid?..... Lost time?..... Compensable?.....

Plant frequency rate all accidents?..... Plant severity rate all accidents?.....

Frequency rate lost time eye cases?..... Severity rate lost time eye cases?.....

Please make any comments you believe would be helpful. .....

.....

.....

.....

.....

.....

.....

.....

.....

CONFIDENTIAL

.....

(Signature) (Title) (Date)

These comments will be summarized, but in no case will any data be identified by company name. A copy of the summary will be sent to all members.

National Safety Council
20 North Wacker Drive, Chicago 6

Printed in USA
6-46-8M

which should be made available to employees to protect them against specific hazards.

(2) *That*: The issuing, fitting, servicing and maintenance of plano hardened glass eye protection be done by persons who have received thorough training for this work, preferably by by professional personnel.

(3) *That*: Wherever an eye hazard has been shown to exist, the agreed-upon eye protection method should be strictly enforced.

(4) *That*: The current American Standards Safety Code for the Protection of Heads, Eyes and Respiratory Organs, Z2-1928, or existing government specifications such as the Federal Specification for Rubber Frame Goggles (GGG-G521); Federal Specification for Eyecup, Protective Impact Resisting Goggles (Chippers; Grinders; etc). (GGG-G501b) and Navy Department Specifications for Protective Goggles (Frames and Lenses); (Glare and Welders) 37G16, be used as guides in selecting eye protection equipment for specific applications.

(5) *That*: Employers furnish plano hardened glass eye protection devices to employees free of charge and each employee should be furnished his own pair. If goggles are re-issued, they should first be sterilized.

*Prescription Goggles*:

(6) *That*: All employees already wearing glasses or needing correction to see properly should be provided with prescription hardened lens eye protection. New employees should be examined prior to placement on jobs.

(7) *That*: The financial policy selected for making prescription ground hardened lens glass eye protection equipment available to employees be given careful consideration by company executives.

(8) *That*: Management make every effort to arrange for or advise proper professional eye examination for those employees needing it.

(9) *That*: Descriptive details of the normal working distance (distance between the employee's eyes and the actual work point) be made available to those writing prescriptions for corrected hardened glass lens eye protection.

(10) *That*: When corrected lenses are received, the lenses be verified and the goggles be fitted to the employee's face by a professionally trained person.

(11) *That*: Corrected hardened safety lenses be supplied only in frames meeting the specifications of the American Standard Safety Code for the Protection of Head, Eyes, and Respiratory Organs, Z2-1928, or the specifications of other government agencies as noted in Recommendation No. 4.

(12) *That*: The use of the "cover-all" type of hardened plano glass eye protection used over spectacles should be limited to those employees whose work, according to the job analysis, makes it impracticable to use the usual type of corrected hardened lens eye protection. Their use is recommended for employees or plant visitors wearing corrected glasses whose exposure to the plant operation is only casual and of limited duration or for employees who are awaiting delivery of corrected hardened lens safety glasses.

*Supplementary Recommendations*:

(13) *That*: The National Safety Council and its affiliated organizations are urged to do everything possible to encourage the use of eye protection devices in primary, secondary, and college vocational training shops and laboratories. Safety training materials can be provided that will show students the need for protecting their eyes against shop hazards. This will condition them to the eye protection programs they will meet when they enter industry.

## BLIND VETERANS FIND EMPLOYMENT

NEARLY half of the veterans who lost their sight in World War II either are working or in training for employment, a Veterans Administration survey disclosed.

The study, made by VA's Vocational Rehabilitation and Education staff, included 1,133 blinded veterans who had been discharged by the Army and Navy, up to the end of November, 1946.

From *National Safety News*, June, 1947

Twenty-eight per cent, or 321, were employed either full or part time or were working for themselves. Another 20 per cent, or 231, were in schools, colleges and on-the-job training.

Business and sales attracted 150 blinded veterans. Of these, 31 were in educational institutions learning to manage retail shops, sell advertising and insurance, and to handle administrative positions. Twenty-two were training on-the-job, most of them as salesmen and vending stand operators. Sixty-six were employed full or part-time. They held positions ranging from managing an oil company to bartending. Another 31 were self-employed in businesses such as filling stations, salvaging, magazine agencies, bakery, liquor stores, trucking, gift shops and motion picture houses.

Professional and scientific fields accounted for 129 of the sightless veterans. Over two-thirds of this group, or 88, were in schools and colleges studying law, social work, teaching, writing, economics, vocational guidance and numerous other subjects. Ten were taking on-the-job training as masseurs, reporters, beauty operators and claim adjusters. Thirty were employed in such specialized fields as teaching, employment, public relations, radio, and physical therapy. Two of these were representatives in the legislatures. One was self-employed as a lawyer.

One hundred and sixty veterans were at work in, or training for, positions in trades and industrial fields. Seventeen were in educational institutions, with their most popular subjects piano tuning, automobile mechanics and radio repairing. Fifteen were taking job training as machine operators, assemblers, automobile mechanics and panel board wiremen. Of the 112 who were employed full and part-time, 34 were assemblers; 12 leather workers, and the remainder, machine operators.

## CONTINUING BIBLIOGRAPHY ON OCCUPATIONAL EYE PROBLEMS


ALVIS, B. Y. *The Use of the Berman Locator In Removal of Intraocular Foreign Bodies.* South. M. J., 39:125, Feb., 1946

ANON. *Light for Seeing.* Nat. Safety News, 53:17, March, 1946.

ANON. *Eye Defects Which Might Interfere With a Person Driving Safely at Times.* National Driver Examiner, March 15, 1946.

ANON. *Better Eyesight Will be Sought by American Workers.* Scientific American, 173:106, Aug., 1945.

ANON. *Micrometers for the Blind.* Canad. M. A. J., 55:305, Sept., 1946.

ANON. *Color Blindness in Coal Mining.* United Mine Workers, Feb. 1, 1946.

BALTIN, M. M. *The Extraction of Intraocular Foreign Bodies.* Vestnik Oftal., 23:20, 1944.

BEAUMONT, W. *The Blind in War-time Employment.* Brit. J. of Phys. Med. 7:17, Jan.-Feb., 1944.

BELLESINI, C. *Picric Acid in Therapy of Caustic Soda Burns on Eyes.* Med. d. lavoro, 37:222, June, 1946.

*BIRREN, FABER. *Color and Lighting Co-ordination in Industrial Interiors.* Lighting and Lamps. 45:6, 22-23, Aug., 1944.

CAPEL, ERNEST H. *Seeing the Job.* Brit. J. Phys. Med. & Indus. Hyg. 8:105-111, July-Aug., 1945.

CASHELL, G. B.,SHAY, H. B. and BODENHAM. *Case of Sulphuric Burns.* Brit. J. Surg. 31: 373-377, April, 1945.

COHEN, JOZEF. *Color Adaptation of the Human Eye.* Am. J. Psychol. 59:84-110, Jan., 1946.

CROUCH, C. L. *The Relation Between Illumination and Vision.* Illum. Engin. 40:747-84, Nov., 1946.

*DREYER. *Personalize Your Eye Protection Program.* Nat. Safety News. 55:22-23, 79. Apr., 1947.

ELIAS, J. P. *Medical Grading of Worker* Brit. J. Indus. Med. 3:11-14, 1946.

ELYRICH, R. *How's Your Shop Eye.* Popular Science, 148:165, June, 1946.

ESTEBAN, MARIO. *The Estimation of Ocular Disability in Industrial Injuries.* Arch. de la Soc. Oft. Hisp.-Amer. 5:965-980, Nov., 1945.

FARMER, E. *Adjustment of Blind in Industry.* Lancet 2:474-475, Oct. 13, 1945.

FEIGENBAUM, A., MICHAELSON, I. C. and KORNBLUTH, W. *Epidemic Keratoconjunctivitis in the Middle East; Clinical and Experimental Study.* Brit. J. Ophth. 29:389, Aug., 1945.

FINTELEN, F. *Etiology and Therapy of the So-called Keratoconjunctivitis Epidemic.* Ophthalmologica 3:101, Feb.-March, 1946.

GIBSON, A. and CARLISLE, J. W. *Industrial First Aid in Chemical Injuries of the Eye.* Sight Saving Review; 15:220-227, Winter, 1945, N.S.P.B. Pub. 439.

GONZALES, J. B. *Dacryostomy in Industrial Accidents.* Arch. de la Soc. Oft., Hisp.-Amer. 5:887-895, Oct., 1945.

GRAHAM, ROBERT. *Reduction of Reflections.* Arch. Ophth. 36:3, 315-320, Sept., 1946.

GRIEVE, JOHN. *Incidence of Defective Colour Vision.* Nature, 157:376, March 23, 1946.

HAGEDORN, A. *Seed Grafting of the Conjunctiva In the Treatment of Symblepharon.* Am. J. Ophth. 29:12, 1568-1570, Dec., 1946.

*HARDY, LEGRAND H. and RAND, GERTRUDE AND RITTLER, M. CATHERINE. *Effect of Quality of Illumination on the Results of the Ishihara Test.* Arch. Ophth. 36:6; 685-699, Dec., 1946.

HUGHES, WM. F. *Treatment of Lewsite Burns of the Eye with Dimercaprol* ("Bal"). Arch. Ophth. 37:1, 25-41, Jan., 1947.

JAHN, T. L. *Brightness Discrimination and Acuity as Functions of Intensity.* J. Optic. Soc. America, 36:83-86, Feb., 1946.

JONES, LEONARD W. *Common Eye Injuries.* Indust. Med. 15:692-693, Dec., 1946.

*KEPHART, N. C. *An Example of Increased Production Through an Industrial Vision Program.* Optical J. and Review of Optometry, Sept. 15, 1946.

*KOVEN, A. L. *Right Eyes for the Right Job: Testing Vision in Industry.* Indust. Hyg. News Letter, 7:4-6, Apr., 1947.

*KOZLOSKI, H. R. *Vision Testing at Western Electric, Baltimore, Md.* The Pointer, Western Electric Co. 15:2, July, 1946.

KUGELBERG, I. *Eye Symptoms in Cases of Producer Gas Poisoning.* Nord. Med. 28: 2691, Dec. 21, 1945.

*LUDVIGH, E. and KINSEY, V. E. *Effect of Long Ultraviolet Radiation on the Human Eye.* Science 104-246, Sept. 13, 1946.

LUZ, B. DA, AND SEBAS, S. R. *Foreign Body Tolerated for 11 Years.* Rev. Brasileira de Oft. 4:313-316, June, 1946.

McLAUGHLIN, A. I. G., MILTON, R. AND PERRY, K. M. A. *Toxic Manifestations of Osmium Tetroxide.* Brit. J. Indust. Med. 3:183, July, 1946.

MANN, IDA, *"Blue Haloes" in Atebrin Workers.* Brit. J. Ophth. 31:1-40-46, Jan., 1947.

RAGLE, L. *Switchyard Lighting Speeds Rail Traffic.* Electrical World. Dec. 8, 1945.

RAGGIO, F. W., JR. *The Management of Common Eye Injuries in General Practice.* New Orleans M. & S. J. 99:165-169, Oct., 1946.

RESNICK, LOUIS. *Accomplishments and Further Needs in Industrial Eye Protection.* J. of Social Ophth. 3:14-21, July, 1946.

SAMUELS, BERNARD. *Foreign Bodies in the Eye.* Sight Saving Rev. 16:63-67, Summer, 1946.

*SNELL, ALBERT C., JR. *Perforating Ocular Injuries.* Am. J. Ophth. 28:263-281, March, 1945.

*TIFFIN, JOSEPH AND LONG, W. F. *Visual Standards and Job Requirements.* Am. J. Optom. and Arch. Am. Acad. Optom. 23:463-467, Nov., 1946.

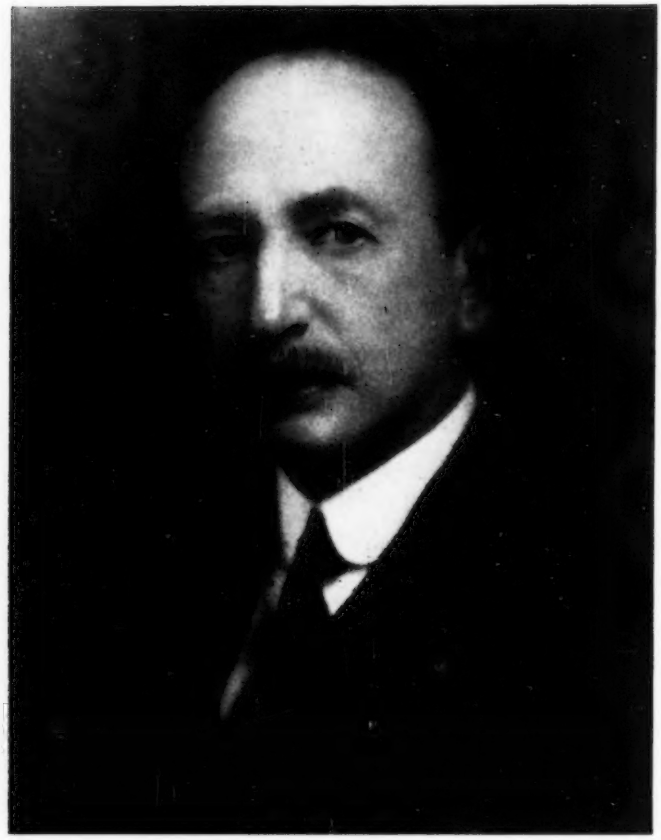
VAIL, DERRICK. *Early Treatment of Ocular Injuries.* Indust. Med. 16:173-174, April, 1947.

*WRIGHT, J. S. *Welder's Flash—Its Causes and Cure.* Welding Engr. 30:52-54, May, 1945.

WYLIE, ROBERT RUSSELL. *Practical Color Harmony with Fluorescent Light Sources* Illum. Engin. 41:10, 847-858, Dec. 1946

* Articles especially pertinent

# *Guest of Honor*



**FREDERICK HERMAN VERHOEFF, Ph.B., M.A., M.D.**

Graduate of the Johns Hopkins University School of Medicine; Consulting Chief of Ophthalmology, Massachusetts Eye and Ear Infirmary; Emeritus Professor of Ophthalmic Research, former Director of Howe Laboratory of Ophthalmology, Harvard University; diplomate American Board of Ophthalmology; Fellow, American College of Surgeons; American Academy of Ophthalmology and Otolaryngology; American Academy of Arts and Sciences; American Association for Advancement of Science; American Medical Association (Chairman, Section of Ophthalmology, 1932); member, American Ophthalmological Society (President, 1937-38); New England Ophthalmological Society (President, 1920-22); Recipient of Knapp Medal and Ophthalmic Research Medal of the American Medical Association, and of Howe Medal of the American Ophthalmological Society; served as Major, Medical Corps, U.S.A., during World War I; Chief of Ophthalmic Service, Base Hospital, Camp Devens.

FIFTY-SECOND ANNUAL CONVENTION

American Academy of Ophthalmology and Otolaryngology

OCTOBER 12 TO 17, 1947

PALMER HOUSE

*Chicago, Illinois*

# OFFICIAL NOTICES

## Convention Call

Under the authority of the Constitution and with the approval of the Council, I herewith proclaim that the Fifty-Second Annual Convention of the American Academy of Ophthalmology and Otolaryngology will convene at the Palmer House, Chicago, Illinois, on Sunday, October 12, 1947, and continue through noon Friday, October 17, 1947.

ALAN C. WOODS, M.D.
*President*

*Attest:*
WILLIAM L. BENEDICT, M.D.
*Executive Secretary-Treasurer*

## Local Committees

WILLIAM A. MANN, M.D.
*General Chairman*

### *Reception Committee*

John F. Delph, M.D., *Chairman*
Thomas D. Allen, M.D.
L. B. Bernheimer, M.D.
Louis Bothman, M.D.
James W. Clark, M.D.
Arthur J. Coombs, M.D.
Francis M. Crage, M.D.
Beulah Cushman, M.D.
Ralph A. Davis, M.D.
Earle B. Fowler, M.D.
Alice K. Hall, M.D.
Louis G. Hoffman, M.D.
Paul H. Holinger, M.D.
Arlington C. Krause, M.D.
Abraham M. Lazar, M.D.
James E. Lebensohn, M.D.
Earl S. McRoberts, M.D.
G. Henry Mundt, M.D.
Glenway W. Nethercut, M.D.
Richard A. Perritt, M.D.
Elias Selinger, M.D.
George E. Shambaugh, Jr., M.D.
Clifford P. Sullivan, M.D.
Virgil Wescott, M.D.

### *Committee on Arrangements*

J. Robert Fitzgerald, M.D., *Chairman*
Albert H. Andrews, Jr., M.D.
Carl Apple, M.D.
Nora B. Brandenburg, M.D.
Carl H. Christoph, M.D.
Jack P. Cowen, M.D.
Cyril V. Crane, M.D.
Noah B. Fabricant, M.D.
Richard C. Gamble, M.D.
Elmer W. Hagens, M.D.
Jerome W. Hayden, M.D.
Helen Holt, M.D.
Bertha A. Klien, M.D.
Peter C. Kronfeld, M.D.
Vernon M. Leech, M.D.
Earl H. Merz, M.D.
Herbert T. Nash, M.D.
Walter H. Theobald, M.D.

### *Ladies' Committee*

Mrs. William A. Mann, *Chairman*
Mrs. Thomas D. Allen
Mrs. Ralph A. Davis
Mrs. John F. Delph
Mrs. Richard C. Gamble
Mrs. Earle B. Fowler
Mrs. Myron M. Hipskind
Mrs. Louis G. Hoffman
Mrs. G. Henry Mundt
Mrs. Richard A. Perritt
Mrs. Peter C. Kronfeld
Mrs. Irving Puntenney
Mrs. Kenneth L. Roper
Mrs. Gail R. Soper
Mrs. George E. Shambaugh, Jr.
Mrs. Derrick T. Vail
Mrs. Edwin A. Wegner
Mrs. Virgil Wescott

## Council Meetings

The Council of the American Academy of Ophthalmology and Otolaryngology will convene at 9 o'clock, Saturday morning, October 11, 1947, in Private Dining Room No. 4 on the 3rd floor of the Palmer House, Chicago. There will also be a meeting of the Council on Wednesday, October 15, at 12:45 in Private Dining Room No. 5. Members desiring to be heard or to present business will confer with the Executive Secretary for appointments.

ALAN C. WOODS, M.D.
*President*

## New Business

Proposals for new activities often are presented too late during the Academy session to afford time for deliberation. Therefore, new items requiring Council action should be presented in writing to the Executive Secretary early so that the Committee concerned may have an opportunity to study the matter prior to presentation to the Council.

## Public Relations

Contributors to the scientific program are requested to send copies of their papers to the Secretary for Public Relations, Dr. Erling W. Hansen, Medical Arts Building, Minneapolis 2, Minnesota, at the earliest possible moment.

ALAN C. WOODS, M.D.
*President*

## Exhibits

The Scientific and Technical Exhibits will open at 9 o'clock Sunday morning, October 12.

## Registration

*General registration will begin at 9 o'clock Sunday morning, October 12, in the Foyer to the Grand Ball Room, Fourth Floor.*

The regulation badge secured at the time of registration is essential for admittance to all activities. To facilitate registration, the following sections will be provided:

**Advance Orders:** Members having advance ticket orders register here and collect tickets.

**Members without Advance Orders:** Please present Receipt Card for 1947 dues. Dues outstanding may be paid at this section.
*(Application blanks for Fellowship in the Academy may be obtained here.)*

**Instructors:** All members and guests presenting instruction courses register here to secure special badge.

**Non-Members:** A registration fee of $5.00 is required of all guests with the following exceptions:

1. Candidates for Fellowship listed in the July-August issue of the TRANSACTIONS and eligible for election at this meeting.
2. Residents presenting exemption card previously obtained by Head of Department of institution where training.
3. Orthoptic Technicians.
4. Guests taking part in the Scientific Programs or Exhibits.

## Instruction Course Tickets

To expedite the purchasing of instruction course tickets, the tickets for courses in ophthalmology and the tickets for courses in otolaryngology and maxillofacial surgery will be sold at separate desks. The bulletin boards behind each desk will indicate what tickets are available. Please consult these boards in making your selections. Bear in mind that individual courses are for one hour only; continuous courses run for two or more hours. Price of tickets is $1.50 per hour.

## Available Supplies

Abstracts, monographs, and manuals may be obtained at this desk.

## Dinner Tickets

A special desk will be provided for the purchase of tickets for Alumni Dinners and the Annual Academy Dinner. Sale of tickets closes at 2 o'clock in the afternoon of the day of the dinner.

## Ladies' Registration

The wives of members and lady guests are urged to register at the Ladies' Headquarters in Rooms 15 and 16 on the Club Floor. Registration will open at 10 o'clock Sunday morning, October 12. Special functions for the ladies will include a luncheon and style show at Marshall Field's and a sightseeing tour and tea at the South Shore Country Club. Bridge tables and refreshments will be available in the Club Rooms.

## Guests

Physicians are welcome to attend the meeting of the Academy and to participate in all the activities. All guests, however, will be required to register and a fee of $5.00 will be charged.

## Residents Registering as Guests

Men in training in accepted residencies, or those now completing a full academic year (nine months) in basic study, registering as guests at the Academy Convention will be relieved of paying a guest fee only when the Department Head concerned officially requests this favor at least 10 days prior to the opening date of the convention.

## Dues

Because of second class mail regulations the TRANSACTIONS can be sent only to members in good standing. Receipt card for 1947 dues is required for registration at the Academy Convention in Chicago, October 12-17, 1947. Those who have not paid dues for 1947 are requested to remit to Dr. W. L. Benedict, Executive Secretary-Treasurer, 100 First Avenue Bldg., Rochester, Minnesota.

## Constitutional Changes in Fellowship Status

### Life Fellows — 1948

Atkinson, D. T. ..... San Antonio, Texas
Auerbach, Julius ..... New York, N. Y.
Barkan, Hans ..... San Francisco, Calif.
Beyer, Louis J. ..... Buffalo, N. Y.
Boeckmann, Egil ..... St. Paul, Minn.
Bone, Herman D. ..... Gardner, Mass.
Bordley, James, Jr. ..... Baltimore, Md.
Campbell, C. H. ..... Columbus, Neb.
Cary, G. C. ..... Grand Junction, Colo.
Chase, E. Frank ..... Seattle, Wash.
Clement, C. C. ..... Chicago, Ill.
Curdy, R. J. ..... Kansas City, Mo.
Dayton, G. O. ..... Los Angeles, Calif.
Decker, J. C. ..... Sioux City, Ia.
Edwards, Slocomb R. ..... El Centro, Calif.
Gale, M. Jean ..... Denver, Colo.
Guggenheim, L. K. ..... Los Angeles, Calif.
Guyton, B. S. ..... Oxford, Miss.
Halsted, Fred S. ..... Denver, Colo.
Haskell, Alfred W. ..... Portland, Me.
Heeb, Harry J. ..... Milwaukee, Wis.
Henton, George E. ..... Portland, Ore.
Hodsdon, B. F. ..... Miami, Fla.
Irvine, A. Ray ..... Beverly Hills, Calif.
Kistner, F. B. ..... Portland, Ore.
Kress, George H. ..... Los Angeles, Calif.
LaVigne, A. A. ..... New York, N. Y.
Lefler, Anna B. ..... Los Angeles, Calif.
Loomis, E. A. ..... Minneapolis, Minn.
Maiden, Sydner D. ..... Memphis, Tenn.
McDiarmid, H. Oliver ..... Brandon, Manitoba, Canada
McKeown, Elmer E. ..... Denver, Colo.
Minor, H. F. ..... Memphis, Tenn.
Mitchell, E. K. ..... Philadelphia, Pa.
Morris, A. G. ..... Rochester, N. Y.
Nelson, Louis A. ..... St. Paul, Minn.
Nisselson, Max ..... New York, N. Y.
O'Connor, D. F. ..... South Orange, N. J.
O'Connor, Roderic P. ..... Oakland, Calif.
Post, M. Hayward ..... St. Louis, Mo.
Prout, A. W. ..... Columbus, Ohio
Ralston, William W. ..... Houston, Texas
Reiche, O. C. ..... Hazleton, Pa.
Richie, L. T. ..... Trinidad, Colo.
Robertson, Edw. N. ..... Concordia, Kan.

Silbernagel, C. E. ..... Columbus, Ohio
Smith, D. S. ..... La Crosse, Wis.
Smith, H. A. ..... Whittier, Calif.
Spencer, W. H. ..... Philadelphia, Pa.
Sturges, Leigh F. ..... New York, N. Y.
Walker, C. E. ..... Colorado Springs, Colo.
Westlake, Samuel B. ..... St. Louis, Mo.

## Senior Fellows — 1948

Annon, W. T. ..... Philadelphia, Pa.
Blassingame, C. D. ..... Memphis, Tenn.
Bozer, Hermann E. ..... Buffalo, N. Y.
Bruner, Abram B. ..... Cleveland, Ohio
Clerf, Louis H. ..... Philadelphia, Pa.
Copps, Lyman A. ..... Marshfield, Wis.
Curtin, Eugene A. ..... Scranton, Pa.
Davis, Thomas W. ..... Winston-Salem, N. C.
Dean, Alfred ..... Grand Rapids, Mich.
Dorge, Richard I. ..... Minneapolis, Minn.
Gill, G. Bache ..... Washington, D. C.
Goldman, H. G. ..... New York, N. Y.
Harris, Robin ..... Jackson, Miss.
Hetrick, L. E. ..... New Rochelle, N. Y.
Howard, Lloyd G. ..... Council Bluffs, Ia.
Johnson, Thomas H. ..... New York, N. Y.
Lerner, Macy L. ..... Rochester, N. Y.
Lombardo, Melchiore ..... Brooklyn, N. Y.
Looper, Edward A. ..... Baltimore, Md.
Lyman, Harry W. ..... St. Louis, Mo.
Marshall, George G. ..... Rutland, Vt.
McKinney, A. R. ..... Saginaw, Mich.
Myerson, Mervin C. ..... Beverly Hills, Calif.
Parrish, Robert E. ..... San Antonio, Texas
Proetz, Arthur W. ..... St. Louis, Mo.
Rowland, W. D. ..... Boston, Mass.
Somberg, J. S. ..... Cleveland, Ohio
Tomassene, R. A. ..... Wheeling, W. Va.
Townsend, John F. ..... Charleston, S. C.
Tucker, Gabriel ..... Philadelphia, Pa.
Wendel, J. S. ..... Detroit, Mich.
Winter, John Arthur ..... Duluth, Minn.

## Examination by the American Board of Otolaryngology

The American Board of Otolaryngology will conduct an examination in Chicago, October 7, 8, 9 and 10, 1947. The completed application must be filed 120 days in advance of the examination at which the candidate desires to appear. For information, write to the Secretary, Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

## Examination by the American Orthoptic Council

The American Orthoptic Council will hold written examinations for technicians at various points throughout the country on September 11, 1947. Those who succeed in passing the written examination will be invited to come to Chicago for the practical and oral examinations on Saturday, October 11, 1947. The oral-practical examination will be held at Northwestern University Orthoptic Clinic. For further information write Dr. LeGrand H. Hardy, 23 E. 79th St., New York 21 N. Y.

## American Association of Eye, Ear, Nose, and Throat Society Secretaries

The eighth annual meeting of the American Association of Eye, Ear, Nose, and Throat Society Secretaries will be held during the convention of the American Academy on Wednesday evening, October 15 at 5:30 p.m. in Private Dining Room No. 18 at the Palmer House, Chicago. The Academy officials again have graciously offered to provide the dinner for this group of secretaries. Every secretary of an eye, or ear, nose and throat society is invited to attend. It is not required that you be a member of the Association. If you cannot be present please arrange for your president or another officer of your society to represent your group at this meeting.

In order to make the necessary arrangements, it is advisable that we know soon the approximate number to provide for. Will you please notify the undersigned as soon as possible if you will be present at this meeting. If you are unable to attend, will some other officer represent your society?

KENNETH L. CRAFT, M.D., *Secretary*
1002 Hume Mansur Bldg.
Indianapolis 4, Ind.

# CONVENTION ACTIVITIES

## PRESIDENT'S RECEPTION

**Grand Ball Room**
**Sunday, 4:30 p.m.**

President Alan C. Woods and Mrs. Woods invite all members and guests to a reception honoring Dr. Frederick H. Verhoeff and the officers of the Academy.

## ALUMNI DINNERS

**Monday, 6:30 p.m.**

### Dr. Francis Heed Adler Alumni

Palmer House
Private Dining Room No. 1
Francis Heed Adler, M.D., Secretary
313 South 17th St., Philadelphia 3, Pa.

### Dr. John F. Barnhill Alumni

Palmer House
Private Dining Rooms Nos. 15 and 16
C. H. McCaskey, M.D., Secretary
608 Guaranty Bldg., Indianapolis 4, Ind.

### Bellevue Alumni Association

Palmer House
Private Dining Room No. 4
W. W. Wilkerson, Jr., M.D., Secretary
1022 Bennie-Dillon Bldg., Nashville 3, Tenn.

### Brooklyn Eye and Ear Hospital Alumni

Palmer House
Private Dining Room No. 5
Leo J. Croll, M.D., Secretary
1326 Maccabees Bldg., Detroit 2, Mich.

### Cleveland Clinic Alumni—Otolaryngology

Stevens Hotel
Private Dining Room No. 1
Harold E. Harris, M.D., Secretary
Department of Otolaryngology, Cleveland Clinic, Cleveland 6, Ohio

### Cook County Hospital Alumni

Stevens Hotel
Paul V. Carelli, M.D., Secretary
3626 W. Chicago Ave., Chicago, Ill.

### Gill Memorial Hospital—Annual Spring Graduate Course

Stevens Hotel
Private Dining Room No. 2
Frank C. Furlong, M.D., Secretary
713 Union St., Schenectady, N. Y.

### Harper Hospital Alumni

Palmer House
Private Dining Room No. 3
Wesley G. Reid, M.D., Secretary
974 Fisher Bldg., Detroit 2, Mich.

### Illinois Eye and Ear Infirmary and University of Illinois Alumni Association

Palmer House
Private Dining Room No. 14
Earl S. McRoberts, M.D., Secretary
55 E. Washington St., Chicago, Ill.
and
Noah D. Fabricant, M.D., Secretary
185 N. Wabash Ave., Chicago, Ill.

### Iowa University Alumni

Palmer House
Private Dining Room No. 7
Byron M. Merkel, M.D., Secretary
1112 Equitable Bldg., Des Moines 9, Ia.

### Dr. John R. Lindsay Alumni

Stevens Hotel
Private Dining Room No. 3
M. M. Hipskind, M.D., Secretary
6060 Drexel Ave., Chicago, Ill.

### Manhattan Eye, Ear, and Throat Hospital Ex-House Surgeons

Location to be announced
at Registration Desk
W. H. Turnley, M.D., Secretary
1 Atlantic St., Stamford, Conn.

### Massachusetts Eye and Ear Infirmary Alumni

Stevens Hotel
Lorand V. Johnson, M.D., Secretary
Western Reserve University, Cleveland 6, Ohio

**Mayo Clinic and University of Minnesota Alumni**

Palmer House
Private Dining Room No. 18

Mark E. Nesbit, M.D., Secretary for Mayo Clinic
616 First Central Bldg., Madison, Wis.
George M. Tangen, M.D., Secretary for University of Minnesota
Medical Arts Bldg., Minneapolis 2, Minn.

**Michigan Joint Alumni**
(For former staff members only)

Palmer House
Private Dining Room No. 11

Walter Parker Club
Ralph O. Rychener, M.D., Secretary
1720 Exchange Bldg., Memphis 3, Tenn.

R. Bishop Canfield Society
James H. Maxwell, M.D., Secretary
First National Bank Bldg., Ann Arbor, Mich.

**Dr. Harris P. Mosher Alumni**

Palmer House
Private Dining Room No. 17

August L. Beck, M.D., Secretary
421 Huguenot St., New Rochelle, N. Y.

**New York Eye and Ear Infirmary Alumni**

Palmer House
Crystal Room

Brittain F. Payne, M.D., Secretary
17 E. 72nd St., New York 21, N. Y.

**New York Postgraduate Medical School and Hospital Alumni**

Palmer House
Private Dining Room No. 8

James W. Smith, M.D., Secretary
1016 Fifth Ave., New York 28, N. Y.

**Pennsylvania University and Wills Hospital Alumni—Ophthalmology**

Stevens Hotel
West Ballroom

George H. Stine, M.D., Secretary
305 Burns Bldg., Colorado Springs, Colo.

**Pennsylvania University Alumni—Otolaryngology**

Palmer House
Private Dining Room No. 9

Oram R. Kline, M.D., Secretary
514 Cooper St., Camden, N. J.

**Presbyterian Hospital Institute of Ophthalmology Alumni Association**

Palmer House
Private Dining Room No. 2

Arthur Gerard DeVoe, M.D., Secretary
635 W. 165th St., New York 32, N. Y.

**Washington University Alumni—Ophthalmology**

Bismarck Hotel
Reception—Lounge, 3rd floor
Dinner—Dining Room G

Richard G. Scobee, M. D., Secretary
Department of Ophthalmology, Washington University, St. Louis 10, Mo.

**Washington University Alumni—Otolaryngology**

Bismarck Hotel
Reception—Lounge, 3rd floor
Dinner—Mural Room

Arthur W. Proetz, M.D., President
Beaumont Bldg., St. Louis 8, Mo.

**Wilmer Institute John Hopkins Hospital**

Palmer House
Private Dining Room No. 6

William C. Owens, M.D., Secretary
Wilmer Institute, John Hopkins Hospital, Baltimore 5, Md.

---

**SMOKER**

**Grand Ball Room**
**Monday, 9:00 p.m.**

The ladies are invited. Entertainment and refreshments will be provided.

---

**ANNUAL ACADEMY DINNER**

**Grand Ball Room**
**Tuesday, 7:00 p.m.**
*Dress Optional*

### JACKSON MEMORIAL LECTURE

*Sponsored by the Ophthalmic Publishing Co.*

### WHERRY MEMORIAL LECTURE

*Sponsored by the Wherry Memorial Fund*

**ANNUAL BUSINESS MEETING**
*and*
**INDUCTION OF NEW MEMBERS**

**Grand Ball Room**

**Wednesday, 8:30 p.m.**

Dr. Francis Heed Adler will deliver the Jackson Memorial Lecture.

Dr. Robert Thompson Tidrick will deliver the Wherry Memorial Lecture. The title of his address will be *Recent Advances in Surgical Hemostasis: Evaluation of Absorbable Agents.*

A short business meeting will follow.

### FACULTY LUNCHEON

**Private Dining Room No. 14**

**Thursday, 12:45 p.m.**

President Alan C. Woods invites all those presenting scientific papers, motion pictures, scientific exhibits, and courses of instruction to be guests of the Academy at luncheon.

## SPECIAL SCIENTIFIC PROGRAMS

### CONSERVATION OF HEARING

**Grand Ball Room**

**Sunday, 8:00 p.m.**

DEAN M. LIERLE, M.D., *Chairman*

### AMERICAN ORTHOPTIC COUNCIL
*and*
### AMERICAN ASSOCIATION OF ORTHOPTIC TECHNICIANS

**Private Dining Room No. 14**

**Sunday, 8:00 p.m.**

LEGRAND H. HARDY, M.D., *Chairman*

1. Analysis of Strabismus Cases
   **William C. Owens, M.D., Baltimore, Md.**
2. Some Aspects of Binocular Vision
   **Kenneth C. Swan, M.D., Portland, Ore.**
3. Barriers and Aids to Fusion
   **Marianne Eyles, Seattle, Wash.**

### OCCUPATIONAL OPHTHALMOLOGY

**Grand Ball Room**

**Thursday, 8:00 p.m.**

A. D. RUEDEMANN, M.D., *Chairman*

HEDWIG S. KUHN, M.D., *Secretary*

1. Experiences in Industrial Eye Problems
   **David A. McCoy, M.D., Birmingham, Ala.**
2. Effect of Infra-red on Eyes
   **Charles F. Kutscher, M.D., Pittsburgh, Pa.**
3. Chemical Eye Injuries
   **Ralph S. McLaughlin, M.D., Charleston, W. Va.**

# PROGRAM IN BRIEF

## 1947 Meeting — Chicago

SATURDAY, OCTOBER 11, 1947

9:00 a.m.—Council Meeting — Private Dining Room No. 4

SUNDAY, OCTOBER 12, 1947

9:00 a.m.—Registration — Foyer to Grand Ball Room

9:00 a.m.—Official Opening of Exhibits
Scientific — Red Lacquer Room
Technical—Exhibition Hall

10:00 a.m.—Ladies' Registration—Club Floor Rooms 15 and 16

10:00 a.m.—Meeting of Joint Committee on Occupational Ophthalmology—Dr. Kuhn's Suite

10:00 a.m.—Meeting of Executive Committee of the Committee on Conservation of Hearing—Rooms 795 and 796

10:00 a.m.—Meeting of American Committee on Optics and Visual Physiology—Private Dining Room No. 1

12:30 p.m.—Luncheon for Home Study Faculty — Private Dining Room No. 9

2:00 p.m.—Meeting of Advisory Committee to the Registries of Pathology—Private Dining Room No. 8

4:30 p.m.—President's Reception — Grand Ball Room

8:00 p.m.—Special Scientific Program—Conservation of Hearing—Grand Ball Room

8:00 p.m.—Special Scientific Program—American Orthoptic Council and American Association of Orthoptic Technicians — Private Dining Room No. 14

MONDAY, OCTOBER 13, 1947

9:00 a.m.—Joint Scientific Session—Grand Ball Room

2:00 p.m.—Section on Otolaryngology—Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—Instruction Courses—EYE—Seventh Floor

3:15 p.m.—Instruction Courses—EYE—Seventh Floor

4:30 p.m.—Instruction Courses — EYE Seventh Floor

6:30 p.m.—Alumni Dinners

9:00 p.m.—Smoker—Grand Ball Room

TUESDAY, OCTOBER 14, 1947

9:00 a.m.—Section on Ophthalmology—Scientific Papers and Motion Pictures—Grand Ball Room

9:00 a.m.—Instruction Courses — ENT—Seventh Floor

10:15 a.m.—Instruction Courses — ENT—Seventh Floor

11:30 a.m.—Instruction Courses — ENT—Seventh Floor

12:45 p.m.—Technical Exhibitors' Luncheon—Crystal Room

2:00 p.m.—Section on Otolaryngology—Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—Instruction Courses — EYE—Seventh Floor

3:15 p.m.—Instruction Courses — EYE—Seventh Floor

4:30 p.m.—Instruction Courses — EYE—Seventh Floor

7:00 p.m.—Annual Dinner—Grand Ball Room

WEDNESDAY, OCTOBER 15, 1947

9:00 a.m.—Section on Ophthalmology—Scientific Papers and Motion Pictures—Grand Ball Room

9:00 a.m.—Instruction Courses — ENT—Seventh Floor

10:15 a.m.—Instruction Courses — ENT—Seventh Floor

11:30 a.m.—Instruction Courses — ENT—Seventh Floor

12:45 p.m.—Council Meeting and Luncheon—Private Dining Room No. 5

2:00 p.m.—Section on Otolaryngology—Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—Instruction Courses — EYE Seventh Floor

3:15 p.m.—Instruction Courses — EYE Seventh Floor
4:30 p.m.—Instruction Courses — EYE Seventh Floor
5:30 p.m.—Dinner Meeting of American Association of Eye, Ear, Nose and Throat Society Secretaries — Private Dining Room No. 18
8:30 p.m.—Jackson Memorial Lecture, Wherry Memorial Lecture, Business Meeting — Grand Ball Room

THURSDAY, OCTOBER 16, 1947

9:00 a.m.—Section on Ophthalmology —Scientific Papers and Motion Pictures—Grand Ball Room
9:00 a.m.—Instruction Courses — ENT Seventh Floor
10:15 a.m.—Instruction Courses — ENT Seventh Floor
11:30 a.m.—Instruction Courses — ENT Seventh Floor
12:45 p.m.—Faculty Luncheon—Private Dining Room No. 14
2:00 p.m.—Section on Otolaryngology —Scientific Papers and Motion Pictures—Grand Ball Room
2:00 p.m.—Instruction Courses — EYE Seventh Floor
3:15 p.m.—Instruction Courses — EYE Seventh Floor
4:30 p.m.—Instruction Courses — EYE Seventh Floor
8:00 p.m.—Special Scientific Program — Occupational Ophthalmology—Grand Ball Room

FRIDAY, OCTOBER 17, 1947

9:00 a.m.—Section on Ophthalmology —Scientific Papers and Motion Pictures — Grand Ball Room
9:00 a.m.—Instruction Courses — ENT Seventh Floor
10:15 a.m.—Instruction Courses — ENT Seventh Floor
11:30 a.m.—Instruction Courses — ENT Seventh Floor

## THE HOTEL SITUATION

For many years, the American Academy of Ophthalmology and Otolaryngology has been most fortunate in having the complete co-operation of the Chicago hotels in aiding Fellows to procure rooms during the convention. It has been the policy for centrally located hotels to earmark a certain percentage of their accommodations for the use of physicians requesting reservations and announcing their intentions of attending our convention. The same policy was carried out this year with even a greater number of rooms guaranteed us by the loop hotels. However, all the rooms in Chicago hotels, earmarked for Academy Fellows, have now been allotted, and no rooms are available at the Palmer House. Rooms are available at other hotels. Those who have not yet made reservations will be more successful in securing accommodations, if, when requesting rooms, they do not designate their Academy affiliations.

# SCIENTIFIC SECTIONS

## JOINT SESSION

**Monday, October 13, 1947**
**9:00 a.m.**

**GRAND BALL ROOM**

*Presiding Officer:*
ALAN C. WOODS, M.D.
*President*

*Secretary in Charge:*
ALGERNON B. REESE, M.D.

Call to Order.....The President

Announcements, the General Chairman
William A. Mann, M.D., Chicago, Ill.

Introduction of Guest of Honor

Address of the President

*An Examination of the Present Policies of the Specialty Board and the National Societies in Relation to the Progress of Ophthalmology*

**Alan C. Woods, M.D., Baltimore, Md.**

Address of the Guest of Honor

*Some Problems Concerning Convergence*

**Frederick H. Verhoeff, M.D., Boston, Mass.**

Symposium: PSYCHOSOMATIC MANIFESTATIONS

*General Aspects*
**Thomas A. C. Rennie, M.D., New York, N. Y.** BY INVITATION

*Otolaryngology*
**C. Stewart Nash, M.D., Rochester, N. Y.**

*Ophthalmology*
**David O. Harrington, M.D. San Francisco, Calif.**
BY INVITATION

## SECTION ON OTOLARYNGOLOGY

**Monday, October 13, 1947**
**2:00 p.m.**

**GRAND BALL ROOM**

*Presiding Officer:*
ARTHUR C. JONES, M.D.
*First Vice President*

*Secretary:*
O. E. VAN ALYEA, M.D.

### DEMONSTRATION OF NEW INSTRUMENTS AND NEW METHODS

**2:05 p.m.**

**THE CONTROL OF BLEEDING DURING THE FENESTRATION OPERATION BY INDUCED HYPOTENSION (IRVING PAGE PROCEDURE)**

**Harold E. Harris, M.D.**
**Donald E. Hale, M.D.**
BY INVITATION
**Cleveland, Ohio**

Discussers:

James H. Maxwell, M.D.
Ann Arbor, Mich.
Howard House, M.D.
Los Angeles, Calif.

It has been found that at least 30 to 40 per cent of the patients that undergo the fenestration operation have an in-

### Authors of Papers

Please be prepared to submit papers to the recorder immediately following presentation.

There will be discussion of papers presented at the scientific sections Monday afternoon through Friday morning.

creased tendency to bleed during that procedure. Any method that will reduce the amount of blood entering the fenestrum during its preparation is a distinct advantage. There is less postoperative labyrinthitis, and the percentage of good results can thereby be increased.

The Page Procedure has been found to be effective in controlling this troublesome bleeding, and also, in the hands of properly trained personnel, it is a safe procedure.

---

**2:45 p.m.**

**LEMPERT FENESTRA NOV-OVALIS OPERATION FOR THE RESTORATION OF PRACTICAL AND SERVICEABLE UNAIDED HEARING**

**(A Motion Picture)**

**Julius Lempert, M. D.**
**New York, N. Y.**

---

The motion picture depicts each step in the technic of the fenestration operation in its complete detail. The three endaural incisions are demonstrated. The exenteration of the mastoid semicircular canals is demonstrated. The exposure of the incudomalleolar joint including the removal of the incus and head and neck of the malleus are clearly shown. The preparation and fitting of the tympanomeatal membrane which is destined to cover the fenestra nov-ovalis is clearly depicted. The creation of the fenestra nov-ovalis within the surgical dome of the vestibule is shown in its various stages of creation. The burnishing of the fenestra rim of the newly created fenestra with pure lead for the purpose of preventing osteogenesis is demonstrated. The burnishing with pure lead has resulted in the prevention of osteogenesis in the Rhesus monkey and it is with the hope that it will do the same in the human that lead burnishing of the fenestra is now conclusively employed. The covering and sealing of the fenestra with the tympanomeatal membrane is shown. The postoperative mastoid cavity is skin-grafted and the dressing of the wound and the anchoring of the tympanomeatal membrane into proper position with paraffin mesh gauze is demonstrated. The appearance of the patient's ear three weeks postoperative is shown.

**3:30 p.m.**

**SOME EXPERIMENTS WITH CALORIC STIMULATION OF THE HUMAN LABYRINTH TO DETERMINE THE VALIDIDITY OF EWALD'S LAWS**

**W. J. McNally, M.D.**
**E. A. Stuart, M.D.**
BY INVITATION
**J. S. Jamieson, M.D.**
BY INVITATION
**Gordon Gaulton, M.D.**
BY INVITATION
**Montreal, Canada**

Discussers:

J. R. Lindsay, M.D.
Chicago, Ill.
Edward Walzl, M.D.
BY INVITATION
Baltimore, Md.

---

**4:10 p.m.**

**ORGANIC DISORDERS OF THE LARYNX**

**(A Motion Picture)**

**Paul H. Holinger, M.D.**
**Albert H. Andrews, Jr., M.D.**
**George C. Anison, M.D.**
BY INVITATION
**Chicago, Ill.**

---

This film has been prepared to illustrate the organic disorders of the larynx as visualized with the laryngeal mirror and the direct laryngoscope. Special emphasis is placed on the manner in which various lesions affect sound production. Inflammatory, neurologic and neoplastic lesions are shown as they influence the physiologic processes of phonation.

---

**4:40 p.m.**

**ORAL LESIONS**

**Russell A. Sage, M.D.**
**Indianapolis, Ind.**

Discussers:

Francis Senear, M.D.
BY INVITATION
Chicago, Ill.
C. H. McCaskey, M.D.
Indianapolis, Ind.

---

A slide presentation and description of interesting oral lesions.

## SECTION ON OPHTHALMOLOGY

### Tuesday, October 14, 1947 9:00 a.m.

**GRAND BALL ROOM**

*Presiding Officer:*
PHILLIPS THYGESON, M.D.
*Second Vice President*

*Secretary:*
ALGERNON B. REESE, M.D.

**SYMPOSIUM: CORNEAL TRANSPLANTATION**

**9:00 a.m.**

*Pathology*

**Alfred E. Maumenee, M.D.**
**Walter Kornbluth, M.D.**
BY INVITATION
**Baltimore, Md.**

**9:30 a.m.**

*Indications*

**R. Townley Paton, M.D.**
**New York, N. Y.**

**10:00 a.m.**

*Technic*

**John M. McLean, M.D.**
**New York, N. Y.**

**10:30 a.m.**

*Complications*

**Ramon Castroviejo, M.D.**
**New York, N. Y.**

**11:00 a.m.**

*Results*

**William Councilman Owens, M.D.**
**The Wilmer Institute**
**Baltimore, Md.**
*In collaboration with*
Joseph J. Frank, M.D.
Manhattan Eye and Ear Infirmary
New York, N. Y.

Brendan Leahey, M.D.
Massachusetts Eye and Ear Infirmary
Boston, Mass.

Paul E. Messier, M.D.
New York Hospital
New York, N. Y.

Harold G. Scheie, M.D.
Hospital of the University of Pennsylvania
Philadelphia, Pa.

B. Richard Vincent, M.D.
New York Eye and Ear Infirmary
New York, N. Y.

Joseph A. C. Wadsworth, M.D. and Frederick C. Stansbury, M.D.
The Eye Institute, Presbyterian Hospital
New York, N. Y.

## SECTION ON OTOLARYNGOLOGY

### Tuesday, October 14, 1947 2:00 p.m.

**GRAND BALL ROOM**

*Presiding Officer:*
NORTON CANFIELD, M.D.
*Third Vice President*

*Secretary:*
O. E. VAN ALYEA, M.D.

**DEMONSTRATION OF NEW INSTRUMENTS AND NEW METHODS**

**2:15 p.m.**

**CLINIC ON DEAFNESS**
**(A Motion Picture)**
**Samuel J. Kopetzky, M.D.**
**Ralph Almour, M.D.**
**New York, N. Y.**

The differentiation of types of deafness is becoming the paramount question of the moment. This needs solution not only to indicate the approach to therapy, but particularly now, when surgical therapy is having a vogue. Therapy needs stress, especially in the preoperative stages, so that maximal recovery of hearing acuity attainable before operation is achieved. The film is not a description of surgical technic, although this is touched upon in passing. It comprises a clinic of types of deafness and discusses the therapeutical approach, based upon the differentiation of the cases. It is less a clinic of how to treat deafness, as it is a

discussion of means to treat the patient for what is disabling him, and causing, among other things, his deafness.

The film presents graphically the prevalent theories of hearing. It portrays diagnostic determination. Audiometry is explained, and a sketch of both medical and surgical therapy is presented.

The factors are shown by means of animations and drawings, supplementing clinical material. The film is in color and sound.

---

**2:45 p.m.**

## THE HISTORY OF THE DEVELOPMENT OF LARYNGOLOGY

**Edwin N. Broyles, M.D.**
BY INVITATION
**Baltimore, Md.**

Discussers:

Louis H. Clerf, M.D.
Philadelphia, Pa.

Lyman Richards, M.D.
Brookline, Mass.

---

Before the discovery of the indirect method of laryngoscopy by Garcia in 1854, treatment of laryngeal diseases was symptomatic, though a few courageous surgeons were able in a very small number of cases to remove benign growths by thyrotomy or by guiding a curved blade into the larynx by palpation.

Following Garcia's discovery, Austrian physicians were quick to appreciate its great aid in the diagnosis and treatment of laryngeal diseases.

Ludwig Turck in 1866 published his textbook on "The Diseases of the Larynx," and must be considered the founder of laryngology.

Two Americans were prominent in the pre-laryngoscopic period for their works on laryngeal diseases: Horace Green for his intralaryngeal medication and Gurdon Buck for his intralaryngeal surgery in edema of the larynx.

The first laryngeal society in any part of the world was founded in New York City by Clinton Wagner in 1873.

The next great step in laryngoscopy was the development of direct view of the larynx by Czermack and Killian.

A general discussion of the most publicized case of cancer of the larynx along with other developments in the treatment of some diseases of the larynx.

---

**3:25 p.m.**

## DISEASES OF THE EAR, NOSE AND THROAT

**(A Motion Picture)**

**Paul H. Holinger, M.D.**
**Albert H. Andrews, Jr., M.D.**
**George C. Anison, M.D.**
BY INVITATION
**Chicago, Ill.**

---

An entirely new endoscopic motion picture camera has been constructed with which this film has been prepared. The film shows pictures of the camera and gives a brief discussion of its essential features. A review of the normal endoscopic anatomy and some of the more common types of pathology visualized by the otorhinolaryngologist make up the body of the film. The ear drum, anterior nasal chamber and postnasal space, pharynx, larynx, tracheobronchial tree and esophagus are shown. The new camera permits better color rendition, larger image on the screen, and greater depth of focus than the previous camera used for endoscopic photography.

---

**3:55 p.m.**

## PENICILLIN AEROSOL

**David Davis, M.D.**
**Washington, D. C.**

Discussers:

Louis R. Krasno, M.D.

Elmer Hagens, M.D.
Chicago, Ill.

---

Penicillin aerosol, in treatment of upper and lower respiratory conditions, has been followed extensively in office practice. Better results are attained by using a combination of penicillin and streptomycin in propylene glycol. A method is being devised for inhalation of iodized oil for bronchography.

**4:35 p.m.**

### ANATOMY OF THE EAR, NOSE AND THROAT

**(Three dimensional photography)**

**Irwin G. Spiesman, M.D.**
**Maywood, Ill.**

---

The photography to be presented is one of special interest as a teaching technic in the field of ear, nose and throat. The anatomatical material employed for this photography comes from the Department of Ear, Nose and Throat of the University of Illinois.

The three dimension method employed here in the presentation of this subject matter permits the showing of pictures of depth and clarity wth such visual realism as has not heretofore been possible even at the dissecting table where, at most, two or three students can participate. The pictures are magnified and lend themselves to view by large audiences with such astounding perception as to leave an indelible impression.

In every instance when a three dimension picture is presented, two images are actually seen. One of these is being viewed with the right and the other with the left eye. The picture is taken as an ordinary photograph except that instead of one view, two are taken from positions lateral to each other, in such a manner as to correspond to the right and left eye views.

This new method of projecting three dimension pictures has been devised within the last ten years. It is based on the use of a product known as Polaroid which is a layer of film, molecular in structure, laminated to acetate or glass that has the property of polarizing light that passes through it. Polarized light differs from ordinary light in that its plane of vibration is only in one direction whereas ordinary light vibrates in all planes.

It will be demonstrated that the modern tendency to use visual aids in teaching is most vividly and effectively met by the use of the three dimension photography.

## SECTION ON OPHTHALMOLOGY

**Wednesday, October 15, 1947**
**9:00 a.m.**

**GRAND BALL ROOM**

*Presiding Officer:*
ALAN C. WOODS, M.D.
*President*

*Secretary:*
ALGERNON B. REESE, M.D.

---

### THE ROLE OF INHERITANCE IN GLAUCOMA

**Adolph Posner, M.D.**
**Abraham Schlossman, M.D.**
BY INVITATION
**New York, N. Y**

Discussers:

Peter C. Kronfeld, M.D.
Chicago, Ill.

Harold F. Falls, M.D.
Ann Arbor, Mich.

---

In the course of a critical survey of 390 unselected cases of primary glaucoma from the joint practices of the late Dr. Mark J. Schoenberg and one of us (Adolph Posner), we were impressed by the fact that 10 per cent showed familial tendencies.

A review of the literature reveals a total of 84 published family trees of primary glaucoma. Only ten of these have appeared in American journals. From the fact that the published reports deal with isolated instances of large pedigrees, it may be deduced that the profession has hitherto focussed its attention on the relatively few cases which lend themselves to such exhaustive study.

The purpose of this paper is to show that hereditary tendencies are present in a large percentage of glaucoma cases seen in any routine practice. Furthermore, we feel that a knowledge of the behavior of glaucoma in one member of the family may be of aid in the prognosis and treatment of another member.

In 13 families we were able to follow the course of the disease in two or more relatives for four to twenty-five years; and in eight more families, only one member of the family has been under observation long enough to warrant inclusion in this study.

Correlations are made regarding the various phases of the disease as exhibited by different members of the same family. The following factors are stressed:

1. Age of onset
2. Type of glaucoma
3. Response to miotics and surgery
4. Refractive errors
5. Reaction to provocative tests
6. Depth of anterior chamber and width of angle
7. Fundus changes
8. Field changes
9. Pupillographic studies
10. Associated diseases

Tables in parallel columns will be presented to illustrate graphically these correlations. Tension curves and field studies followed up for many years will also be shown.

Pedigrees of these families are being prepared and will be subjected to careful analysis in order to determine the mechanisms of inheritance.

Note: A preliminary report on some pupillographic studies will be included to demonstrate its value in detecting the tendency toward glaucoma in the unaffected members of a family.

---

**9:40 a.m.**

## TWO CORNEAL TRANSPLANTATIONS FROM ONE DONOR EYE
**(A Motion Picture)**

**George P. Landegger, M.D.**
**Los Angeles, Calif.**

The film depicts excision of a rectangular piece of cornea from the donor eye which is divided in half, as it was devised by Dr. Castroviejo. The two squares which are formed thus are transplanted into the eyes of two young men who had an advanced keratoconus and were beyond the stage of wearing contact lenses. Each graft is held in place with a suture which forms a cross. Air is injected immediately after surgery to reform the anterior chamber. The final results are also shown on the boys in close-up. The grafts have healed perfectly clear and each patient sees 20/20 now, eleven months after surgery.

---

**10:00 a.m.**

## RECONSTRUCTION OF THE CANTHI

**Alston Callahan, M.D.**
**Birmingham, Ala.**

Discussers:

Wendell L. Hughes, M.D.
Hempstead, N. Y.
Sidney A. Fox, M.D.
New York, N. Y.

---

Severe injuries to the palpebral ligaments, often complicated by fractures of the orbital rim, are responsible for major deformities and displacements of the canthi and avulsion of the eyelids. Penetrating lacerations, which may sever the ligaments, occur frequently in automobile accidents as well as in combat injuries. The eyeball may suffer irreparable damage and the socket may be distorted. The major concern in the immediate repair of such conditions is reattachment of the severed ends of the ligament or the tarsi with the ligament. Simple closure of the skin when a detached ligament exists will not prevent marked shortening of the lids and retraction of the canthi.

Our experience has been derived from 36 patients, most of whom were injured in military service and were not seen by us until two to four months after the initial trauma. Therefore, we did not render immediate care, but it was our responsibility to repair them at a later stage.

The anatomical relationships of the medial and lateral palpebral ligaments are considered. Horner's muscle exerts a posteriorly directed force on the medial palpebral ligament and both the palpebral raphe and lateral palpebral ligament maintain the lateral canthus. The influence of these various structures must be kept in mind when reconstructive procedures are performed.

We were unsuccessful in achieving the desired improvement by those methods in general use, and so utilized bone for support rather than periosteum, and wire of an inert nature in preference to sutures. Stainless steel wire 0.009" was found most useful. The technic of its insertion through the bony openings is shown.

Local anesthesia was used for all of these cases. Procaine hydrochloride (2%) with epinephrine hydrochloride (1 :100,000) was injected freely into the operative site. In cases complicated by extensive cicatrization, morphine sulphate (gr. ¼) was administered intravenously as the operation was begun.

Eight cases have been selected which show typical deformities from injuries of both medial and lateral canthi. The preoperative and postoperative appearances are shown by Kodachrome lantern slides, and drawings are used to show the method of correction employed.

---

**10:40 a.m.**

## ONCHOCERCIASIS IN GUATEMALA (A Motion Picture)

**William B. Clark, M.D.**
**New Orleans, La.**

---

Onchocerciasis, a filarial disease caused by infection with the nematode, **Onchocerca volvulus**, is manifested clinically by nodules in the subcutaneous or connective tissue and by various pathologic changes in the eye which may terminate in blindness. Man, the true host of this parasite, harbors the adult worm, the ova, and the microfilarial stage. The larval stage occurs in the intermediate host, the Simulium fly (coffee fly); larvae are deposited on the skin as the fly bites the human host.

The disease is found only in Africa and in Guatemala and southern Mexico, where it is limited to an area located behind or on the Pacific side of a high range of mountains, over which the vector cannot fly. In this region all the environmental factors, including the altitude, the abundance of fast mountain streams, the heavy rainfall, and the method of growing coffee in the shade, make the fincas (coffee plantations) likely places for the infection of native workers.

The study of ocular onchocerciasis reported herewith was made under the auspices of the Pan-American Sanitary Bureau, in co-operation with the Sanidad Publica de Guatemala, in Yepocapa, a small town on the periphery of one of the larger zones of infection in Guatemala, where these agencies maintain a laboratory and are studying the life cycle of **O. volvulus** and its intermediate host under controlled conditions.

The investigation covered 1,215 patients known to be infected with **O. volvulus.** Skin biopsy and a complete ophthalmologic examination were carried out in each case. In all, 534 patients, 44 per cent of the total, had ocular lesions apparently due to this disease. Pathologic changes included keratitis, iritis, and characteristic lesions of the fundus (believed to be degenerative and not inflammatory) consisting of optic nerve atrophy, choroidal sclerosis, and irregular deposits of pigment. Twenty-three patients were completely blind. An interesting phase of the investigation was the removal of live, motile microfilariae from the anterior chamber of eyes which otherwise seemed practically normal.

The demonstration will include illustrations of the environment in which infection occurs, of **O. volvulus** at various phases of the life cycle, and of degenerative lesions of the fundus believed to be typical of ocular onchocerciasis.

---

**11:00 a.m.**

## EXPERIENCES WITH TUMORS OF THE LIMBUS

**Kenneth C. Swan, M.D.**
**Thomas H. Emmens, M.D.**
BY INVITATION
**Leonard Christensen, M.D.**
BY INVITATION
**Portland, Ore.**

Discussers:

Phillips Thygeson, M.D.
San Jose, Calif.

John S. McGavic, M.D.
Bryn Mawr, Pa.

---

Tumors are common in the limbic region and present a diversity of important diagnostic and therapeutic problems. Experiences with various types of limbic tumors are summarized with particular reference to differential diagnosis and

changing concepts of prognosis. Varied methods of treatment are presented and discussed.

---

**11:40 a.m.**

### INTRACAPSULAR CATARACT SURGERY

**(A Motion Picture)**

**Daniel B. Kirby, M.D.**
**New York, N. Y.**

---

The system of intracapsular extraction of cataract.

I Indirect Rupture of the Zonule

1. Point pressure at six, eight and four o'clock inside the corneal limbus.
2. Application of forceps above; combination of simultaneous pressure directly opposite point of traction.
3. Rotation.

II Direct Rupture of the Zonule

4. Separation or stripping of the zonule from the capsule in cases of resistant zonule.

The understanding of the exaggeration of the progress of the operation of the speed and motion, size and apparent trauma by the magnification of the projected film is solicited.

Lantern slides to preview details of action in the motion picture film.

---

## SECTION ON OTOLARYNGOLOGY

**Wednesday, October 15, 1947**
**2:00 p.m.**

**GRAND BALL ROOM**

*Presiding Officer:*
ARTHUR C. JONES, M.D.
*First Vice President*

*Secretary:*
O. E. VAN ALYEA, M.D.

### DEMONSTRATION OF NEW INSTRUMENTS AND NEW METHODS

---

**2:10 p.m.**

### CRANIAL AUTONOMIC PATHWAYS

**David R. Higbee, M.D.**
**San Diego, Calif.**

Discussers:

Edwin N. Kime, M.D.
BY INVITATION
Bloomington, Ind.

A. W. Proetz
St. Louis, Mo.

---

The autonomic nervous system has definite anatomical and physiological characteristics. It is the mechanism through which many symptoms and signs of disease are recognized. Anatomists and physiologists define it as an efferent or motor system distributed to glandular tissue and unstriated muscle. In the practice of otolaryngology, our diagnoses and therapy indicate that both sensory and motor functions are attributed to the autonomic system. Nerve fiber tracts have specific functions—they transmit stimuli in only one direction and cannot be both sensory and motor.

A review of anatomy and physiology will suggest a revision of our ideas and associate symptoms with the nerve tracts through which they are mediated.

There will be a report on anatomical dissections of the vidian nerve. Descriptions of the nerve in text books do not point out the complete independence of the component nerves. The vidian nerve is composed of two nerves which are anatomically distinct and have no functional relation in the pterygopalatine canal. Both are visceral motor fiber tracts and have no function in relation to pain.

---

**2:50 p.m.**

### EXTERNAL FRONTAL ETHMOIDAL OPERATION

**(A Motion Picture)**

**J. Swift Hanley, M.D.**
**New York, N. Y.**

---

Radical frontal sinus operation.
Original incision is made above hairline of brow.
Trough is cut in the bone with V-chisel opening the frontal sinus.

Probe is passed into sinus to determine its extent in all directions.

X-ray shows extent of large frontal sinus on right side, small one on left.

Supra-orbital ethmoids extending across the roof of the orbit to the zygoma.

Right frontal sinus is outlined.

The outer table over the sinus is entirely removed.

This right frontal sinus extends well past the midline and the incision is lengthened.

The thinness of the normal membrane which lines the sinus is demonstrated.

The supra-orbital ridge is thinned and smoothed down.

The shelf of the outer table is smoothed down to the plane of the inner table.

Looking under the supra-orbital ridge showing three supra-orbital ethmoid cells extending into the floor of the frontal.

This cell is partially uncapped.

The probe enters the nasofrontal duct.

The four supra-orbital ethmoid cells are outlined. They drain by separate ducts into the nose.

The bone which overlies the most temporal of the supra-orbital ethmoid cells must be completely removed.

The supra-orbital ethmoid cells are removed.

The probe first enters the nasofrontal duct and then passes into the entirely separate opening into the nose from the ethmoid cells.

The septa between the ethmoid cells are removed.

The roof of the orbit is removed leaving the periosteum intact.

The orbital contents will fill in the space of the supra-orbital ethmoid cells as shown here by pressure over the eyeball.

The supra-orbital ridge is thinned down.

The separate external ethmoid incision is made.

The lacrimal sac is carefully displaced from its fossa.

The lacrimal fossa is outlined.

The ethmoid labyrinth is exenterated.

The tendon of the superior oblique is shown passing through the trochlea and spreading out for its attachment for the eye.

The probe enters the nasofrontal duct from above and the portion of bone to be removed is shown.

The middle turbinate is removed.

Looking down through the ethmoidal space into opening in the anterior wall of the sphenoid. Its walls are outlined.

The large cigarette drain is passed down and emerges through the external nares.

The ethmoid incision is closed using a rubber dam drain.

The frontal incision is closed with a rubber dam drain temporally.

The "postage stamp" pressure dressing to completely obliterate the cavity is applied and bandaged tightly in place.

---

**3:20 p.m.**

## CONGENITAL CHOANAL OCCLUSION

**W. W. Wilkerson, Jr., M.D.**
**Nashville, Tenn.**

Discussers:

Lawrence Boies, M.D.
Minneapolis, Minn.

J. J. Shea, M.D.
Memphis, Tenn.

---

The paper covers the surgical anatomy, symptoms and methods of diagnosis Various types of surgical procedures and simplification of technic are summarized, as well as general surgical considerations. Occlusion of the choanae in the infant and adult are presented separately due to the widely varying pictures.

Six case reports are given: Three of sisters, eleven to eighteen years of age, with bilateral bony choanal occlusion; a day-old infant whose bilaterally occlud-

ing wall consisted of bone and cartilage; and two teen-age girls with unilateral bony occlusion, one of which had a very interesting congenital anomaly in the region of the sphenoid. This anomaly will be discussed in detail.

**4:00 p.m.**

**NEW METHOD OF SCIENTIFIC INVESTIGATION OF THE EAR**
**(A Motion Picture)**

**H. G. Kobrak, M.D.**
**Chicago, Ill.**

This film is a cinematographic recording of the middle and inner ear during the process of hearing. A method was developed which permits quantitative estimates of the amplitudes. Observations were made on ear-normal persons, patients, experimental animals and fresh human cadaver specimens. A normal ear drum is shown vibrating due to sound. The ossicular chain and its lever action is demonstrated. The movements of the stapes due to contractions of the stapedius muscle are shown on patients who had a radical mastoid operation. Pathologic vibrations in a case of adhesions in the middle ear are shown. The acoustic vibrations of the stapes and round window membrane are shown and their opposite phase is demonstrated. Acoustic movements of the intralabyrinthine fluid are made visible. The response of the ear to music is demonstrated.

**4:15 p.m.**

**PHYSIOLOGY OF THE NOSE; SURGICAL CONSIDERATIONS**

**Dean Lierle, M.D.**
Iowa City, Iowa

Discussers:

Anderson Hilding, M.D.
Duluth, Minn.

T. C. Galloway, M.D.
Evanston, Ill.

**4:55 p.m.**

**LARYNGO-PHARYNGECTOMY FOR PHARYNGEAL CARCINOMA**
**(A Motion Picture)**

**Julius W. McCall, M.D.**
**Cleveland, Ohio**

A long broad skin flap is raised from the neck which is utilized for reconstruction of the pharynx and upper esophagus. After removal of the larynx and diseased portion of the pharynx, the skin flap is sutured above to the remaining portion of the pharynx and below to the severed esophagus. The denuded area of the neck is covered by Thiersch grafts taken from the leg.

# SECTION ON OPHTHALMOLOGY

## Thursday, October 16, 1947

## 9:00 a.m.

**GRAND BALL ROOM**

*Presiding Officer:*
PHILLIPS THYGESON, M.D.
*Second Vice President*

*Secretary:*
ALGERNON B. REESE, M.D.

**THE GROWTH OF EPITHELIUM INTO THE ANTERIOR CHAMBER FOLLOWING CATARACT EXTRACTION**

**Georgiana D. Theobald, M.D.**
**Oak Park, Ill.**
**Joseph S. Haas, M.D.**
BY INVITATION
**Chicago, Ill.**

Discussers:

Edmund B. Spaeth, M.D.
Philadelphia, Pa.

Benjamin Rones, M.D.
Washington, D. C.

At the Illinois Eye and Ear Infirmary during an eleven year period (1934 through 1944), 11 cases of epithelization of the anterior chamber occurred after 8,462 cataract extractions.

The histories are studied to determine method of operation, site of incision and length of time elapsing before enucleation.

The enucleated eyes were sectioned serially and studied to find the manner in which epithelium entered the anterior chamber.

The literature will be reviewed.

**9:40 a.m.**

## TEMPORAL TRANSCONJUNCTIVAL APPROACH FOR THE REMOVAL OF HEMANGIOMA OF THE ORBIT
**(A Motion Picture)**

**F. Bruce Fralick, M.D.**
**Ann Arbor, Mich.**

The transconjunctival approach for the removal of orbital tumors does not supplant other operations for this purpose but is a very convenient one for those far forward in the orbit either within or without the muscle cone. It is not applicable to large tumors since there is a maximum of 11 mm. horizontally and 10.7 mm. vertically between the orbital rim and the globe through which the mass may be removed intact. For tumors suspected of being larger than this the Krönlein approach is advisable.

The moving picture demonstrates the temporal transconjunctival approach to a hemangioma lying in the muscle cone. When so situated it is necessary to remove the lateral rectus muscle and retract out of the way so it cannot be injured. An adequate conjunctival incision is necessary to give free access to the orbit. The addition of a free external canthotomy makes the operation much easier and in no way complicates the outcome.

---

**10:00 a.m.**

## TECHNIC OF GONIOTOMY FOR CONGENITAL GLAUCOMA

**Otto Barkan, M.D.**
**San Francisco, Calif.**

Discussers:

Frederick C. Cordes, M.D.
San Francisco, Calif.

Thomas D. Allen, M.D.
Chicago, Ill.

It has been demonstrated that the cause of increased pressure in congenital glaucoma is an obstruction of the angle of the anterior chamber by residual embryonic tissue. Pre- and postoperative gonioscopy shows that stripping or peeling this tissue from the angle in from one-quarter to one-half of the circumference results in permanent normalization of the intraocular pressure. The operation is performed by direct vision under a surgical contact glass which has been specially devised for this purpose. If cloudiness of the cornea prevents the use of the glass the operation is performed without it. The principal technics are given in detail. Alternative technics are discussed.

---

**10:40 a.m.**

## FASCIA LATA SUSPENSION FOR THE CORRECTION OF COMPLETE PTOSIS
**(A Motion Picture)**

**Lorand V. Johnson, M.D.**
**Cleveland, Ohio**

The operation for correction of complete ptosis, employing fascia lata to suspend the lid border, has been frequently modified since the original description by Lancaster and Derby. Especially is it valuable when there occurs complete third nerve paralysis.

The firm union of the fascia lata to the periosteum above the brow, and the lack of adhesions (with resultant puckering of the skin) allows the creation of a nice lid fold.

Probably the greatest advantage of the periosteal attachment of the continuous fascia lata strip is the mechanical advantage gained, since, if the brow is elevated one millimeter by the frontalis, the lid border is elevated two millimeters.

It is advised that the lid be corrected to a completely open position, since these patients in six or eight weeks learn to close the lids (even in sleep) by active stimulation of the seventh nerve (as in forcibly squeezing the lids closed) and thus the startled appearance of undercorrection is avoided.

Frequent causes for dissatisfaction result from failure to note the following precautions:

1. Be certain that the fascia lata is attached **under** the periosteum (between the periosteum and bone). If not, the end of the strip slips down when the gut suture absorbs.

2. The incision along the lash border should be only deep enough to get a good bleeding line. Too deep an incision allows the sling to pull up subcutaneously

and lessen the correction. Epithelium will grow over the sling even though it appears to lie mostly on top of the skin at the lid border.

3. To obtain a lid that is open more widely at the inner angle, it is important that the sling is almost vertical to the lid border (the brow incision being centered directly above the upper punctum). The temporal incision should be far enough out that the fascia lata will lie in a position about 60 degrees to the lid border (principle of resolution of forces).

4. Instruments designed to pass the fascia lata are shown.

---

**11:00 a.m.**

### A HEREDITARY AND CLINICAL STUDY OF CHOROIDEREMIA

**Clement McCulloch, M.D.**
BY INVITATION
**R. J. P. McCulloch, M.D.**
**Toronto, Canada**

Discussers:

Arthur J. Bedell, M.D.
Albany, N. Y.

F. Bruce Fralick, M.D.
Ann Arbor, Mich.

---

Cases of choroideremia are studied and traced in two families, whose members are scattered from coast to coast in Canada and in many places in United States. One family contains over 600 members, including 79 examined cases of choroideremia; the other, 12 members and 8 cases of choroideremia.

The disease is transmitted by a sex-linked recessive characteristic. The male shows the malignant form of the disease. He is born with pigmentary changes in the fundus. Starting in childhood, or young manhood, and progressing gradually he goes through stages of night blindness, loss of visual field, complete blindness. Accompanying these symptoms the fundus shows atrophy of choroid and pigment epithelium, starting in the mid-periphery and progressing to involve the far periphery and finally the macula. The female can be considered a carrier because she transmits the malignant form of the disease to her sons but she herself only has the benign form. From earliest childhood there is a pigmentary change in her fundus. This does not alter during life and she never suffers from any loss of vision.

---

## SECTION ON OTOLARYNGOLOGY

**Thursday, October 16, 1947**

**2:00 p.m.**

**GRAND BALL ROOM**

*Presiding Officer:*
NORTON CANFIELD, M.D.
*Third Vice President*

*Secretary:*
O. E. VAN ALYEA, M.D.

---

### PLASTIC REPAIR OF THE INJURED FACE

**John Marquis Converse, M.D.**
BY INVITATION
**New York, N. Y.**

Discussers:

T. G. Blocker, Jr., M.D.
BY INVITATION
Galveston, Texas

Fred Z. Havens
Rochester, Minn.

---

**2:40 p.m.**

### LABYRINTHINE TESTS WITH STREPTOMYCIN POISONING

**(A Motion Picture)**

**Aram Glorig, M.D.**
BY INVITATION
Washington, D.C.

---

A film showing the labyrinthine tests necessary for the evaluation of the toxic effects of streptomycin therapy. Particular stress is laid on quantative caloric tests, after the methods of Fitzgerald, Cawthorne and Hallpike.

Tilt table and gait. These tests are of considerable value in the study of labyrinthine lesions from other causes.

---

**3:00 p.m.**

## STREPTOMYCIN TREATMENT OF VERTIGO

**Edmund P. Fowler, Jr., M.D.**
**New York, N. Y.**

Discussers:

Paul A. Campbell, M.D.
Chicago, Ill.

---

On a suggestion of Joseph E. Hawkins, Jr., of Merck & Co., streptomycin has been tried as a therapeutic agent in patients with severe attacks of vertigo. The majority of these were typical of labyrinthine hydrops. Properly selected cases seem to be benefited. It is questionable whether this treatment should be used on any case over 50 years of age for compensation for vestibular loss is likely to be deficient in old people. Patients whose occupations involved sudden turning or activity in the dark were eliminated. The treatment consists of dosage which will produce partial bilateral vestibular dysfunction as controlled with quantitative caloric tests after the manner of Fitzgerald, Cawthorne and Hallpike. Most of the cases had been previously tried on histamine, anti-histamine, nicotinic acid, salt free diet, dehydration and the like.

---

**3:30 p.m.**

## THE REHABILITATION OF THE LARYNGECTOMIZED

**Herbert Koepp-Baker, M.S., Ph.D.**
BY INVITATION
**Chicago, Ill.**

Discussers:

Paul A. Moore, M.D.
BY INVITATION
Evanston, Ill.
J. W. McCall, M.D.
Cleveland, Ohio

---

The loss of the larynx with its attendant obliteration of the speech function produces a grave adjustive psychosocial crisis, the full significance of which becomes apparent only by a proper evaluation of the primacy of social communication in mental health.

The satisfactory solution of the train of intellectual, emotional and vocational problems initiated by surgery requires a tripartite re-educational approach:

(1) Rehabilitation should be begun before, not after, surgery has intervened.

(2) The technics of esophageal speech or the proper use of a pneumatic or electronic speech-aid, must be taught systematically and be varied in terms of the intellectual, emotional and experiential endowments of the patient.

(3) The rehabilitation is neither adequate nor complete unless new attitudes toward self, the disability, and the social environment are structured, and general non-speech adjustment skills are developed.

Proper management of the laryngectomized patient must include early referral to, and continuous co-operative clinical planning with the speech pathologist.

---

**4:10 p.m.**

## THE USE OF RADIUM IN THE TREATMENT OF NASOPHARYNGEAL LYMPHOID TISSUE AND IN THE PREVENTION OF CONDUCTION DEAFNESS

**(A Motion Picture)**

**Gilbert E. Fisher, M.D.**
**Birmingham, Ala.**

---

This is a 16 mm. Kodachrome motion picture from the Department of Otolaryngology, Medical College of Alabama, Birmingham, Alabama. It shows the routine ear, nose and throat examination prior to the use of radium for treatment of hypertrophied nasopharyngeal lymphoid tissue in the adult and also the examination and audiometric study prior to and following radium treatment for the prevention of conduction deafness in children. The nasopharyngoscopic examination is shown, as well as the application of the radium. The film closes with pictures of audiograms showing the hearing before and after treatment with radium.

**4:20 p.m.**

### A METHOD OF TREATMENT OF EXTENSIVE STRICTURE IN THE OROPHARYNX

**Sam H. Sanders, M.D.**
**Memphis, Tenn.**

Discussers:

Henry M. Goodyear, M.D.
Cincinnati, Ohio

V. H. Kazanjian, M.D.
Boston, Mass.

---

This paper offers an artificial oropharynx which the author developed and used successfully in the surgical treatment of an extensive oropharyngeal stricture. The appliance involved is essentially an artificial oropharynx designed to:

(a) Maintain the essential orifice of the oropharynx following surgical removal of a stricture;

(b) Re-establish a lumen sufficiently stable to support a skin graft as the final stage in the restoration of a normal oropharynx.

This appliance is adaptable for use in cases with strictures in the oropharynx or hypopharynx.

A discussion of the cause of stricture is given. The literature has been reviewed and is reported.

A case report is given in detail of a girl, age ten, whose tonsils and adenoids had been removed by a competent surgeon. The oropharynx and nasopharynx practically closed off within a month without any apparent cause. Three attempts were made to relieve the stricture by the operating surgeon, and the details were reported to the author.

A description of the findings, operative procedures and evolution of the appliance used to prevent the recurrence and contracture of the scar tissue, and to hold the skin graft in place is given.

Slides showing the lumen of the pharynx before operation, the first appliance used and second or improved appliance in place, a photograph of the patient after four years, a reproduction of the soprano voice that won a state high school contest is offered.

---

## SECTION ON OPHTHALMOLOGY

### Friday, October 17, 1947
### 9:00 a.m.

**GRAND BALL ROOM**

*Presiding Officer*:
ALAN C. WOODS, M.D.
*President*

*Secretary*:
ALGERNON B. REESE, M.D.

---

### DACRYOCYSTORHINOSTOMY

**Michael J. Hogan, M.D.**
**San Francisco, Calif.**

Discussers:

Harold Gifford, M.D.
Omaha, Neb.

Alton V. Hallum, M.D.
Atlanta, Ga.

---

The indications for dacryocystorhinostomy are described. The Toti-Mosher operation is discussed and the results are presented in the follow-up of 52 operative cases. Case reports which illustrate methods of handling some complications of dacryocystitis are presented.

**9:40 a.m.**

### WOUND CLOSURE IN CATARACT EXTRACTION

**(A Motion Picture)**

**Harold G. Scheie, M.D.**
**Philadelphia, Pa.**

Various types of sutures used in cataract extraction are reviewed and illustrated. A type of closure used at the Hospital of the University of Pennsylvania is demonstrated. A conjunctival flap is reflected toward the cornea over the area of the section. Three double arm sutures are placed in the sclera adjacent to the limbus at eleven-thirty, twelve and one-thirty o'clock. Following completion of the section these sutures are then continued through the base of the conjunctival flap at the margin of the cornea. After delivery of the lens the sutures are tied. The conjunctival flap is rejoined to the conjunctiva above by the use of a continuous suture, occasional bites being taken in the episclera for firm fixation.

**10:00 a.m.**

### EXPERIMENTAL TREATMENT OF RETINITIS PIGMENTOSA

**Dan M. Gordon, M.D.**
**New York, N.Y.**

Discusser:

Alan C. Woods, M.D.
Baltimore, Md.

Over 100 patients with retinitis pigmentosa were treated with injections of cod liver oil, shark liver oil, vitamins A, and A and D, and subconjunctival placental implantations after the methods recently described in the Russian literature. Studies, including visual acuity, visual fields and dark adaptation failed to substantiate the results claimed by Filatov.

**10:40 a.m.**

### LATERAL DECOMPRESSION OF THE ORBIT

**(A Motion Picture)**

**Jack S. Guyton, M.D.**
**Baltimore, Md.**

A curved skin incision about 7 cm. long, concave forward, is made approximately 5 cm. back of the superior temporal orbital margin, just within the hairline. The skin is undermined over the temporal fascia to expose the lateral orbital margin. The periosteum is elevated from outside the lateral orbital wall, and the temporal muscle retracted posteriorly. The orbital periosteum is also elevated from the inside of the lateral orbital wall. The lateral edge of the zygoma is rongeured away and a wedge-shaped section of bone, approximately 8 sq. cm. in area, is removed from the lateral orbital wall with rongeurs. The bone is removed inferiorly to the body of the maxilla and the inferior orbital fissure, posteriorly and superiorly to the inner table of the great wing of the sphenoid and to the body of the frontal bone. The orbital periosteum is carefully opened with a "T" shaped incision. The skin incision is closed, the lids are sutured temporarily and a pressure bandage is applied to the eye without pressure over the temporal fossa. This pressure is maintained most of the time for one month after operation.

**11:00 a.m.**

### OCULAR HYPOTONY

**Parker Heath, M.D.**
**Boston, Mass.**

Discussers:

John H. Dunnington, M.D.
New York, N. Y.

Brittain F. Payne, M.D.
New York, N. Y.

Anderson C. Hilding, M.D.
Duluth, Minn.

Relatively little attention has been paid to pathology of low intraocular pressure. The deterioration of the visual fields associated with intraocular pressure above 26 mm. of Hg (Schiötz) has an immense literature. Those measurements of 10 mm. of Hg or less have received little attention. The subject of hypotony will be discussed under the following headings:

1. Etiology:
   - Contusions
   - Myopia
   - Uveitis and inflammation
   - Detached retina
   - General disease states as diabetes and anemias
   - Endocrine disease
   - Nervous origin
   - Malformation
   - Therapeutic origin (drug and surgical)
2. Pathology
3. Treatment
4. References

---

**11:40 a.m.**

## A REFRACTION CHART

**(A Motion Picture)**

**Walter H. Fink, M.D.**
**Minneapolis, Minn.**

---

The movie describes a chart which is designed specifically for refraction. An attempt is made in this chart to eliminate certain defects which are found in the standard chart. The technic of using it is described.

# BUSINESS MEETING

**Grand Ball Room**

**Wednesday, October 15, 1947**

Following the presentation of the Jackson and Wherry Memorial Lectures at 8:30, Wednesday evening, the Annual Academy Business Meeting will be held. The proceedings of the meeting will be published in the November-December issue of the TRANSACTIONS.

ORDER OF BUSINESS

1. Call to order.
2. Report of Executive Secretary-Treasurer.
3. Report of Necrologist.
4. Report of Editor of TRANSACTIONS.
5. Committee reports.
6. Report of American Board of Ophthalmology.
7. Report of American Board of Otolaryngology.
8. Unfinished business.
9. New business.
10. Report of the Senior Member of the Council.
11. Election of Fellows. (See Candidate list below.)
12. Election of officers (Senior Member of Council report).

---

## CANDIDATES — 1947

Objections to the election of a candidate must be filed with the Executive Secretary previous to Saturday, October 11, 1947.

**CANDIDATES CERTIFICATED BY BOARDS**

**Ophthalmology (OP)**
**Otolaryngology (ALR)**
**Plastic Surgery (Pl S)**

**Adair, Albert Franklin, Jr., 822 Lowry Medical Arts Bldg., St. Paul, Minn., OP.**

**Aderhold, James P., Medical Arts Bldg., San Antonio, Tex., ALR.**

Agin, Lambert James, 610 N. Market, Canton, Ohio, OP.

Ahroon, William Alstrom, Clifton Springs Sanitarium, Clifton Springs, N. Y., ALR.

Aiken, Samuel D., 384 Post St., San Francisco, Calif., OP.

Alexander, Maurice Harry, 1915 Spruce St., Philadelphia, Pa., ALR.

Alonso, Miguel, Box 703, Rio Piedras, P. R., ALR.

Alport, Benjamin, 121 Chestnut St., Springfield, Mass., OP.

Anthony, Eugene William, 429 South Fourth St., Fulton, N. Y., OP.

Aycock, Byron Wolverton, 608 C. Ave., Lawton, Okla., ALR.

Backer, Meyer, 3935 W. Chicago Ave., Chicago, Ill., OP.

Bailey, Arthur Thomas, 7605 Seville Ave., Huntington Park, Calif., ALR.

Baisinger, Leo Fenimore, 2026 17th St., Bakersfield, Calif., OP.

Ball, Simon, 255 South 17th St., Philadelphia, Pa., ALR.

Ballenger, John Jacob, 723 Elm St., Winnetka, Ill., ALR.

Ballweg, Harry Albert, 66-58 Fresh Pond Road, Ridgewood, N. Y., OP.

Bannon, Frederick M., 300 Main St., Stamford, Conn., ALR.

Barkley, Douglas Ferguson, 1000 Lavaca, Austin, Tex., ALR.

Barton, Richard Thomas, 120 S. Lasky Drive, Beverly Hills, Calif., ALR.

Bates, Charles Edward H., 350 Post St., San Francisco, Calif., ALR.

Beery, Edwin Newman, 96 Remsen St., Brooklyn 2, N. Y., OP.

Behrens, Herbert Charles, 226 N. Greenleaf Ave., Whittier, Calif., OP.

Berlove, Ira Jay, 35 East 65th St., New York, N. Y., ALR.

Blake, Emerson Millen, 1301 Broadway, Lubbock, Tex., ALR.

Blanchard, Edward James, Bank of America Bldg., Mez. Floor, Whittier, Calif., ALR.

Blase, Charles Stenger, 2600 Union Central Bldg., Cincinnati, Ohio, ALR.

Blondis, Robert Richard, Schofield Bldg., Cleveland 15, Ohio, OP.

Bloom, Edward I., 255 S. 17th St., Philadelphia, Pa., ALR.

Bloom, Henry Rostler, 311 Commonwealth Ave., Boston 15, Mass., OP.
Bloomfield, Sylvan, 1010 Fifth Ave., New York, N. Y., OP.
Blum, Harry, 865 St. Marks Ave., Brooklyn, N. Y., ALR.
Bodian, Martin, 349 Eastern Parkway, Brooklyn 16, N. Y., OP.
Bolstad, Donald Stanford, 2799 W. Grand Blvd., Detroit 2, Mich., ALR.
Boon, Clifton Udonna, 25 S. Broadway, Aurora, Ill., ALR.
Botvin, Morris, 155 Angell St., Providence 6, R. I., OP.
Bowerman, Harold H., 607 North Grand, St. Louis 3, Mo., ALR.
Braconier, Harry E., 264 Beacon St., Boston 16, Mass., OP.
Bregande, Samuel C., 327 State Tower, Syracuse 8, N. Y., ALR.
Brennan, Russell James, 6100 Walnut St., Philadelphia, Pa., ALR.
Brick, Enoch B., 526 Third St., Wausau, Wis., ALR.
Brinckerhoff, Albert Johnson, 490 Post St., San Francisco 2, Calif., OP.
Brogan, Edmund James, 1909 Spruce St., Philadelphia 3, Pa., ALR.
Brown, Seymour Milton, 1 Ridge Road, Baltimore, Md., ALR.

Caldemeyer, Everett S., 1826 R Street N.W., Washington, D. C., OP.
Campion, George Stuart, 490 Post St., San Francisco 2, Calif., OP.
Chan-Pong, Norman Ronald, 88 Frederick St., Port of Spain, Trinidad, B. W. I., OP.
Childrey, Edgar, Jr., 605 Professional Bldg., Richmond, Va., OP.
Clark, Ernest Walton, 414 Cooper St., Camden, N. J., ALR.
Cooley, Justus H., 150 W. High St., Somerville, N. J., ALR.
Crage, Michael James, 25 East Washington St., Chicago, Ill., ALR.
Cramer, Irving I., 836 Rose Bldg., Cleveland 15, Ohio, ALR.
Crigler, Fielding Jason, 104 E. Market St., Charlottesville, Va., OP.
Curran, Timothy L., 689 Asylum Ave., Hartford 5, Conn., ALR.

Davis, James S., 2 Gibson Court, So. Norwalk, Conn., ALR.
Davol, Rector Thomson, 63 North St., Greenwich, Conn., ALR.
Deichler, John Waller, 1930 Chestnut St., Philadelphia, Pa., OP.
Derlacki, Eugene L., 55 E. Washington St., Chicago 2, Ill., ALR.
Dickson, Owen C., 2628 Telegraph Ave., Berkeley 4, Calif., OP.
Dorne, Ralph M., 25 E. Washington, Chicago 2, Ill., ALR.
Duane, Thomas David, College of Medicine, Iowa City, Iowa, OP.
Dunkelberger, John Alfred, 621 East Market St., York, Pa., ALR.
Dunlap, Edward A., 525 E. 68th St., New York 21, N. Y., OP.

Edgerly, Sherburn Edward, 220 Engle St., Englewood, N. J., ALR.
Eisenhower, Charles Earl, 426 W. Market St., York, Pa., OP.
Elvin, Norris C., 465 Ocean Ave., Brooklyn, N. Y., OP.
Erdel, Milton William, 306 Peoples' Life Bldg., Frankfort, Ind., ALR.
Escapini, Humberto, 9 East 91st St., New York, N. Y., OP.

Feldberg, Irving, Veterans Administration, Bath, N. Y., OP.
Filak-Perez, Ann, Harwood Bldg., Scarsdale, N. Y., OP.
Fine, Irvin Jack, 280 Hobart St., Perth Amboy, N. J., ALR.
Fischer, Arthur J., 623 Union Trust Bldg., Pittsburgh, Pa., ALR.
Fishman, Jerome, 30 N. Michigan Ave., Chicago 1, Ill., ALR.
Flatley, Robert Edward, 1630 5th Ave., Moline, Ill., OP.
Forman, Frank S., 1110 Professional Bldg., Kansas City 6, Mo., ALR.
Forney, Claudius Langdon, 4619 S. South Parkway, Chicago 15, Ill., OP.
Frank, Walter L., 313 Medical Arts Bldg., Minneapolis 2, Minn., OP.
Friedman, Orel, 469 Madison Ave., Albany 6, N. Y., ALR.
Friend, Amos Edgar, 935 Main St., Manchester, Conn., ALR.
Futch, Charles Edward, 1930 Wilshire Blvd., Los Angeles, Calif., ALR.

Gagliardi, George R., 56 Proctor St., Framingham, Mass., OP.
Ganz, Samuel Evans, 155 East 72nd St., New York 21, N. Y., ALR.
Gardiner, Frederick S., 614 Rose Bldg., Cleveland 15, Ohio, ALR.
Gillespie, Albert Ritchie, Corner Lewis & Frederick Sts., Staunton, Va., ALR.
Ginsberg, Norman A., 720 Bryant Bldg., Kansas City 6, Mo., ALR.

Glassburn, Edward Myler, 6102 Jenkins Arcade, Pittsburgh 22, Pa., ALR.
Glorig, Aram, Jr., Veterans Administration (Room 880), Washington, D. C., ALR.
Gordon, Betty Constance, 99 Lafayette Ave., Brooklyn 17, N. Y., OP.
Gordon, Orville E., 30 N. Michigan Ave., Chicago, Ill., OP.
Grafton, Edwin G., Jr., 3916 Worth St., Dallas, Tex., OP.
Gray, Claude Cleveland, Medico-Dental Bldg., Sacramento, Calif., OP.
Gray, Leon Fowler, 509 Marshall St., Shreveport 4, La., OP.
Gray, Raymond John, 5095 Jenkins Arcade, Pittsburgh 22, Pa., OP.
Green, Frank Murray, 1502 Mott Ave., Far Rockaway, N. Y., OP.
Greenfield, Philip, Veterans Hospital, Temple, Tex., OP.
Grimes, Martin Osmond, 57 Kay St., Newport, R. I., OP.
Grunfeld, Rudolph, 320 Ocean Parkway, Brooklyn 18, N. Y., OP.
Guerin, Leon Henry, 324 E. Wisconsin Ave., Milwaukee 2, Wis., ALR.

Haas, Joseph Stahl, 58 E. Washington St., Chicago, Ill., OP.
Hale, Channing Ward, 281 S. Thomas St., Pomona, Calif., OP.
Hall, Albert Winter, 6820 Windsor Ave., Berwyn, Ill., ALR.
Hall, James Tidwell, McMillan Hospital, St. Louis 10, Mo., ALR.
Hanna, William Henry, 1801 K Street, N.W., Washington 6, D. C., OP.
Hanson, Henry V., Veteran's Hospital, Minneapolis, Minn., ALR.
Harbert, Frederick, Capt. (MC) USN, U.S. Naval Hospital, Philadelphia, Pa., OALR.
Harbin, Thomas Shelor, Harbin Hospital, Rome, Ga., OP.
Harrington, David O., 384 Post St., San Francisco, Calif., OP.
Henderson, Charles William, 317 West Pueblo St., Santa Barbara, Calif., OALR.
Henderson, John Warren, Mayo Clinic, Rochester, Minn., OP.
Henderson, John Woodworth, University Hospital, Ann Arbor, Mich., OP.
Hendricks, John Ellsworth, 14 West Locust St., Newark, Ohio, ALR.
Henry, Margaret, 384 Post St., San Francisco 8, Calif., OP.
Hepp, Virgil Eugene, 261 Hamilton, Palo Alto, Calif., ALR.
Hilsinger, Raymond Louis, 380 Doctors Bldg., Cincinnati 2, Ohio, ALR.
Himmel, Jacob Gordon, 206 Commonwealth Bldg., Cleveland, Ohio, ALR.
Hoeper, Philip George, Mankato Clinic, Mankato, Minn., OP.
Holmberg, Conrad Joel, 527 Medical Arts Bldg., Minneapolis 2, Minn., ALR.
Hulse, William Francis, 1314 Hanna Bldg. Cleveland 15, Ohio, ALR.
Hultgen, William James, 65 N. Cretin Ave., St. Paul 4, Minn., OP.
Hume, Charles Raymond, 1301 Washington, Vicksburg, Miss., ALR.
Humphries, Marion Kemper, Jr., 104 E. Market St., Charlottesville, Va., ALR.
Hyman, Joseph, 645 Ocean Ave., Brooklyn 26, N. Y., OP.

Igersheimer, Joseph, 483 Beacon St., Boston 15, Mass., OP.
Iliff, Charles Edwin, 12 W. Read St., Baltimore 1, Md., OP.

Jakobovits, Rafael, 1801 Bush St., San Francisco 9, Calif., OP.
Janssen, Louis William Oscar, 710 Medical Professional Bldg., Corpus Christi, Tex., ALR.
Jeancon, Etta Charlotte, 523 West 6th St., Los Angeles 14, Calif., OP.
Jenkins, William Herndon, 1150 Connecticut Ave. N.W., Washington 6, D. C., ALR.
Jennes, Milton Leo, 76 Center St., Waterbury 18, Conn., ALR.
Johnson, Raymond A., Medical Arts Bldg., Oak Ridge, Tenn., ALR.
Johnson, Robert Marion, 1422 Medical Arts Bldg., Houston, Tex., OP.
Johnson, W. F., 218 2nd Ave., New York 3, N. Y., OP.
Jordan, Valdemar Mandell, 10515 Carnegie Ave., Cleveland 6, Ohio, ALR.
Joseph, Gerald Feitel, 2221 Plank Road, Baton Rouge, La., ALR.

Kadin, Maurice, 535 Main St., Racine, Wis., OP.
Kane, Louis Allin, 329 Bloor St. W., Toronto, Ont., Canada, ALR.
Kaplan, Morris, 1612 Tremont Place, Denver 2, Colo., OP.
Keller, Paul Kenneth, 317 Marble Arcade, Lakeland, Fla., ALR.

Kempthorne, Charles Roosevelt, First Nat'l Bank Bldg., Manhattan, Kan., ALR.
Kerschgens, Lambert Joseph, 405 Nat'l Exchange Bank Bldg., Steubenville, Ohio, ALR.
King, Merrill J., 264 Beacon St., Boston, Mass., OP.
Koester, Carl Cornelius, Genesee Trust Bldg., Batavia, N. Y., ALR.
Kuhlman, William Keim, 209 South Nevada Ave., Colorado Springs, Colo., OP.

Lake, Clifford Franklin, 102 2nd Ave., S.W., Rochester, Minn., ALR.
Laub, George Rudolph, 1627 Bull St., Columbia, S. C., ALR.
Laughlin, Robert Clark, 321 Stimson Bldg., Seattle 1, Wash., OP.
Lawlor, Edward Francis, 267 Moody St., Waltham 54, Mass., ALR.
Lemoine, Albert N., Jr., 1100 Rialto, 906 Grand Ave., Kansas City 6, Mo., OP.
Lerner, Sydney Alex, 7905 Cottage Grove, Chicago 19, Ill., ALR.
Levine, Jack Louis, 1217 Valley Ave., S.E., Washington, D. C., ALR.
Linksz, Arthur, 6 East 76th, New York 21, N. Y., OP.
Linn, Jay G., Jr., 7075 Jenkins Arcade, Pittsburgh 22, Pa., OP.
Lobel, Mervyn Joseph, 25 W. 54th St., New York 19, N. Y., ALR.
Lobsenz, Nathan Philip, 294 Broadway, Paterson 1, N. J., OALR.
Long, Theodore Kohr, 504 Cumberland St., Lebanon, Pa., OP.
Loring, Roland M., 30 N. Michigan Ave., Chicago 2, Ill., ALR.
Lucie, Hugo, 233 'A' St., San Diego 1, Calif., OP.

Machamer, R. Wenner, 1027 Rose Bldg., Cleveland, Ohio, ALR.
Macrae, Henry Mitchell, Medical Arts Bldg., Toronto, Ont., Canada, OP.
Maddox, John Daniel, 528 Pearl St., Joplin, Mo., OP.
Mancoll, Morris Max, 242 Trumbull St., Hartford, Conn., ALR.
Marshall, Wilbur James, 618 Lander's Bldg., Springfield, Mo., OP.
Martin, Walter Humbert, 7 E. Mission, Santa Barbara, Calif., ALR.
Martz, George Everett, 505 North Second St., Harrisburg, Pa., OP.
Mastny, Val Joseph, 610 Hanna Bldg., Cleveland 15, Ohio, ALR.
Mazique, Douglas Wilkerson, 5508 S. Kimbark, Chicago 37, Ill., ALR.
McCall, James Donovan, Williams Clinic, Mineral Wells, Tex., ALR.
McCannel, Malcolm A., McCannel Clinic, Minot, N. D., OP.
McCormack, Charles Goodsell, Capt. (MC) USN, U.S. Naval Hospital, Bethesda 14, Md., ALR.
McCoy, David A., 501-6 Woodward Bldg., Birmingham, Ala., OP.
McCoy, Harold J., 616 B. T. Bldg., Des Moines, Iowa, ALR.
McCulloch, John Clement, Suite 830 Medical Arts Bldg., Toronto, Ont., Canada, OP.
McEachran, Hugh Douglas, 521 Stephenson Ave., Iron Mountain, Mich., ALR.
McGlade, Thomas Henry, 719 Cooper St., Camden, N. J., ALR.
McKenzie, Rodney Jones, 229 Doctor's Bldg., Cincinnati, Ohio, ALR.
McKenzie, Walter Holt, 1012 Medical Arts Bldg., Fort Worth, Tex., OP.
McKey, Earle Sinclair, Jr., 400 W. Central, Valdosta, Ga., OP.
McNaught, Robert Chamberlain, Stanford Hospital, San Francisco 15, Calif., ALR.
Mehney, Gayle Henry, 504 Medical Arts Bldg., Grand Rapids, Mich., OP.
Merideth, Howard W., 5159 S. Damen Ave., Chicago 9, Ill., ALR.
Merrifield, Frederick William, 122 S. Michigan Ave., Chicago 3, Ill., Pl S.
Montano, Rocco A., 725 Fourth St., Marysville, Calif., OP.
Moore, James I., 11 E. Chase St., Baltimore 2, Md., OP.
Morrissey, Arthur Michael, 185 High St., Medford 55, Mass., OP.
Muir, Everett Barclay, 804 Boston Bldg., Salt Lake City 1, Utah, OP.
Munsen, Henry TenEycke, 987 E. Jefferson, Detroit 7, Mich., ALR.

Naples, Carmon Robert, 1746 K St., N.W., Washington 6, D. C., OP.
Nicholls, John VanVliet, 1414 Drummond St., Montreal, Que., Canada, OP.

O'Neal, Buford Lee, 478 Peachtree St. N.E., Atlanta, Ga., ALR.
Ostrov, Norman N., 355 West 51st St., New York, N. Y., ALR.
Owens, Daniel Edwin, 22-A.W. Micheltorena St., Santa Barbara, Calif., ALR.

Pang, Lup Quon, 52 S. Vineyard St., Honolulu 39, T. H., ALR.
Parisi, Peter Joseph, Crile V. A. Hospital, Cleveland 9, Ohio, OP.
Patt, Albert J., M.D., 1727 New Jersey St., Los Angeles 33, Calif., ALR.
Patton, William George, 2 North Garfield Ave., Alhambra, Calif., ALR.
Penn, Samuel E., 3401 Fifth Ave., Pittsburg 13, Pa., ALR.
Pice, Jose Theodore, Ponce de Leon Ave., Box 7063, Santurce, P. R., ALR.
Poole, Gerald Ogden, 600 Delaware Ave., Wilmington, Del., ALR.

Quantz, Herman Celestian, 707 Guaranty Bank Bldg., Alexandria, La., ALR.
Quisling, Rolf Andreas, 2 W. Gorham, Madison 3, Wis., ALR.

Ravin, Oscar G., 625 Broadway, San Diego 1, Calif., OP.
Reinhorn, Abraham John, 295 Broadway, Paterson 1, N. J., OP.
Regal, Nathan, 105 E. 63rd St., New York, N. Y., OP.
Renken, Paul William, 812 Gravier St., New Orleans 12, La., OP.
Rich, Benjamin S., 404 Medical Arts Bldg., Baltimore 1, Md., ALR.
Riddle, Ransford John, Corner W. State & Porter Way, Sharon, Pa., OP.
Robbins, Alfred R., 1930 Wilshire Blvd., Los Angeles 5, Calif., OALR.
Roberts, Walter L., 727 W. 7th St., Los Angeles, Calif., OP.
Robertson, Gaynelle, 411 3rd Ave. North, Texas City, Texas, OP.
Romaine, Hunter H., 111 E. 65th St., New York, N. Y., OP.
Romejko, Walter J., 1801 K St. N. W., Washington 6, D. C., OP.
Rosenberg, Abner Samuel, 142 Joralemon, Brooklyn 2, N. Y., OP.
Rossby, Allan, 49 E. 51 St., New York 22, N. Y., OP.
Ruben, Maurice Richard, 311 Commonwealth Ave., Boston, Mass., ALR.

Sachs, James Wilbur, 151 W. Washington St., Hagerstown, Md., ALR.
St. Germain, Ellis Louis, 1008 N. Main St., Bloomington, Ill., OP.
Sakler, Allen M., 524 Francis Bldg., Louisville 2, Ky., OP.
Sanger, Welborn W., 117 North Broadway, Oklahoma City 2, Okla., OP.
Saradarian, Albert Victor, 2401 New York Ave., Union City, N. J., OP.
Saunders, George Chancellor, 601 Medical Arts Bldg., Portland, Ore., ALR.
Scharfe, Ernest Edward, 1414 Drummond St., Montreal, Que., Canada, ALR.
Schott, Edward George, Sheboygan Clinic, Sheboygan, Wis., OP.
Schultz, Alfred George, Dunlap Hotel Jacksonville, Ill., OP.
Schumacher, James Charles, 2600 Far Hills Ave., Dayton, Ohio, OP.
Schuman, Irving, 3507 Lawrence Ave., Chicago 25, Ill., OP.
Schwarz, Wilfred John, Donaghey Bldg., Little Rock, Ark., ALR.
Scimeca, Anthony A., 111 E. 61st St., New York 21, N. Y., OP.
Sekerak, Richard John, 938 E. Main St., Bridgeport 8, Conn., ALR.
Sellitto, Anthony Michael, 115 Connett Place, South Orange, N. J., OP.
Shankel, Harry Weylman, 1612 Tremont Place, Denver 2, Colo., OP.
Sharbaugh, George B., 212 West State St., Trenton 8, N. J., OP.
Shattuck, Robert Cummings, 1612 Tremont Place, Denver 2, Colo., ALR.
Sheldon, Maurice Gerald, 604 Exchange Bank Bldg., Olean, N. Y., ALR.
Sherwood, Robert O., 128 Primrose Road, Burlingame, Calif., OP.
Smart, Frank P., 315 Medical Arts Bldg., Norfolk 10, Va., OP.
Smith, Bryant P., Jr., Highland Clinic, Shreveport, La., ALR.
Smith, Byron, 927 Park Ave., New York, N. Y., OP.
Smith, John William, 1041 Donaghey Bldg., Little Rock, Ark., ALR.
Soifer, Nathaniel, 627 Salem Ave., Dayton 6, Ohio, ALR.
Souders, Benjamin Franklin, 143 North 6th St., Reading, Pa., OP.
Spence, Elbert Lafayette, 115 St. Francis St., Kennett, Mo., ALR.
Spero, Gerald David, 2317 David Broderick Tower, Detroit, Mich., OP.
Stayer, Glenn Emory, 420 Woodward Bldg., Birmingham, Ala., OALR.
Steinberg, Theodore, 1801 Bush St., San Francisco, Calif., OP.
Stellwagen, William John, 1322 Madison St., Seattle 4, Wash., OP.
Sullivan, Richard Leo., 910 State Tower Bldg., Syracuse 2, N. Y., ALR.

Taterka, Henry D., 302 W. 87th, New York 24, N. Y., ALR.
Teal, Frederick Franklin, 910 Sharp Bldg., Lincoln 8, Neb., OP.

Tenaglia, Eutimio D., 634 N. Grand Ave., St. Louis 3, Mo., OP.
Thompson, John Barkwell, 1308 3rd Ave., Columbus, Ga., ALR.
Tipple, Albert McCoun, 309 W. 20th St., Pueblo, Colo., ALR.
Tirrell, C. Malcolm, 1421 State St., Santa Barbara, Calif., ALR.
Titus, Bruce Linville, 1020 S.W. Taylor St., Portland 5, Ore., ALR.
Tresley, Ira J., 25 E. Washington St., Chicago, Ill., ALR.

Walzl, Edward McColgan, Johns Hopkins Hospital, Baltimore 5, Md., ALR.
Warder, Frank M., Professional Bldg., Anderson, S. C., ALR.
Watson, Edwin Josiah, 404 M St. N.W., Washington, D. C., OP.
Weller, Wendell A., Col., M.C., U.S.A., Letterman General Hosp., San Francisco, Calif., ALR.
Wherley, Harold F., 104 Fair Ave. N.E., New Philadelphia, Ohio, OP.
Whitsell, **Ora Earl, 824 Edmond** St., St. Joseph 54, Mo., ALR.
Whitworth, Clyde Walton, 123 N. Main St., Gainesville, Ga., ALR.
Williams, Walter F., Mayo Arcade, Ashland, Ky., ALR.
Wilson, James Sharp, 800 First Nat'l Bldg., Canton 2, Ohio, ALR.
Winborn, Claude De, 300 Medical Arts Bldg., Dallas 1, Tex., ALR.
Wipperman, Frederic Francis, 1921 Medical Arts Bldg., Minneapolis, Minn., OP.
Wolfe, Herman E., 200 E. 16th St., New York 3, N. Y., OP.
Worden, Donald King, 214 Salsberg Bldg., Lewiston, Idaho, ALR.

Yates, June, 210 Medical Professional Bldg., Corpus Christi, Tex., ALR.

Zeiss, Edward J., 103 W. College, Appleton, Wis., OP.

## CANDIDATES NOT CERTIFICATED

### (Eligible for Membership After Board Certification)

Gros, Joseph Conrad, Calle 25 No. 960, Vedado, Havana, Cuba.
Joshi, Sadashiva Gopal, 212 Kamala Kung, Matunga, Bombay 19, India.
Poirier, Ralph Alex, 1405 Stroh Bldg., Detroit 26, Mich.
Rowland, Alan Lawrence, 1031 Bank of America Bldg., San Diego, Calif.
Seelye, Norman Lee, 36 North Ayer St., Harvard, Ill.
Sullivan, Charles Joseph, 57 Paterson St., New Brunswick, N. J.
Thornhill, Gabriel Felder, 1304 Norwood Bldg., Austin, Tex.
Vermillion, Dale Dewitt, Goodland, Kan.
Vetter, Harmut, Otolaryngological Clinic Kantonsspital, St. Gallen, Switzerland.
Zarbaugh, Guy Frederic, 4150 5th Ave., Los Angeles 43, Calif.

# Necrology

## IN MEMORIAM

George Viven Allen, Topeka, Kan.
Joseph Henry Allen, Pasadena, Calif.
Ross Epting Anderson, Jackson, Miss.
Frederick James Bentley, Seattle, Wash.
Robert Beauchamp Boucher, Vancouver, B. C., Can.
Andrew Johnson Browning, Portland, Ore.
Joseph Marie Louis Bruno, Brooklyn, N. Y.
Thomas Edward Carmody, Denver, Colo.
Howard S. Clark, Minneapolis, Minn.
Robert Lewis Crockett, Oneida, N. Y.
Warren Beagle Davis, Philadelphia, Pa.
Louis Samuels Deitchman, Youngstown, Ohio
Wells Phillips Eagleton, Newark, N. J.
Charles John Edwards, Vicksburg, Miss.
Edward Coleman Ellett, Memphis, Tenn.
Raymond Edwin Elliott, Rochester, N. Y.
George Freiman, Brooklyn, N. Y.
Edward Frothingham, Woodhaven, L. I., N. Y.
Charles Willard Geiger, Kankakee, Ill.
Robert Gewanter, Long Island City, N. Y.
Matthew Gerard Golden, Brooklyn, N. Y.
Edgar Leonard Goodman, Washington, D. C.
Louis Richard Haas, Pittsburg, Kan.
Gaylord Crawford Hall, Louisville, Ky.
Edward Peter Halton, Holyoke, Mass.
Howard Eugene Hamilton, Cleveland, Ohio
Morris Louis Harris, Brooklyn, N. Y.
John Marvin Ingersoll, Miami, Fla.
George Baigrie Jobson, Franklin, Pa.
Thomas Otis Klingner, Springfield, Mo.
Robert K. Lambert, New York, N. Y.
Franklin Fayette Lane, Philadelphia, Pa.
Walter Ivan Lillie, Philadelphia, Pa.
James Orville MacDonald, New York, N. Y.
Charles Alford Maghy, San Diego, Calif.
John Joseph McDermott, St. Joseph, Mich.
Thomas D'Arcy McGregor, Buffalo, N. Y.
Claude Worth McKee, Greensburg, Pa.
J. Hallock Moore, Huntington, W. Va.
Edward Rodgers Neeper, Colorado Springs, Colo.
William Edward Patterson, Minneapolis, Minn.
Howard Wilber Peirce, Detroit, Mich.
Harold Vincent Phelan, Cleveland, Ohio
Albert Ernest Rector, Appleton, Wis.
Charles Tiffany St. Clair, Bluefield, W. Va.
G. William Schlindwein, Erie, Pa.
William Augustus Schwartz, Phoenix, Ariz.
O. W. Sedgwick, Ionia, Mich.
Thomas Hall Shastid, Duluth, Minn.
Samuel Anise Shoemaker, Orlando, Fla.
Wayne Pulley Sirles, Herrin, Ill.
Carl Miller Sneed, Columbia, Mo.
Theodore Lasater Terry, Boston, Mass.
Andrew Jay Timberman, Columbus, Ohio
Richard Joseph Tivnen, Chicago, Ill.
George Loring Tobey, Jr., Boston, Mass.
Robert Von der Heydt, Chicago, Ill.
Arthur Morris Zinkhan, Washington, D. C.

## GEORGE VIVEN ALLEN

Dr. G. V. Allen was born in 1872. He was graduated from Kansas Medical College in Topeka in 1898, and assumed his practice of diseases of the eye, ear, nose and throat in that city. Dr. Allen became a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1921 and was a Senior Fellow at the time of his death in 1946.

## JOSEPH HENRY ALLEN

Born in Madison, Wisconsin, April 6, 1873, Dr. Allen received his preliminary education in the Madison schools and earned his medical degree from Gross Medical College, Denver, Colorado in 1898.

For ten years he was clinical instructor in Gross Medical College, assistant to Dr. Robert Levy. His hospital appointments included Staff County Hospital, St. Anthony's Hospital, National Jewish Hospital, and Mercy Hospital. He was a specialist for the Union Pacific R. R. and the Veteran's Administration. During World War I he served in the Medical Corps.

A diplomate of the American Board of Otolaryngology, Dr. Allen's society affiliations included the American Academy of Ophthalmology and Otolaryngology, the Denver Medical Society, as well as county and state groups. He was a member of the Denver Athletic Club and the Lakewood Country Club.

In 1930 he retired from active practice because of a leg amputation. Dr. Allen died in Pasadena, California, September 8, 1946. He is survived by his sister, Miss Emma F. Allen, his wife, Grace M. Allen, a son, Joseph H. Allen, Jr., a daughter, Genevieve G. Stevens, and four grandchildren.

## ROSS EPTING ANDERSON

Dr. Ross E. Anderson died January 15, 1947 in Jackson, Mississippi, where he had practiced for twenty years. Born in Booneville, Mississippi, Dr. Anderson attended Mississippi College, where he received his B.A. degree, University of Mississippi, and Tulane University at New Orleans where he obtained his medical degree in 1922. He did graduate work at George Washington University and served his internship at Mississippi State Hospital.

A veteran of World War I, he was a member of the American Legion, a Mason, and a Shriner. He belonged to the American Medical Association, the Mississippi State Medical Association, the Central Medical Association, the Southern Medical Association, the American Academy of Ophthalmology and Otolaryngology, the Louisiana-Mississippi Otolaryngology Society, and was a diplomate of the American Board of Otolaryngology.

He is survived by his mother, Mrs. W. G. Anderson of Booneville, Mississippi, one brother, Walter G. Anderson, and a sister, Mrs. Edmund Miller.

## FREDERICK JAMES BENTLEY

Dr. Frederick James Bentley, one of Seattle's leading pioneer ophthalmologists, died on January 1, 1947, at the age of 71 years. He was born April 3, 1875, in Freeport, Illinois, and when still a child, his parents moved to Grand Island, Nebraska, where he attended the public schools. He later enrolled at the Poughkeepsie Military School of New York. Preparatory to entering the Physicians and Surgeons Medical School of New York, he attended Johns Hopkins University. He received his degree of Doctor of Medicine from Rush Medical School in 1901.

He then did general practice for a time at or near Kankakee, Illinois. He went abroad and specialized in ophthalmology at the Royal London Ophthalmic Hospital, from which he was certificated in 1903. Before returning to the United States, he attended the leading European clinics, especially the eye clinics in Vienna and Berlin. In 1904, he was licensed to practice in the State of Washington, locating in Seattle. In 1908 he went to India, as one of the first American ophthalmologists in that country, and took special work under Colonel Smith, the proponent of the intracapsular cataract extraction.

He was a member of the King County Medical Society, Washington State Medical Society, American Medical Association, American Academy of Ophthalmology and Otolaryngology, Pacific Coast Oto-Ophthalmological Society, Puget Sound Academy of Ophthalmology and Otolaryngology, American College of Surgeons, as well as holding a certificate of the American Board of Ophthalmology.

His interests and avocations were diversified and many. At one time he was an ardent mountain climber, being a member of the German Alpine Club while in Europe, as well as the Mountaineer's Club of Seattle. His leisure time and efforts were later devoted to his farm on Whidby Island. He gave generously of his time and efforts to many worthy charities, including the Children's Orthopedic Hospital and the Lighthouse for the Blind. The Seattle Symphony Orchestra and other musical enterprises were fortunate in having him as their enthusiastic sponsor and supporter.

He is survived by his brother, Arthur Bentley of Paoli, Indiana, and a sister, Mrs. Baird Paine of Grand Island, Nebraska.

---

ROBERT BEAUCHAMP BOUCHER

Dr. Boucher was born in Peterborough, Ontario, December 8, 1873. He received his preliminary education in the schools there and at Trinity College, Port Hope. Following his graduation from McGill University in 1895, he interned at Montreal General Hospital and then joined the Marine department of the Canadian Pacific Railway, where he served as Medical Officer on the Empress of India for two years. In 1899, Dr. Boucher moved to the mining town of Phoenix in the boundary country where he practiced surgery for a mining and smelting company until 1906.

Deciding at this time to limit his work to diseases of the eye, ear, nose and throat, Dr. Boucher traveled to Europe and studied in London, Vienna and other centers before settling in Vancouver in 1909 where he became a member of the staff of the Vancouver General Hospital.

Dr. Boucher was a member of the American Academy of Ophthalmology and Otolaryngology, Pacific Coast Oto-Ophthalmological Society, and the American College of Surgeons. In addition to his profession, Dr. Boucher was keenly interested in mining and proficient in the field of sports. He was an ardent curler and in later years, an enthusiastic golfer.

Dr. Boucher died July 22, 1946, in Vancouver, B. C. He is survived by his wife, Mrs. Robert Boucher, a brother, and two sisters.

ANDREW JOHNSON BROWNING

Dr. Browning was born in 1881. He received his Doctor of Medicine degree from George Washington University School of Medicine in 1908. A resident of Portland, Oregon, he had limited his practice to diseases of the eye since 1921.

Dr. Browning was a member of the staffs of the Good Samaritan, Multnomah, and Doernbecher hospitals. He was associate clinical professor of ophthalmology at the University of Oregon Medical School.

A diplomate of the American Board of Ophthalmology, Dr. Browning was a member of his local, county and state medical societies, the Oregon Academy of Ophthalmology and Otolaryngology, the Pacific Coast Oto-Ophthalmological Society, the Western Ophthalmological Society, and a Fellow of the American Academy of Ophthalmology and Otolaryngology.

His death occurred May 20, 1947. Surviving is a son, Dr. Charles W. Browning, of Portland.

---

JOSEPH MARIE LOUIS BRUNO

Dr. Joseph Bruno began his practice of medicine in Brooklyn, N. Y., in 1919. He obtained his preliminary education at St. John's College in Brooklyn, and received his medical degree in 1917 from Long Island College of Medicine. In 1932 he did postgraduate work in Vienna, studying and observing at the Ophthalmological Clinic.

He was on the staff at St. Catherine's Hospital and Brooklyn Eye and Ear Hospital. His society affiliations included King's County Medical Society, New York State Medical Society, American Medical Association, Brooklyn Ophthalmological Society, American Academy of Ophthalmology and Otolaryngology, the American College of Surgeons and the American Broncho-Esophagological Association. He was a diplomate of the American Board of Otolaryngology.

He served during World War I, and recently received a government citation for his work on the Selective Service Board during World War II.

Dr. Bruno died July 14, 1946. He is survived by his wife, Mrs. Jean D. Bruno, and four daughters.

## THOMAS EDWARD CARMODY

Dr. Thomas Edward Carmody, President of the American Academy of Ophthalmology and Otolaryngology in 1923, died August 30, 1946.

Dr. Carmody was born May 22, 1875, in Shiawassee county, Michigan and spent his early life in Owosso, Michigan. After receiving the degree of D.D.S. from the University of Michigan Dental School in 1897, he came to the University of Denver Dental College where he served as professor of bacteriology and histology. He continued his studies and was awarded the degree of M.D. in 1903 from the Denver and Gross College of Medicine and in 1908, he received his D.D.Sc. from the University of Michigan Dental School. Dr. Carmody was professor of oral surgery and rhinology at the University of Denver Dental College until 1932. At the medical school he served as assistant in laryngology and otology, and later as chief of otolaryngology, Child Research Council until 1936.

He was elected to the American College of Surgeons in 1913, American College of Dentists in 1929 and the International College of Surgeons in 1941. During World War I he served as a Major, Medical Corps, United States Army.

Dr. Carmody always gave freely of his time and effort to the various organizations of which he was a member. He held many offices in the local and state societies and served as first president of the reorganized Colorado Otolaryngological Society in 1919. He also served as president of the Colorado Society for Crippled Children, American Broncho-Esophagological Association, American Laryngological, Rhinological and Otological Society, the American Laryngological Association, and was a charter member of the American Board of Otolaryngology.

His contributions to publications included many articles covering diseases of the mouth, nose, throat and ears and a chapter, "Diseases of the Mouth and Tongue" which appeared in a volume by Jackson and Jackson, 1945. His courses in the Instructional Program sponsored by the Academy were very popular.

Surviving him are his widow; two daughters, Mrs. Ruth Carmody Sumners of Denver, Mrs. Mary Alice Carmody Cobb, Minneapolis; and a son, David W. Carmody, Santa Fe, New Mexico.

Dr. Carmody was respected for his skill and wisdom; his pleasant companionship and leadership are missed.

## HOWARD S. CLARK

Howard S. Clark was born in Auburn, New York, October 2, 1873. He received his preliminary education in Austin, Minnesota, taking his medical degree at the University of Minnesota in 1897. In 1913, he took postgraduate work in Berlin, Germany, continuing his studies during 1914 in Vienna, Austria.

For several years he was assistant professor of ophthalmology at the University of Minnesota. Dr. Clark was a member of the American Medical Association, the Minnesota Academy of Ophthalmology and Otolaryngology and the American College of Surgeons.

In Minneapolis he was a member of the Minneapolis Athletic Club, the Minneapolis Golf Club, and Rotary.

He died May 9, 1946 in Minneapolis, and is survived by one son, John Clark.

## ROBERT LEWIS CROCKETT

Dr. Crockett died May 27, 1946, aged 70, in Oneida, New York. Born in Sandy Creek, N. Y., Dr. Crockett received his preliminary education there, receiving his medical degree from Syracuse University Medical College. He began his practice in Sandy Creek in 1897. During this part of his career he also did postgraduate work at the New York Eye and Ear Infirmary, Manhattan Eye and Ear Hospital, the New York Post-Graduate Hospital, and Philadelphia Polyclinic Hospital.

In 1905, he moved to Oneida, where he served as ophthalmologist and pathologist at the Broad Street Hospital, posts which he held in the new City Hospital at the time of his death. He was Madison County bacteriologist and was originator of the county bacteriological laboratory established in 1907.

Dr. Crockett was active in civic affairs, serving as mayor of Oneida in 1916. He was a member of the Rotary Club, B. P. O. Elks, the Commandery and the Shrine, the Oneida Chamber of Commerce, American Legion, and a former member of the Oneida Board of Health. He was a member of the First Presbyterian Church. During World War I he served in the Army Medical Corps in France,

and at the time of his death held the rank of Colonel in the U. S. Medical Reserve Corps.

His medical affiliations were: the American Medical Association, Fellow of the American College of Surgeons, American Public Health Association, American Association for the Advancement of Science, American Academy of Ophthalmology and Otolaryngology, New York State Medical Society, Madison County Medical Society, and Oneida Doctor's Club.

His work as a naturalist, specializing in wild plant life and flowers, was of great interest to him. He was a member of three Syracuse University expeditions into South America, the Gaspé Peninsula, and Yucatan. He also made many field trips into the Adirondacks for unusual specimens. All together he collected over 8,000 specimens for the Museum Herbarium, furnishing also excellent Kodachrome slides of many plants.

Dr. Crockett is survived by his wife, two daughters, and one son.

---

## WARREN BEAGLE DAVIS

Born in Jessamine County, Kentucky September 6, 1881, Dr. Davis received his medical degree from Jefferson Medical College of Philadelphia in 1910. A diplomate of the American Board of Otolaryngology, his society affiliations included being a member of the founders group of the American Board of Plastic Surgery; member of the American Society of Plastic and Reconstructive Surgery, serving as a member of the board and as president in 1936-37; fellow of the American College of Surgeons, College of Physicians of Philadelphia, Academy of Surgery of Philadelphia, American Academy of Ophthalmology and Otolaryngology and the American Laryngological Association. He was also a member of the American Association of Anatomists and the Association of Military Surgeons of the United States.

During World War I Dr. Davis served on the medical advisory board of the War Department for Pennsylvania and later as Captain in the Medical Corps of the U. S. Army, where he had been in charge of the school of maxillofacial surgery at Camp Greenleaf, Fort Oglethorpe, Georgia. During World War II he was appointed by the Surgeon General to serve as a member of the National Faculty of Plastic and Maxillofacial Surgery.

Dr. Davis was a staff member of the Jefferson and Frankford hospitals and the Kensington Hospital for Women. He served as clinical professor of plastic and reconstructive surgery at his alma mater and in 1942 was made president of his alumni association. In 1938 he received an honorary degree of Doctor of Science from Georgetown College Georgetown, Kentucky. A member of the Pennsylvania state board of plastic surgery since 1941, he was also well known as editor of **Plastic and Reconstructive Surgery.** He died July 7, 1947.

---

## LOUIS SAMUELS DEITCHMAN

Dr. Louis S. Deitchman was born in Russia, September 23, 1903. He attended Ohio State University, University of Missouri, and the University of Pennsylvania Medical school, where he received his Doctor of Medicine degree in 1921. He did postgraduate work at New York Eye, Ear, Nose, and Throat Hospital and the Chicago Eye and Ear Hospital, starting his practice in Youngstown, Ohio in 1922.

Dr. Deitchman was on the staff of the Youngstown Hospital. He was associated with the Mahoning County Medical Society, Ohio State Medical Society, American Medical Association, Society of American Medical Historians, and the American Academy of Ophthalmology and Otolaryngology.

He was very much interested in the Revolutionary period of American medical history and wrote a series of articles on this period. He was also a collector of medical incunabula. He was editor of the Mahoning County Medical Bulletin, and a trustee of the Mahoning Tuberculosis Sanatorium.

He began active duty as a Major in the Medical Corps, Army of the United States, in August, 1942 and was separated from service with the rank of Lieutenant Colonel in April, 1946. He died September 4, 1946, aged 52. He is survived by his wife, Anna Galen Deitchman, three brothers and three sisters.

---

## WELLS PHILLIPS EAGLETON

Dr. Eagleton was born September 18, 1865, in Brooklyn, N. Y., son of Thomas Aston Proud and Mary Emma Phillips Eagleton of American and English ancestry and a descendent of Thomas Guy,

founder of Guy's Hospital, London, England.

He was graduated from the polytechnic Institute, Brooklyn, and in 1888 was awarded his medical degree by the College of Physicians and Surgeons of Columbia University. After an internship of two years at the Newark City Hospital he began the practice of medicine in Newark, New Jersey, in 1890. He was awarded a Doctor of Science degree from the University of Newark in 1942 and Rutgers University in 1943.

He was married May 24, 1913, to Mrs. Florence Peshine Riggs who survives him.

Dr. Eagleton was medical director of the Newark Eye & Ear Infirmary, secretary of the board of trustees and chief of cranial surgery. He was chief of the division of head surgery at Newark City Hospital. He served as consulting cranial surgeon at the following: St. Barnabas Hospital and Hospital for Women and Children; St. Michael's Hospital (Newark); Newark Memorial Hospital; Presbyterian Hospital( Newark); Beth Israel Hospital (Newark); Mountainside Hospital (Montclair); Muhlenberg Hospital (Plainfield); All Soul's Hospital (Morristown); Orange Memorial Hospital (Orange); Irvington General Hospital; Elizabeth General Hospital; and West Hudson Hospital of Kearny in addition to his duties as consulting ophthalmologist and otologist and cranial surgeon in numerous local and county hospitals.

In 1934, Dr. Eagleton was President of the American Academy of Ophthalmology and Otolaryngology. He was also a past president and member of the Essex County Medical Society (1908); Academy of Medicine of Northern New Jersey (1931); Academy of Medicine of New York; Medical Society of New Jersey (1923) (Chairman Welfare Committee 1923-24); American Otological Society (1921); New York Otological Society (1929); and the American Laryngological, Rhinological & Otological Society. He was a member of the American Medical Editors and Authors Association; A.M.A. (House of Delegates); American College of Surgeons (Fellow member Board of Governors); and associate member of the International College of Surgeons in which he was inducted in 1941. He held honorary membership in Sociedad Otorinoloargingingologica M a d r i l e n e, Spain; Academia Nacional de Medicine, Rio de Janerio, Brazil, 1936; Washington Medical and Surgical Society, Washington, D. C., Guy's Hospital, London, England; and Kansas City Society of Ophthalmology and Otolaryngology.

In World War I he volunteered in the United States Army, April 10, 1917, was commissioned a Major in the Medical Corps and served as chief of surgery of the head in the Base Hospital at Camp Dix, a post he held until after the signing of the Armistice. After the war he was made a Colonel in the Medical Reserve Corps, a commission he held until his death.

He was a member of the Essex County Country Club, Essex Club and Practitioners Club and the Browning Society of New York.

He contributed extensively to the medical periodical literature in his field. He was author of "Brain Abscess: Its Surgery, Pathology and Operative Treatment" 1922; "Cavernous Sinus Thrombophlebitis" 1925; (both translated into French); "Eagleton's Index"—Abstracts of progress in Intracranial Lesions related to Aural and Nasal Conditions 1941; and more than 100 monographs on cranial surgery.

In May, 1939, Dr. Eagleton was presented the Dr. Edward J. Ill Award by the Academy of Medicine of Northern New Jersey, the first to be given that honor. In 1940 he received the Award of Merit from the New Jersey State Medical Society, and in 1938 was awarded the Gold Medal by Kings County Medical Society, N. Y.

Dr. Eagleton was distinguished not only in his medical career but also as a citizen of his community. He was president of the Newark Council of Social agencies and a trustee of the Welfare Federation of Newark, New Jersey and of the Newark Museum. He was also a member of the New Jersey Commission for the Blind.

Dr. Eagleton's voice was frequently heard from the public platform and in the press, supporting health legislation, opposing political interference in medical matters, taking part in educational controversies and seeking to improve social welfare conditions in his city and state.

In 1941, Dr. and Mrs. Eagleton presented a building adjoining headquarters of the Academy of Medicine of Northern

New Jersey to the Academy to provide space for a larger library and for meetings.

Dr. Eagleton was deeply interested in international affairs and he and Mrs. Eagleton traveled to many parts of the world. On these journeys he always exhibited keen interest in medical and civic affairs of other nations and returned full of enthusiasm with knowledge gained while there.

Up to the very end of his long and brilliant career in his chosen speciality he contributed to the literature. His most recent articles were a complete review of advances in the understanding of rhinologic and otologic conditions related to the nervous system, published in the Archives of Otolaryngology for February, May, June and July, 1946. Dr. Eagleton was a man of strong convictions as to the trends and nature of medical practice, inclined usually to an intense individualism in his leadership.

Dr. Eagleton died September 11, 1946.

---

## CHARLES JOHN EDWARDS

Dr. Edwards was born in 1884. He attended Tulane University of Louisiana School of Medicine, receiving his M.D. degree from that institution in 1907.

Dr. Edwards practiced the specialty of treatment of diseases of the eye, ear, nose and throat in Vicksburg, Mississippi where he was on the staff of the Vicksburg Hospital, Mercy Hospital and Vicksburg Infirmary. He was a Life Fellow of the American Academy of Ophthalmology and Otolaryngology having been elected in 1913.

Dr. Edwards died March 13, 1947, aged 62.

---

## EDWARD COLEMAN ELLETT

Edward Coleman Ellet, son of Judge Henry T. and Katherine Coleman Ellett, was born in Memphis, Tennessee, December 18, 1869. He was educated in private schools in Memphis, attended for two years the Southwestern Presbyterian University at Clarksville, Tennessee (now Southwestern College at Memphis), and received his A.B. degree from the University of the South, Sewanee, Tennessee. His medical degree was received at the University of Pennsylvania in 1891 where he was also honored with the alumni medal for the highest scholastic average in his class. After a year as resident physician in St. Agnes Hospital, Philadelphia, and a year as house surgeon at Will's Eye Hospital, Philadelphia, he returned to Memphis in 1893, confining his practice to diseases of the eye, ear, nose and throat and since 1917 to diseases of the eye only.

In 1896 he married Nina Polk Martin and their mutual devotion for a half century made their union one of joy and beauty. Both interested in travel, she accompanied him on journeys to numerous medical meetings in this and foreign lands and before the late war on annual trips to Europe, especially France where he had served during World War 1. She was with him throughout his last illness.

Dr. Ellett was professor of ophthalmology in the medical department of the University of Tennessee from 1908 to 1922, and chief of staff of the Memphis Eye, Ear, Nose and Throat Hospital from its opening in 1926 until his death. He was also a member of the staff of the Methodist Hospital.

Dr. Ellett contributed generously to the medical literature on ophthalmology. His papers were mainly on clinical subjects, but his interest in research was keen as evidenced by his constant attendance at meetings of the Association for Research in Ophthalmology and participation in the discussions.

He wrote the chapter on "Lacrimal Diseases" in Berens' text book "Diseases of the Eye" and will long be remembered for his advocacy of intracapsular cataract extraction and the use of the corneal-scleral suture, a procedure which he introduced to this country after watching Kalt, and which he lived to see in almost universal use in some form or other.

No man has received more honors at the hands of his colleagues. In 1914 he served as chairman of the Section on Ophthalmology of the American Medical Association and was President of the American Academy of Ophthalmology and Otolaryngology in 1926 and of the American Ophthalmological Society in 1932. In addition he held executive positions in all local and sectional medical organizations having been president of the Memphis and Shelby County Medical Society, of the Tennessee Academy of Ophthalmology and Otolaryngology, and vice-president of the Southern Medical Association. He was active in the Ameri-

can College of Surgeons and in the Pan-American Congress of Ophthalmology, and was in constant attendance at sessions of the International Congress of Ophthalmology.

In November, 1939, the Southern Medical Association honored him with "Ellett Day" dedicated to a review of his achievements as a physician, citizen and soldier. In October, 1942, Dr. Ellett was Associate Guest of Honor of the American Academy of Ophthalmology and Otolaryngology and was given the Award of Merit in recognition of his services as President, Member of the Council and Instructor, as well as Scientist, Teacher and Soldier. In May, 1943, the Memphis and Shelby County Medical Society honored him with a testimonial dinner to celebrate his fiftieth year of medical practice in Memphis, at which were nine Memphis ophthalmologists who had been associated with him in private practice during those years.

For many years a member of the Board of Directors of the National Society for the Prevention of Blindness and a vice-president at his death, he gave freely of his time to the labors of this lay organization in the field of sight-conservation. In 1939 in cooperation with the St. Louis Society for the Blind he was given the Leslie Dana Medal "for outstanding achievements in the prevention of blindness and the conservation of vision."

A member of the original American Board for Ophthalmic Examinations he helped to conduct the first examinations for certification in ophthalmology at the first meeting in Memphis some thirty years ago. This interest was maintained to the end and he continued to review candidates' examination papers until last year when he retired from the American Board of Ophthalmology after serving as president for several years. This, too, was the occasion of a testimonial by the Board.

During World War I, Dr. Ellett served in France as commanding officer of Base Hospital 115 with the rank of Lieutenant Colonel and received a citation for "exceptionally meritorious and conspicuous service." Discharged as a full Colonel in the Medical Corps of the United States Army Reserve he has been known to all his Memphis colleagues as "The Colonel," a term of honor, endearment and respect, which he accepted modestly.

In June, 1942, he received the honorary degree of LL.D. from Southwestern College, and in September, 1943, the honorary degree of D.Sc. from the University of the South.

His gentleness and kindly interest in his patients was rewarded by an enormous practice which he was delighted to share with his associates. A deft surgeon, his inclinations were always toward the simplest and safest procedures. In the past two years his practice was limited to ophthalmic consultation and surgery but his manual dexterity and steadiness was maintained to the very end and was praised by all who watched him.

---

## RAYMOND EDWIN ELLIOTT

Dr. Raymond E. Elliott was born November 9, 1890 in Rochester, N. Y. After attending public schools there, he went to McGill University where he obtained his Doctor of Medicine degree in 1915. After postgraduate work in New York and Vienna, Dr. Elliott returned to Rochester to practice. A staff member of St. Marys Hospital, he was a diplomate of the American Board of Otolaryngology, a member of local, county and state medical organizations as well as a Fellow of the American Academy of Ophthalmology and Otolaryngology. Dr. Elliott was active in Masonic circles, civic and fraternal clubs of Rochester.

His death occurred March 4, 1947. He is survived by his widow, Mrs. Edna Loeffler Elliott, and a son, Raymond F. Elliott.

---

## GEORGE FREIMAN

Dr. George Freiman died January 3, 1947 at the Long Island College Hospital, Brooklyn, New York, where for many years he had been a member of the staff. Dr. Freiman received his medical degree from Long Island College in 1914, taking postgraduate work at Columbia University. In 1915, he began the practice of medicine at the Long Island College Hospital, where at the time of his death he was clinical professor of ophthalmology.

Dr. Freiman was a Fellow of the American Academy of Ophthalmology and Otolaryngology, the American College of Surgeons, and a diplomate of the American Board of Ophthalmology. He was a member of the King's County Medical Society, the Brooklyn Ophthalmological Society, and various other societies.

In 1942-1943 he was president of the Alumni Association of the Long Island College of Medicine, in which association he had taken a very active part. During World War I, he served as a Lieutenant in the Medical Corps.

Dr. Freiman is survived by his wife, Dr. Betty C. Gordon, a practicing ophthalmologist, and two sisters, Mae and Eve Freiman.

---

EDWARD FROTHINGHAM

Dr. Frothingham was born in 1893 in Brooklyn, New York. Before taking up the study of medicine at the New York Homeopathic Medical College, he attended the New Jersey College of Pharmacy where he received his Ph.G., and then served four years as a hospital steward in the United States Navy. Dr. Frothingham attained his medical degree in 1918 and interned at Flower Hospital. Commissioned as acting assistant surgeon, USNR, in 1918, he became particularly interested in eye, ear, nose and throat work. Upon his release from service in 1921, he became associated with the Manhattan Eye, Ear, Nose and Throat Hospital. Other hospital appointments included attending ophthalmologist at Flushing, attending otolaryngologist at Mary Immaculate in Jamaica, and a member of the attending staff at Brooklyn Eye and Ear Hospital.

Dr. Frothingham was a diplomate of both specialty boards. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and the American College of Surgeons. In 1933, he received his degree of Bachelor of Science from New York University. He was a Mason and a Shriner.

Dr. Frothingham died March 22, 1947. Surviving are his wife, Irene O'Hare Frothingham, four sons and a daughter.

---

CHARLES WILLARD GEIGER

Dr. Charles W. Geiger, 75, a practicing physician in Kankakee, Illinois, for more than forty years, died July 19, 1946.

Born at Paxton, Illinois, he was in practice with his brother, the late Dr. Louis Geiger, at Gilman, Illinois, for about ten years before establishing himself in Kankakee. He was a graduate of Iowa State University, Chicago Homeopathic Medical College, (1896), and the University of Illinois College of Medicine (1904).

He was a member of the American Academy of Ophthalmology and Otolaryngology and past president of the Kankakee County Medical Society. Through his extensive practice and long years of active participation in civic endeavors, Dr. Geiger was well known throughout northeastern Illinois. He was a former president of both the Chamber of Commerce and the Rotary Club, and a member of the Kankakee County Tuberculosis Sanatorium Commission.

He is survived by his wife, Mrs. Viola Geiger, and a sister, Miss Minnie J. Geiger of Chicago.

---

ROBERT GEWANTER

Dr. Robert Gewanter, aged 44, passed away November 19, 1946, at his home in Long Island City, N. Y. Born in New York City, Dr. Gewanter had received his medical degree from the New York University College of Medicine in 1926 and interned at Bellevue Hospital before establishing his practice in 1929. He instructed in otorhinolaryngology at New York University for many years and was on the staffs of Greenpoint Hospital and Bellevue. Certificated by the American Board of Otolaryngology in 1934, Dr. Gewanter was a member of the Queens County and Long Island Medical Societies and a Fellow of the American Academy of Ophthalmology and Otolaryngology. His hobbies were gardening and woodworking. In his leisure time Dr. Gewanter turned out expert carving and inlay work in mahogany and walnut woods.

Surviving him are his wife, Esther Gast Gewanter, and two sons, Robert and Theodore.

---

MATTHEW GERARD GOLDEN

Born in New York City, March 13, 1894, Dr. Golden was educated at Brooklyn Prep. and Colby College and received his degree of Doctor of Medicine from Fordham University School of Medicine in 1919. Limiting his practice to diseases of the ear, nose and throat, he opened an office in Brooklyn in 1921.

Dr. Golden was associate clinical professor of otolaryngology at Long Island College Hospital, attending otolaryngologist at Kings County Hospital, St. Peter's Hospital, Bay Ridge Hospital and consulting otolaryngologist at Midwood Hospital.

Certificated by the American Board of Otolaryngology, Dr. Golden was a Fellow of the American Academy of Ophthalmology and Otolaryngology, the American College of Surgeons, and a member of the Kings County and the New York State medical societies. Dr. Golden was a founder and the first managing editor of the "Linacre Quarterly" established in 1932.

Dr. Golden died November 11, 1946. His wife, Evelyn Drew Golden, and three children survive him.

---

EDGAR LEONARD GOODMAN

Dr. E. L. Goodman was born July 4, 1902 in Washington, D. C. He received both his literary and medical degrees from George Washington University. His internship was served in Garfield Hospital, followed by a residency at Wills Hospital, Philadelphia. Returning to Washington in 1933, Dr. Goodman became associated with the Episcopal Eye, Ear, Nose and Throat Hospital of which he was acting chief from 1942-46, Garfield Hospital, Doctors Hospital, and Gallinger Hospital. He was also on the teaching staff of George Washington University School of Medicine. In addition to being a diplomate of the American Board of Ophthalmology, and a Fellow of the American Academy of Ophthalmology and Otolaryngology, Dr. Goodman belonged to the Southern Medical, Washington, D. C. Ophthalmological, Washington Medical and Surgical, and Macknall societies. He was an ardent boatsman and fisherman and a member of the Mile River Yacht Club.

Dr. Goodman died February 28, 1947. He is survived by his widow, Isobel Goodman, and a son.

---

LOUIS RICHARD HAAS

Dr. L. R. Haas was born March 29, 1903, in Union City, New Jersey. He was graduated from the Cornell University Medical College in 1929, and continued his medical training at the hospital of the New York Society for the Ruptured and Crippled, the Lenox Hill Hospital, the Bellvue Hospital, and the Manhattan Eye and Ear and the Brooklyn Eye and Ear hospitals.

In 1938, Dr. Haas moved to Pittsburg, Kansas where he was associated with the Smith Clinic as an eye, ear, nose and throat specialist.

A diplomate of the American Board of Ophthalmology, Dr. Haas was a member of the American College of Surgeons, the International College of Surgeons, and a Fellow of the American Academy of Ophthalmology and Otolaryngology. He was the author of an article on allergy recently published in the "Eye, Ear, Nose and Throat Monthly."

Dr. Haas died June 30, 1947. Survivors include his father, his wife, Anne E. Haas, and two sisters.

---

GAYLORD CRAWFORD HALL

Dr. G. C. Hall was born in Louisville, Kentucky, July 22, 1879. He received his degree of Doctor of Medicine from the University of Louisville in 1902. After internship at Vanderbilt Clinic of Columbia University, New York, he began his practice in Louisville. From 1908 to 1915 he served as professor of diseases of the eye, ear, nose, and throat at the University of Louisville School of Medicine. He was a member of the original staff at Kentucky Baptist Hospital and was on the St. Anthony Hospital Staff for forty years.

Dr. Hall was a pioneer in bronchoscopic work in Kentucky. He was one of the founders of the League for the Hard of Hearing and the workshop at the Kentucky School for the Blind.

His professional affiliations included the American, Kentucky State, and Jefferson County Medical Associations, the American Academy of Ophthalmology and Otolaryngology, and the Louisville Eye, Ear, and Nose Association of which he was past president.

In Louisville he was a member of the Masons, the Pendennis and Louisville Country Clubs, and a member of St. Mark's Episcopal Church.

Dr. Hall died June 16, 1946 in Louisville. He is survived by his wife, Gertrude L. Hall, a daughter, Dorothy, a son, Gaylord C., and a grandson, Gaylord C. Hall, III.

---

EDWARD PETER HALTON

Dr. Halton of Holyoke, Massachusetts was born in 1882. He was graduated from Yale University School of Medicine in 1905. An otolaryngologist, he was associated with Holyoke and Providence hospitals. His society affiliations included the Massachusetts Medical and the New England Otolaryngologic socie-

ties as well as the American Academy of Ophthalmology and Otolaryngology of which he became a Fellow in 1937. Dr. Halton was certificated by the American Board of Otolaryngology. His death occurred October 18, 1944.

---

HOWARD EUGENE HAMILTON

Dr. Hamilton was born March 10, 1911. An ear, nose, and throat specialist, he had resided in Cleveland, Ohio since his graduation from the University of Kansas School of Medicine in 1934. He served residencies at the City Hospital of Cleveland and the University Hospital of Cleveland, was otologist to the State Hospital and a demonstrator in the department of otolaryngology, at Western Reserve University. Dr. Hamilton belonged to the Cleveland Academy of Medicine, the Cleveland Otolaryngology Club, the Ohio State Medical Society, and became a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1942.

Dr. Hamilton died December 27, 1946. He is survived by his parents, several brothers and sisters, his widow, Blanche Wilcox Hamilton, and two daughters.

---

MORRIS LOUIS HARRIS

Dr. Harris, born in Russia, in 1887, obtained his preliminary education in the New York city schools and received his medical education at New York University College of Medicine and Bellevue, 1909. His first appointment was as house surgeon at Beth Israel where he later became an associate on the ear, nose and throat service. Dr. Harris was attending bronchoscopist at Israel Zion and an instructor at Long Island College Hospital Medical School. A diplomate of the American Board of Otolaryngology, Dr. Harris included endoscopy and plastic surgery in his private practice. He was a member of Kings County Medical Society and the American Academy of Ophthalmology and Otolaryngology. Both Dr. Harris and his wife were active in affairs of the Shaari Zedek Temple. He was also a contributor of numerous articles to medical journals and conducted a column of medical advice in the "Jewish Examiner."

Dr. Harris died November 28, 1946 at his home in Brooklyn. His wife and two sons survive him.

JOHN MARVIN INGERSOLL

We record with deep regret the death of Dr. John M. Ingersoll on January 16, 1947, at his home on South Bayshore Lane, Miami, Fla. Dr. Ingersoll passed away after a long illness at the age of 77.

Dr. Ingersoll was born in 1869 in Youngstown, Ohio, and was graduated from Western Reserve University in 1893. He did his postgraduate work in Europe and studied under most of the great masters of otolaryngology of his day. He then returned to Cleveland, Ohio and became professor of otolaryngology at Western Reserve University, a post he filled until 1926 when he resigned and retired to live in southern Florida. He was appointed emeritus professor of otolaryngology in 1926.

Dr. Ingersoll leaves his wife, Mrs. Nora Ingersoll, a son, and a daughter.

Always one of the leaders of his specialty, he took great interest in developing younger men and many of the well known nose and throat men of the United States had their training under Dr. Ingersoll. Among the many honors that were bestowed upon him were the presidencies of two great societies in his specialty. He was President of the American Academy of Ophthalmology and Otolaryngology in 1919, president of the American Laryngological, Rhinological and Otological Society and secretary of the American Laryngological Association. He was a fellow of the American College of Surgeons and a member of the American Otological Society.

During his active clinical life he was interested in all phases of the specialty but he gained national renown for his research studies in a comparative anatomy of the ear, nose and throat. His dissections of the heads of vast numbers of animals marked him as a pioneer in comparative anatomy.

During World War I, Dr. Ingersoll left his professorship at Western Reserve and entered the Medical Reserve Corps of the United States Army. He served throughout the war in charge of some of the largest ear, nose and throat services in the Army. It was his request that he be buried in Arlington Cemetery.

---

GEORGE BAIGRIE JOBSON

Dr. George Jobson was born in Hawick, Scotland in 1869. He came to this country with his parents when a small boy

He was educated in schools in New York and Pennsylvania, was graduated from George Washington University, and in 1900 was graduated from the Harvey Medical College in Chicago. In 1922, he did postgraduate work at the University of Vienna.

Dr. Jobson began his practice of medicine in Franklin, Pennsylvania, in 1902. His more than forty years of practice centered in Franklin and Oil City. He was one of the first surgeons to develop plastic surgery, and his proficiency in this line won him national recognition, receiving a citation from the American Board of Plastic Surgery.

His interest in civic affairs included the Oil City and Franklin Rotary Clubs, School Board, Boy Scouts, and Y.M.C.A. He was a member of the First Presbyterian Church. Dr. Jobson was an enthusiastic and capable hunter. In Arizona he participated in a mountain lion hunt, killing a large mountain lion and obtaining remarkable pictures of the hunt.

He was a Fellow of the American Academy of Ophthalmology and Otolaryngology, member of the American College of Surgeons, American Medical Association, American Medical Editors and Authors Association, Founders Group of the American Board of Plastic Surgery, the Venango County Medical Society, of which he was former president. He was also a former president of both the Pittsburgh Otological Society and the Eye, Ear, Nose, and Throat Section of the Pennsylvania State Medical Society.

Dr. Jobson died at his home in Franklin, October 21, 1946. He is survived by his wife, Elizabeth Taylor Jobson, a son, George Blaikie, and two daughters, Jean and Marian Jobson.

## THOMAS OTIS KLINGNER

Dr. T. O. Klingner, eye, ear, nose and throat specialist of Springfield, Mo., who died January 7, 1947, at age 72, was widely known for his philanthropic work. As a life member of the Kiwanis Club, he became interested in its program of social service with young people and is said to have performed more than 200 tonsillectomies without charge for students at the School of the Ozarks at Hollister, Mo.

He was a farm boy born near Fair Grove, Mo., and educated at Morrisville College and Missouri Medical Academy at St. Louis where he received his medical degree in 1898. He did postgraduate work at Tulane and Chicago universities. His practice began at Willard, Mo., was continued in Joplin, and during most of his career, in Springfield. For three years he served in the eye, ear, nose and throat section of the Federal Bureau of Pensions, and for 15 years as physician for the Frisco Railroad. He was also an examiner for pensions for the blind in Missouri.

Dr. Klingner's professional connections included the following: Senior Member, American Academy of Ophthalmology and Otolaryngology; the old Southwest Missouri Medical Society; and the Greene County Medical Society, which he served as secretary from 1912-19 and as president in 1920. In 1946 he was made an Honor Member of the latter organization. He was well known in Masonic circles in Joplin and Springfield as a 32nd degree Mason and a Shriner.

Surviving are his widow, Effie May Klingner, a daughter, Mary E. Klingner Reed, three brothers and a sister.

## ROBERT K. LAMBERT

Dr. Lambert died October 3, 1947 in New York City, at the age of 47. Born in New York City in 1898, he attended Columbia University, receiving his degree of Doctor of Medicine from the Columbia University College of Physicians and Surgeons in 1922.

Dr. Lambert was clinical professor of ophthalmology at Columbia. He was chief of the eye service at the Mount Sinai Hospital, where he served an internship and residency. At Montefiore Hospital for Chronic Diseases he was attending surgeon in charge of ophthalmologic service, and later, consulting ophthalmic surgeon. He served during World War I, and as a Major in the Medical Corps during World War II.

His society affiliations included the American Academy of Ophthalmology and Otolaryngology, American College of Surgeons, New York Academy of Medicine, American Medical Association, and Association for Research in Ophthalmology.

Dr. Lambert was interested in music and painting and published several pa-

pers, most of which appeared in ophthalmic journals.

He is survived by his wife, Gerda, and a daughter, Patricia.

## FRANKLIN FAYETTE LANE

Franklin Lane, Captain (MC) U. S. Navy, retired, was born in Elm, N. J. on August 21, 1888. A graduate of the University of Pennsylvania School of Medicine in 1915, he entered the Medical Corps of the U. S. Navy as a Lieutenant (jg) in 1917 and served actively for over twenty-nine years. Captain Lane was certificated by both the American Board of Ophthalmology and American Board of Otolaryngology, was a member of the American College of Surgeons and a Fellow of the American Academy of Ophthalmology and Otolaryngology since 1933. He was a member of the Naval Retiring Board, Navy Department in Washington, D. C. Captain Lane died January 28, 1947, and is survived by his wife, Agnes Ashford Lane.

## WALTER IVAN LILLIE

Dr. W. I. Lillie, professor and head of the department of ophthalmology at the Temple University School of Medicine, was born in Grand Haven, Michigan, November 5, 1891. Following his graduation from the University of Michigan Medical School in 1915, Dr. Lillie held a fellowship at the Mayo Foundation. He became first assistant in ophthalmology at the Mayo Clinic in 1917, and later associate in ophthalmology at the Clinic and associate professor of ophthalmology at the Foundation, which positions he held until accepting the appointment at Temple University in 1933.

Dr. Lillie served in the British and American Expeditionary forces in France during World War I.

Certificated by the American Board of Ophthalmology, Dr. Lillie was a member of the American Ophthalmological Society, the Association for Research in Ophthalmology, and a fellow of the American College of Surgeons. At the time of his death, Dr. Lillie was serving as a councillor of the American Academy of Ophthalmology and Otolaryngology. He was a frequent contributor to the ophthalmologic literature and a popular speaker. His lectures on the Academy instruction program were always in great demand.

In addition to his professional duties at Temple University, Dr. Lillie was consulting ophthalmologist at the Temple University Hospital, Shriners Hospital for Crippled Children in Philadelphia, Eagleville ( Pa.) Sanatorium for Consumptives and the Norristown (Pa.) State Hospital.

Dr. Lillie died February 21, 1947. He leaves his wife, Opal Jones Lillie, and one son, Robert.

## JAMES ORVILLE MACDONALD

Dr. James MacDonald was born in Ontario, Canada, April 17, 1892. After attending schools in Strathroy, Ontario, he went to Queen's University in Kingston and obtained his degree of Bachelor of Science in mining engineering as well as his medical degree. He engaged in mining engineering in northern Ontario for a short time before going overseas with the Canadian Medical Corps in World War I.

Dr. MacDonald undertook postgraduate work at Columbia University. He became associate clinical professor of otolaryngology at the New York Post-Graduate Medical School and Hospital there, and served as attending otolaryngologist to the hospital and dispensary.

Certificated by the American Board of Otolaryngology, Dr. MacDonald was a Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons. During World War II he served as a Lieutenant Commander in the Medical Corps of the U. S. Naval Reserve.

Dr. MacDonald died suddenly in Savannah, Georgia, January 13, 1947. Surviving are his mother, Mrs. Mary MacDonald of Toronto, Ontario, and four sisters.

## CHARLES ALFORD MAGHY

Dr. C. A. Maghy was born in Lucan, Ontario, in 1883. He was graduated from the University of Illinois School of Medicine in 1905 and after 1912 limited his practice to diseases of the eye. At that time he was serving an internship at the Chicago Charitable Eye and Ear Infirmary. In 1916 he went to England and studied at Moorsfield Eye Hospital, London where he remained as pathologist until 1919. Upon his return to this country, Dr. Maghy practiced in Los Angeles and at the time of his death, in San Diego. He was a member of the London Oph-

thalmological Society, the Oxford Ophthalmological Congress, the Ophthalmological Society of Egypt, the Chicago Ophthalmological and Pacific Oto-Ophthalmologic societies, and the California State and American Medical Associations. He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and a diplomate of the American Board of Ophthalmology.

His death occurred April 27, 1947.

### JOHN JOSEPH McDERMOTT

Born in Hubbardston, Michigan in 1887, Dr. McDermott was educated at Ferris Institute, Big Rapids, Mich., and received his Doctor of Medicine degree from the University of Michigan Homeopathic Medical School in 1914. Postgraduate work was done at the New York Ophthalmic Hospital and in Vienna. He started his practice in St. Joseph, Mich. in 1920 where he was associated with the St. Joseph Sanitorium and with Mercy Hospital in Benton Harbor.

Dr. McDermott was a member of the Michigan Triological Society, the American College of Surgeons and one of the founders of the Southwestern Michigan Academy of Medicine. He was a diplomate of the American Board of Otolaryngology and a Fellow of the American Academy of Ophthalmology and Otolaryngology.

His interests other than medicine were many. Dr. McDermott was president of the St. Joseph Library Board, vice president of the Twin City Concert Association, district chairman of the Boy Scouts, and director of the St. Joseph Chamber of Commerce. He was active in Kiwanis, Knights of Columbus, and the Catholic church. In World War I, he served with the rank of Captain.

Dr. McDermott's death occurred in Chicago, March 19, 1947. Surviving him are his wife, Margaret L. McDermott, three sons and 2 daughters. A fourth son was killed in action in World War II.

### THOMAS D'ARCY McGREGOR

Born in Schreiber, Ontario, Canada, October, 1893, Dr. McGregor attended Woodstock College, Ontario, and received his degree of Doctor of Medicine from McGill University in 1918. His postgraduate work was done at Manhattan Eye, Ear, Nose, and Throat Hospital. He began his practice, in 1920, in Buffalo, New York.

At the University of Buffalo School of Medicine, he was an associate in otolaryngology; was assistant otolaryngologist at the Buffalo General Hospital; and assisting attending otolaryngologist at the Children's Hospital. During World War I he was a Captain in the Canadian Army.

Dr. McGregor was a Fellow of the American Academy of Ophthalmology and Otolaryngology, diplomate of the American Board of Otolaryngology, and a member of the Medical Council of Canada, Medical Society of State of New York, Medical Society of Erie County, and the American Medical Association.

His wife, Mrs. Evelyn McGregor, and a daughter, Catherine D'Arcy, survive him.

### CLAUDE WORTH McKEE

Dr. Claude McKee died at his home in Greensburg, Pa., September 11, 1946. A veteran of World War I, Dr. McKee was born at Cochran Mills, Pa., April 1, 1876. He is survived by his wife, Mrs. Anna McKee, and three children: Mrs. Dorothy McKee Pershing, Mrs. Betty McKee Brinker, and Worth Humphries McKee.

Dr. McKee was graduated from Rush Medical College in Chicago, in 1897 and did postgraduate work at the Royal Ophthalmic Hospital in London and at the University of Vienna under Drs. Reutan and Noyman, and in London at the Eye and Ear Hospital.

He belonged to the American Academy of Ophthalmology and Otolaryngology, the State Medical Society of Pennsylvania, and was a charter member and one of the organizers of the Western Pennsylvania Eye, Ear, Nose, and Throat Society.

During World War I he was attached to a base hospital in France. He was a member of the American Legion, Veterans of Foreign Wars, Masons, and for many years served as chairman of the advisory board of the Salvation Army. He was a member of the First Methodist Church of Greensburg.

### J. HALLOCK MOORE

Dr. J. Hallock Moore of Huntington, W. Va., died suddenly August 3, 1947. Dr. Moore was considered an outstanding otolaryngologist and performed most of the bronchoscopy in the tri-state area surrounding Huntington.

Dr. Moore was born in 1902, attended the Huntington schools, was graduated from Sewanee Military Academy in 1920, received his B. S. degree from Lafayette College and his medical degree from the University of Iowa in 1929. He spent four years in otolaryngology at Bellevue Hospital and then returned to his home to practice his specialty. He was on the staffs of Memorial Hospital and St. Mary's Hospital, where he was chairman of the department of otolaryngology and ophthalmology, and was a consultant in otolaryngology at the Veterans' Hospital.

Dr. Moore was president of the West Virginia Academy of Otolaryngology, a member of the American Laryngological, Rhinological and Otological Society, the American Medical Association, the West Virginia and Cabell County Medical Associations, and a Fellow of the American Academy of Ophthalmology and Otolaryngology as well as a diplomate of the American Board of Otolaryngology. Dr. Moore wrote several papers on Meinere's syndrome and on insufflation of the eustachian tube.

---

EDWARD RODGERS NEEPER

Dr. Neeper, a resident of Colorado Springs, Colorado since 1898, was born October 8, 1867, in Kahoka, Missouri and was graduated from the Keokuk (Iowa) Medical College in 1891.

Dr. Neeper was prominent in the medical and lodge circles of Colorado Springs. One of the original members of the Colorado Ophthalmological Society, he was also a member of the Colorado Springs Clinical Club, the El Paso County Medical Society, the Elks and other social organizations. He became a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1918.

Dr. Neeper's death occurred November 21, 1946. He is survived by his wife, Mrs. Amelia Neeper, a brother, and a sister.

---

WILLIAM EDWARD PATTERSON

Dr. William Patterson, 71, died in Minneapolis, Minnesota, October 30, 1946. He attended public schools in Clarksville, Iowa, the University of Iowa, and took his degree of Doctor of Medicine from the University of Illinois in 1900. His postgraduate work was done at the New York Eye and Ear Infirmary from 1914-1916.

After practicing in Iowa from the time of his graduation Dr. Patterson went to Minneapolis in 1917, where he was associated with Dr. Frank Todd until his death in 1918 after which he became associated with the Eye, Ear, Nose, and Throat Clinic. He served as assistant professor of ophthalmology at the University of Minnesota Medical School and the graduate school, and was ophthalmologist for the Milwaukee and St. Louis R. R. He was on the staffs of Asbury and St. Barnabas Hospitals.

His society affiliations included the American Medical Association, American College of Surgeons, American Academy of Ophthalmology and Otolaryngology, Minnesota State and Hennepin County Medical societies. He was a former vice-president of the Hennepin County Medical Society.

Dr. Patterson is survived by his wife, a daughter, Mrs. J. Webb Hedin, and a son, Max E., all of Minneapolis.

---

HOWARD WILBER PEIRCE

Dr. Howard Peirce of Detroit was born in 1879. A graduate of Detroit College of Medicine, now known as Wayne University College of Medicine, in 1904, Dr. Peirce became assistant professor of clinical otolaryngology of that institution. Certificated by the American Board of Otolaryngology, a Fellow of the American Academy of Ophthalmology and Otolaryngology, he was also a past president of the Detroit Otolaryngological Society and on the staff of the Harper Hospital.

Dr. Peirce died April 17, 1946.

---

HAROLD VINCENT PHELAN

Dr. Harold Phelan died September 1, 1946 in Cleveland, Ohio. Born in Ashford, New York in 1895, Dr. Phelan took his preliminary schooling at Niagra University and at Georgetown University, receiving his degree of Doctor of Medicine from the latter school. In 1924, he did postgraduate work at the University of Vienna, returning to Cleveland, Ohio to begin his practice.

At Western Reserve University, Dr. Phelan was demonstrator in ophthalmology. For sixteen years, he was ophthalmologist at St. Flexis Hospital in Cleveland.

His society affiliations included Academy of Medicine of Cleveland, Ohio State Medical Association, Cleveland Medical Library Association, American Medical

Association, American Medical Association of Vienna, American College of Surgeons, American Academy of Ophthalmology and Otolaryngology, and the Cleveland Ophthalmological Club. He was a diplomate of the American Board of Ophthalmology.

Dr. Phelan was an ardent fisherman and golfer, and was also a member of the Cleveland Skating Club.

He is survived by his wife, Germaine Winters Phelan, a son, Harold, and a daughter, Ann.

---

ALBERT ERNEST RECTOR

Born in 1873 in Fennimore, Wisconsin, Dr. Rector was graduated from Rush Medical College in 1879, and first began practice in Lake Park, Iowa. He later made his home in Appleton, Wisconsin where he was a member of the staff and executive committee of St. Elizabeth Hospital. Dr. Rector continued his studies of diseases of the eye, ear, nose and throat in Chicago, New York, London and Vienna. He was a diplomate of the American Board of Otolaryngology, a Life Fellow of the American Academy of Ophthalmology and Otolaryngology, past president of his county and state medical societies, and fellow of the American College of Surgeons, as well as a member of the Wisconsin State Board of Health.

Dr. Rector died in Appleton, January 13, 1947, leaving his wife, Emma M. Rector, and two sons.

---

CHARLES TIFFANY ST. CLAIR

Dr. St. Clair of Bluefield, West Virginia, was born in 1873. He was graduated from the University of Virginia Department of Medicine, Charlottesville, in 1896. Certificated by the American Board of Otolaryngology in 1929, Dr. St. Clair became a Fellow of the American Academy of Ophthalmology and Otolaryngology in 1930. He was also a member of the American College of Surgeons, the West Virginia Medical Society, Mercer County Medical Society, and Southern Medical Association. He was on the staff of the Bluefield Sanitarium.

Dr. St. Clair passed away November 17, 1946.

---

G. WILLIAM SCHLINDWEIN

Dr. G. William Schlindwein, aged 73, died March 19, 1946 in Rome City, Indiana. Born in Erie, September 8, 1872, he attended parochial schools there and later attended Jefferson Medical College, Philadelphia, where he was graduated in 1904. He interned at Wills Hospital, Philadelphia and then returned to Erie to practice as a physician and surgeon. In 1912 he took postgraduate work in Berlin and Vienna, also studying at Zurich, Switzerland, and Barcellona, Spain.

He belonged to the Erie County Medical Society, Pennsylvania State Medical Society, American College of Surgeons, American Academy of Ophthalmology and Otolaryngology, and the American Medical Association.

Aside from his professional work, Dr. Schlindwein's interests centered on fine pieces of art, photography, and traveling. He acquired an art collection of considerable value, much of which he gave to the Public Museum, Cathedral Prep, St. Joseph's Home for Children, and to the two hospitals where he contributed so much professionally and administratively, St. Vincent's and Hamot. His philanthropies were many, the latest being the gift of a summer home to the Sisters of St. Joseph as a summer retreat. He was one of the founders of the Orphan's Welfare Association in Erie and was serving as president at the time of his death. Active in the Knights of Columbus and similar groups, in 1931, Pope Pius XI conferred the high honor of Knight of St. Gregory upon Dr. Schlindwein in recognition of his outstanding accomplishments, notably of a charitable nature.

Dr. Schlindwein is survived by his wife, Rose Conrath Schlindwein.

---

WILLIAM AUGUSTUS SCHWARTZ

Born in 1885, Dr. Schwartz received his degree of Bachelor of Science from Ohio Northern University and was graduated from Northwestern University Medical School in 1911. A resident of Phoenix, Arizona, Dr. Schwartz had limited his practice in that city to diseases of the eye, ear, nose and throat since 1916. He was certificated by the American Board of Ophthalmology in 1920, was elected to Fellowship in the American Academy of Ophthalmology and Otolaryngology in 1928, and was a member of his county and state medical societies, the American Medical Association, and the American College of Surgeons.

Dr. Schwartz' death occurred August 9, 1945, in Phoenix.

## THOMAS HALL SHASTID

Dr. Thomas H. Shastid, Duluth, Minnesota, ophthalmologist and author, died February 15, 1947, in that city. Born in Pittsfield, Illinois, July 19, 1866, he became his father's medical apprentice at the age of ten years.

He studied at Columbia University and received his degree of Doctor of Medicine with honors from the University of Vermont College of Medicine in 1888. He did postgraduate work at the University of Vienna, received a Bachelor of Arts degree with honors from Harvard in 1893, and added Master of Arts and Bachelor of Laws degrees to his scholastic achievements at the University of Michigan. In 1922 the University of Wisconsin awarded him an honorary Doctor of Science degree.

From 1907 to 1912, Dr. Shastid was professor of history of medicine at American Medical College, St. Louis, serving later as honorary professor. Since 1914 he had specialized in ophthalmology, maintaining offices in Duluth. He was certificated by the American Board of Ophthalmology.

He was a member of the American College of Physicians, American Medical Association, American Academy of Ophthalmology and Otolaryngology, American College of Surgeons, National Society for the Prevention of Blindness, History of Science Society, International Academy of History of Medicine, and various other state, national, and international societies.

Dr. Shastid was author of many books on medicine, a number of novels, one on wartime profiteering, several works on the subject of international peace, and two autobiographies. He translated Helmholtz's "Description of an Ophthalmoscope" and numerous other medical volumes from German, French, Latin, and Greek. He collaborated with Dr. H. A. Kelly on "Cyclopedia of American Medical Biography" (1912) and "American Medical Biographies" (1920); with Dr. J. M. Ball on "Modern Ophthalmology"; and with Dr. Conrad Berens on "The Eye and its Diseases" (1936). He wrote more than 3,000 articles for the American Encyclopedia of Ophthalmology. He was former editorial secretary of the "Ophthalmic Record," former associate editor of the "Michigan Law Review," and served on the staff of "Eugenics" magazine, along with being a frequent contributor to scientific journals of all kinds.

Dr. Shastid is survived by one daughter, Mrs. Donald R. Husband, Eugene, Oregon, and two brothers, John S. and Joseph.

---

## SAMUEL ANISE SHOEMAKER

Dr. Shoemaker was born in 1866. He attended the Medical College of Indiana in Indianapolis, where he received his degree of Doctor of Medicine in 1898. He practiced for some time in Bluffton, Indiana, and then moved to Orlando, Florida. In 1938 he was the victim of an automobile accident which forced him to retire from the practice of medicine. While in Florida he was made an honorary member of the Florida Medical Association. He was elected to Fellowship in the American Academy of Ophthalmology and Otolaryngology in 1917 and was an active Fellow of the Academy for 29 years.

Dr. Shoemaker died in Muncie, Indiana, on November 6, 1946, at the age of 80 years.

---

## WAYNE PULLEY SIRLES

Born in Carterville, Illinois, October 8, 1912, Dr. Sirles attended school in Herrin, Illinois, received his degree of Doctor of Medicine from the College of Medicine of the University of Illinois in 1938. He interned at City Hospital in St. Louis, Missouri.

Dr. Sirles served a residency at the Barnes Hospital in St. Louis, where he had been instructor in otolaryngology at the Washington University School of Medicine. During World War II he was in the Medical Corps of the Army Air Force. In 1946, Dr. Sirles reopened his office in Herrin and limited his work to diseases of the ear, nose and throat. He was on the staff of Herrin Hospital, and the Veteran's Hospital in Merion, Ill.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, and the Williamson County Medical Society. He was a member of the First Baptist Church of Herrin, the Masons, Elks, and Lions. Hunting, fishing, bird dogs, and horses were his greatest hobbies, and he was a member of the Bridle and Saddle Club.

Dr. Sirles died December 11, 1946, aged 34. He is survived by his mother, Mrs. Ethel Sirles, his wife, and three children, Judith Rae, Wayne Rendleman, and Janice Lu.

### CARL MILLER SNEED

Dr. Sneed, born 1877, was graduated from the University of Missouri School of Medicine, Columbia, in 1901, and practiced the speciality of diseases of the eye, ear, nose and throat in that city until the time of his death, November 6, 1946.

Dr. Sneed was a member of the American College of Surgeons, a Senior Fellow of the American Academy of Ophthalmology and Otolaryngology, and on the staff of the Boone County General Hospital. He served with the American Expeditionary Forces in France in World War I.

### THEODORE LASATER TERRY

Dr. Theodore L. Terry was born February 19, 1899, in Ennis, Texas. He received his preliminary schooling in Ennis and at Southern Methodist University. In 1922 he received his degree of Doctor of Medicine from the University of Texas and took postgraduate work in general pathology there. In 1925 he served as instructor in medicine and in 1926 as associate professor of pathology. In 1928 he was appointed assistant pathologist at the Massachusetts Eye and Ear Infirmary in Boston and became head of that department in 1932. He also acted in the capacity of ophthalmic surgeon at the Massachusetts Eye and Ear Infirmary. In 1940 he was appointed assistant professor of ophthalmology at Harvard and was acting head of the department during the war.

He served as an examiner for the American Board of Ophthalmology from 1942 to 1946; he was a trustee for the Association for Research in Ophthalmology; and was a former president of the New England Ophthalmological Society. He was also a member of the American Ophthalmological Society, the American Academy of Ophthalmology and Otolaryngology, the American Association for the Advancement of Science, the American Medical Association, and the American College of Surgeons. He belonged to Sigma Nu, Alpha Kappa Kappa, and the Harvard Club.

Aside from his professional pursuits, Dr. Terry was interested in current events, economics, photography, art, and astronomy. Dr. Terry died on September 28, 1946 and is survived by his wife, Helen.

### ANDREW JAY TIMBERMAN

Dr. Andrew Timberman, 82, retired oculist, surgeon, and Ohio State University professor, died at his home in Columbus, October 15, 1946. A native of Hamilton, Ohio, he was a graduate of Otterbein College in 1884 and received his B.A. degree there in 1903. He obtained his medical degree at Miami Medical College, Cincinnati, 1894. He studied at the University of Michigan, University of Halle (Germany), Vienna, and London. Later he made a special study of cataracts in India.

He began his practice in Columbus in 1896.

He was a member of the American Academy of Ophthalmology and Otolaryngology, American College of Surgeons, and several other medical associations and fraternities. At the time of his death he was a trustee at Otterbein College. Dr. Timberman was past president of the Columbus Academy of Medicine. He served on the faculties of Ohio Medical University, Starling Ohio Medical College, and Ohio State University College of Medicine. During World War I, he was oculist for the advisory board, Columbus district, U. S. Army. He was on the staffs of the Soldiers' and Sailors' Orphan Home in Xenia, the University, and St. Francis Hospital.

Dr. Timberman was interested in many civic affairs, a former member of the Columbus Board of Education, and an elder in the Broad Street Presbyterian Church. He is survived by his wife, Mrs. Leila S. Timberman, and five daughters.

### RICHARD JOSEPH TIVNEN

Born in Charleston, Illinois, Dr. Tivnen received his preliminary education at Notre Dame and Lee's Academy, taking his medical degree at Rush Medical College in Chicago in 1895. From 1901 to 1906, he did postgraduate work in Vienna, Paris, and London. He received honorary Doctor of Laws degrees from Loyola and Notre Dame universities.

For many years he was clinical profes-

sor of ophthalmology at the Loyola University School of Medicine. He was formerly an instructor in ophthalmology at Northwestern University Medical School. Acting as consulting oculist and aurist for the Illinois Central Railroad, he was also head of the eye, ear, nose, and throat departments of Mercy Hospital in Chicago, where he died August 27, 1946, aged 71. Other affiliations were with Provident and Cook County hospitals and the Illinois Charitable Eye and Ear Infirmary.

---

### GEORGE LORING TOBEY, JR.

Dr. G. L. Tobey was born June 3, 1881, in Lancaster, Massachusetts. He received his early education in the public schools of Clinton, Mass. and was graduated from Harvard Medical School in 1903. Postgraduate work in his chosen field of otology included a residency at Massachusetts Eye and Ear Infirmary and further study in Berlin. About 1905, he began his practice in Boston. Dr. Tobey's hospital and teaching appointments included Harvard Medical School, Massachusetts Eye and Ear Infirmary, and the Massachusetts General, Boston City, Brooks, New England Deaconess, New England Baptist, and Choate Memorial hospitals in Boston as well as the Union Hospital in Fall River. He served in the Medical Corps in World War I.

Dr. Tobey was a frequent contributor to the medical literature. A diplomate of the American Board of Otolaryngology, he served as president of the American Otological Society in 1933, and a Vice-president of the American Academy of Ophthalmology and Otolaryngology in 1926. He was also a member of the American Laryngological Society, the American Otological, Rhinological and Laryngological Society, the New England Otolaryngological Society, and the American College of Surgeons.

Dr. Tobey's death occurred May 20, 1947, in Brookline, Mass. His wife, Pauline F. Tobey, a daughter and two brothers survive him.

---

### ROBERT VON DER HEYDT

Dr. Von der Heydt was born in Wiesbaden, Germany, March 3, 1875. He came to this country in 1881, and was educated in Chicago public schools, taking his medical degree at the University of Illinois in 1903, after which he began his practice in Chicago. His postgraduate work was done at the Vogt Clinic in Zurich, Switzerland.

Dr. Von der Heydt taught at the Chicago College of Medicine and Surgery from 1909 to 1917, was associate professor at Rush Medical College and the University of Illinois. Since 1943, he was professor emeritus of ophthalmology at the University of Illinois. He served as ophthalmologist at the Illinois Charitable Eye and Ear Infirmary, Michael Reese Hospital, and Presbyterian Hospital in Chicago and the West Suburban Hospital in Oak Park, Illinois, where he died September 18, 1946, aged 71.

He was a Fellow of the American Academy of Ophthalmology and Otolaryngology and past president of the Chicago Ophthalmological Society.

Dr. Von der Heydt first introduced the slit lamp to the Western hemisphere. He was one of the foremost authorities on the subject in America and contributed many articles to the literature of slit lamp science. The first edition of Vogt's "Slit Lamp Atlas" was translated by him. He was an expert fundus photographer and was the first man to photograph the fundus in color. In conjunction with Dr. Harry Gradle he published an atlas of stereoscopic pictures of external eye diseases. Dr. Von der Heydt was one of the first men to work with contact lenses in America and was one of the most capable and best informed on contact lens fitting and indications. For many years he presented instruction courses at the American Academy of Ophthalmology and Otolaryngology.

---

### ARTHUR MORRIS ZINKHAN

Dr. A. M. Zinkhan was born in 1890. He was graduated from the Georgetown University School of Medicine, Washington, D. C. in 1912. He served as associate professor of otolaryngology at his alma mater and on the staff of the Episcopal Eye, Ear and Throat Hospital. Dr. Zinkhan's society affiliations included the American Academy of Ophthalmology and Otolaryngology, the American Laryngological, Rhinological and Otological Society, and the American College of Surgeons. He served in the Russian Army and later in the U. S. Army during World War I. Dr. Zinkhan died April 30, 1947.

# SCIENTIFIC EXHIBITS

James H. Maxwell, M.D., *Chairman*
Ann Arbor, Mich.

Kenneth L. Roper, M.D.
Chicago, Ill.

---

**Exhibitor:**

**K. W. ASCHER, M.D.**

BY INVITATION

College of Medicine
University of Cincinnati
Cincinnati, Ohio

**Aqueous Veins. The Biomicroscopically Visible Elimination of Intraocular Fluid**

The fact that aqueous humor can be seen biomicroscopically while streaming through the conjunctival and episcleral veins was detected in 1941. Since that time numerous investigators have confirmed this observation. The exhibit shows the anatomy, physiology, pathology, and pharmacology of the aqueous veins, their role in glaucoma and their responsiveness to surgery.

---

**Exhibitor:**

**J. E. ASH, Col., M.C., U.S.A.**

BY INVITATION

Army Institute of Pathology
Washington, D. C.

**Co-Exhibitor:**

**LAWRENCE P. AMBROGI**

BY INVITATION

**Temporal Bones of Aviators**

Prints and transparencies illustrating aerotitis media and the pathology of the eustachian tube.

---

**Exhibitor:**

**LAWRENCE R. BOIES, M.D.**

Secretary for Home Study Courses
American Academy of Ophthalmology and Otolaryngology

**Co-Exhibitors:**

**HENDRIE W. GRANT, M.D.**
**ROBERT E. PRIEST, M.D.**

**Program of the Home Study Courses**

The purpose of this exhibit will be to outline the history of the Home Study Course Program, to acquaint the Academy members with the Program of Instruction, the names of the Faculty, the quality of the work being done by the registrants, and the growth of the Courses.

---

**Exhibitor:**

**ELIZABETH E. CARR**

BY INVITATION

Library, Northwestern University Medical School
Chicago, Ill.

**Co-Exhibitor:**

**GEORGIA PRICE**

BY INVITATION

**History of the Literature of Ophthalmology and Otolaryngology**

The general plan is to divide the material in two groups; one dealing with ophthalmology, the other with otolaryngology. Each will include important and especially interesting books from the first published up to those of the early Twentieth century; some "classic descriptions"; the earliest volumes of the standard periodicals; and pictures of outstanding men. The section on ophthalmology will also include some early manuscripts.

---

**Exhibitor:**

**HOWARD A. CARTER, B.S.**

BY INVITATION

Secretary, Council on Physical Medicine
American Medical Association
Chicago, Ill.

**Co-Exhibitor:**

**F. T. JUNG, M.D.**

BY INVITATION

**Physics of an Audiometer and Hearing Aid**

This exhibit consists of demonstrational lectures, posters and published material for distribution. Demonstrational apparatus will be available to explain significant physical and acoustical principles which are utilized in the make-up and operation of an audiometer and hearing aid. Charts

will be on display showing the most recent trends in the amelioration of the hard of hearing. Demonstrational lectures will be given several times a day. Articles which have been published by the Council of Physical Medicine will be available for distribution.

---

**Exhibitor:**
**HAROLD F. FALLS, M.D.**
Department of Ophthalmology
University Hospital
Ann Arbor, Mich.

**Co-Exhibitor:**
**JANET McLAUGHLIN**
BY INVITATION

**Hereditary Ocular Anomalies**

The exhibit consists of charts, pedigrees, and transparencies which demonstrate the mode of inheritance of choroideremia, anterior ocular segmental anomalies, and atypical retinitis pigmentosa. The exhibit is arranged in a specially constructed viewing box.

---

**Exhibitor:**
**J. BROWN FARRIOR, M.D.**
Tulane Medical School and
Ochsner Clinic
New Orleans, La.

**Ear Surgery**

The exhibit consists of a series of anatomical dissections of the temporal bone with accompanying illustrations. These dissections and illustrations demonstrate the clinical and anatomical points of various ear operations, including the simple complete radical and modified radical mastoidectomy; surgery of the facial nerve; petrous apicectomies; the labyrinthectomy; the Day electrocoagulation of the labyrinth; and the fenestration operation.

---

**Exhibitor:**
**RAYMOND G. INGALLS, M.D.**
Institute of Ophthalmology
Presbyterian Hospital
New York, N. Y.

**Intraorbital Tumors**

Data gathered from an analysis of 217 tumors studied at the Institute of Ophthalmology, Presbyterian Hospital, will be presented.

The details of 21 different types of orbital tumors will be illustrated by photographs of patients, roentgenograms, excised tissue, and photomicrographs.

---

**Exhibitor:**
**JONAS FRIEDENWALD, M.D.**
Chairman, Committee on Standardization
of Tonometers
American Academy of Ophthalmology
and Otolaryngology

**Co-Exhibitors:**
**MEMBERS OF COMMITTEE ON STANDARDIZATION OF TONOMETERS**

**Standardization of Schiotz Tonometers**

As a result of inaccurate construction and general wear, most tonometers now in use differ in their critical physical properties from one another and from the standard instrument which Schiötz used in his calibration work. The exhibit shows the procedure of measuring the critical physical characteristics of the tonometer and of compensating for deviations from the standard tonometer by mathematical correction factors upon which a new conversion table is based that fits only the particular instrument.

---

**Exhibitor:**
**ARTHUR L. JUERS, M.D.**
Northwestern Medical School
Chicago, Ill.

**Co-Exhibitors:**
**C. F. WHITAKER, M.D.**
BY INVITATION
**GEORGE E. SHAMBAUGH, JR., M.D.**

**Fenestration Operation. An Improved Technic Based on Experimental Studies**

The exhibit consists chiefly of a series of wax models in color showing in detail the technic of the fenestration operation. Each model is 5 to 10 times actual size. Twelve transparencies show the clinical basis of deafness due to otosclerosis and the criteria for selection of cases for surgery. A tabulation of results will be included based on both gain in hearing and final level. (The exhibit should be of particular interest to the otolaryngologist who is not doing this type of surgery but needs orientation.)

**Exhibitor:**
**HERBERT M. KATZIN, M.D.**
Eye Bank for Sight Restoration, Inc.
New York, N. Y.

**Co-Exhibitors:**
**P. K. KUO, M.D.**
BY INVITATION
**R. T. PATON, M.D.**

**Corneal Transplantation. Histopathology of Corneal Grafts in Rabbits**

The exhibit consists of a series of black and white transparencies which are photomicrographs of rabbits' corneas which have been the site of corneal transplantation. They illustrate complications of the transplantation with fresh graft material as well as the results obtained in transplanting corneal grafts preserved in formaldehyde or frozen and dehydrated.

---

**Exhibitor:**
**WILLIS S. KNIGHTON, M.D.**
Chairman, Committee on Glaucoma
National Society for the
Prevention of Blindness
New York, N. Y.

**Co-Exhibitor:**
**VIRGINIA SMITH BOYCE**
BY INVITATION

**Preventing Blindness From Glaucoma**

Photographs, charts, and graphic material illustrating signs and symptoms of glaucoma recognizable during general physical check-up will be shown. Information on testing technics will be included.

---

**Exhibitor:**
**JULIUS LEMPERT, M.D.**
Lempert Institute of Otology
New York, N. Y.

**Modern Temporal Bone Surgery**

Anatomical display of step by step dissections describing the following surgical procedures developed and employed by the author:

1. Endaural removal of exostosis from external auditory canal.
2. Endaural correction of postauricular fistula.
3. Endaural complete mastoidectomy.
4. Endaural mastoidotympanectomy.
5. Endaural labyrinthotomy for drainage of suppurative labyrinthitis in sympathetic nonbacterial meningitis.
6. Endaural drainage of middle and posterior fossa abscess.
7. Endaural treatment of sinus thrombosis.
8. Endaural complete mastoido-tympano-apicectomy (exenteration of apex).
9. Endaural apicotomy (drainage of apex).
10. Endaural mastoido-atticotomy.
11. Fenestra Nov-Ovalis operation for clinical otosclerosis.
12. Decompression of labyrinth in Meniere's with preservation of hearing.
13. Decompression of labyrinth in Meniere's without preservation of hearing.
14. Tympanosympathectomy for tinnitus.

---

**Exhibitor:**
**FLORENCE NELSON**
BY INVITATION
Acting Director, Films and Exhibits
National Society for the Prevention
of Blindness, Inc.
New York, N. Y.

**Causes of Blindness**

Three large panels, illustrated, showing leading causes of blindness and indicating problems in research and education which are outstanding in prevention of blindness programs.

---

**Exhibitor:**
**JOHN F. PICK, M.D.**
BY INVITATION
Chicago, Ill.

**Ophthalmological Reconstructive Surgery**

Two hundred large Kodachrome slides on appropriate shadow boxes depicting the methods and results of ophthalmologic reconstructions. Exact moulage reproductions of extensive war injuries of the orbit and methods of repair. Charts illustrating adequate procedures.

**Exhibitor:**
**ADOLPH POSNER, M.D.**
New York, N. Y.

**Co-Exhibitor:**
**ABRAHAM SCHLOSSMAN, M.D.**
BY INVITATION

**Some Newer Aspects of the Glaucoma Problem**

1. The relationship between nerve fiber and circulatory visual field defects. This will be illustrated with a schematic drawing and selected field studies to show the evolution of the field defects.

2. The genetic approach to the study of glaucoma. The mechanism of heredity in glaucoma will be illustrated by charts and pedigrees.

3. Pupillography in glaucoma (by courtesy of Dr. Otto Lowenstein). This method of investigation sheds some light on the relation of the autonomic nervous system to glaucoma.

4. The syndrome of recurrent glaucomatocyclitic crises. This clinical entity is a type of glaucoma intermediate between primary and secondary. It is characterized by recurrent unilateral attacks of ocular hypertension with mild cyclitic signs.

---

**Exhibitor:**
**BENJAMIN RONES, M.D.**
Washington, D. C.

**Ophthalmology in Cartoon**

The exhibit consists of 120 cartoons dealing with various aspects of an ophthalmologist's life. They were collected over the past fifteen years from magazines and newspapers. Each picture is enlarged to an 8 by 10 inch size for easy visibility, and mounted 4 to a card. The humor level is high, and it is felt that the exhibit will afford a pleasant relief during these post-war years.

---

**Exhibitor:**
**MAURICE SNITMAN, M.D.**
University of Illinois
College of Medicine
Chicago, Ill.

**Carcinoma of the Larynx**

This exhibit will consist of a photographic presentation of serial sections of larynges with carcinoma involvement. This is a follow-up of the work which was started in 1941 and first presented in an exhibit before the Academy in 1944.

This additional presentation further emphasizes the necessity for a consideration of the point of origin and manner of extension of laryngeal carcinoma.

---

**Exhibitor:**
**HELENOR CAMPBELL WILDER**
BY INVITATION
Army Institute of Pathology
Washington, D. C.

**Co-Exhibitor:**
**LAWRENCE P. AMBROGI**
BY INVITATION

**Atomic Bomb Pathology of the Eye**

Prints and transparencies illustrating the pathology found in the eyes of atomic bomb casualties.

# TECHNICAL EXHIBITS

*In order to satisfy the growing demand for space in our technical exhibit, a limited number of booths in the Red Lacquer Room have been allotted to technical exhibitors. Booths 1-91, inclusive, are in the Exhibit Hall. Booths 101-110, inclusive, are in the Red Lacquer Room. The Academy is appreciative of the co-operation of all technical exhibitors.*

**Ahlstrom Surgical Co., Chicago, Ill.**
**Booth 106**

**Alkalol Co., Taunton, Mass.**
**Booth 53**

The Alkalol Company, Taunton, Mass. will exhibit ALKALOL, an alkaline, saline solution especially designed for the treatment of the mucous membranes of the eyes, nose, ears and throat. Its "pH" approximates the hydrogen ion concentration of the blood plasma and that of the lacrimal secretions. It is an effective mucus solvent. With no glycerine and only a trace of alcohol, it can be used freely without fear of irritation.

Alkalol was first introduced to the medical profession in 1896 and has won ever increasing favor. It is being shown at Booth 53 by E. W. LeClair and F. L. Cross.

**American Optical Co., Southbridge, Mass.**
**Booths 34 thru 40**

At the Meeting of the American Academy of Ophthalmology and Otolaryngology to be held at the Palmer House, Chicago, October 12-17, 1947, the American Optical Company will have a complete line of its ophthalmological instruments and equipment, as well as some of its scientific instruments, for exhibit and demonstration to ophthalmologists and members of the Academy.

An invitation is cordially extended to all those of the profession attending the meeting to visit the American Optical Company Booths Nos. 34 to 40, inclusive, where a staff of trained representatives from the Instrument Department of the American Optical Company will take pleasure in describing and demonstrating any instrument of interest to the physicians.

**Audio Development Co., Minneapolis, Minn.** **Booth 47**

The Audio Development Company has manufactured audiometers since 1937, maintaining a consistent leadership in research and development to provide a more convenient and dependable method for determining hearing acuity.

ADC Audiometers reveal speech hearing loss directly in decibels; are equipped with double headsets; have one direct reading decibel scale for bone and air conduction.

**Aurex Corp., Chicago, Ill.**
**Booth 48**

The Electro-Larynx, an instrument used for speaking purposes by those persons who have been deprived of their vocal mechanism either through injury or disease, will be on display. A rapidly increasing number of laryngologists are recommending this instrument for their postoperative patients.

In addition, we shall present our two new model wearable hearing aids, as well as our Group and Semiportable instruments.

Physicians who have not yet received a copy of the series of "A Visualization of the Functional Anatomy of the Ear" by W. J. S. Krieg, Ph.D. of Northwestern University Medical School, should ask for one at the Aurex booth.

**Barnett Laboratories, Chicago, Ill.**
**Booth 61**

Barnett Optical Laboratories will present the new electronic arc lamp development which can be used to replace the carbon arcs now in use in fundus cameras.

Also on exhibit will be the new anterior segment eye camera using flash bulbs and bantam size film.

A full line of slide projectors, photoflood lighting systems for anterior segment eye photography, slide libraries of eye diseases and injuries and office examination lamps will be displayed.

**Bausch & Lomb Optical Co., Rochester, N. Y.** **Booth 32**

Of especial interest to ophthalmologists who visit the Bausch & Lomb exhibit at

the annual convention of the American Academy of Ophthalmology and Otolaryngology will be the Allen Gonioscope. The Allen contact prism in this instrument makes practical for the first time routine gonioscopy with the regulation slit lamp while the patient is seated for customary slit lamp examination. While the Allen Gonioscope is extremely valuable in the consideration of glaucomatous involvement, the status of other clinical entities, such as trauma, inflammatory processes, pigmentary anomalies, and the like, may be ascertained.

On display will be Bausch & Lomb ophthalmic instruments for diagnosis and refraction, including the DeLuxe Refracting Unit, Green's Refractor, Poser Slit Lamp, Binocular Ophthalmoscope, and others.

Information regarding developments in industrial vision, based upon new research activities by studies in the plants of subscribers to the Bausch & Lomb Industrial Vision Service and Purdue University, will also be available at the B & L exhibit.

---

**Bausch and Lomb Affiliated Companies**
**Colonial Optical Co., New York, N. Y.**
**McIntire, Magee & Brown Optical Co., Philadelphia, Pa.**
**Riggs Optical Co., San Francisco, Calif., and Chicago, Ill.**
**Southeastern Optical Co., Richmond, Va.**
**Booths 67 thru 69**

The Allen Gonioscopic Prism used in conjunction with the Poser Slit Lamp will be demonstrated daily. The Bausch and Lomb Binocular Ophthalmoscope employing the Hildreth Mercury Lamp will also be set up for practical demonstrations in red-free ophthalmoscopy. A complete Bausch and Lomb Refracting Unit to which will be attached the Greens' Refractor and Cilometer will be available for demonstration. Other instruments on display will include the Wottring Troposcope, Rotoscope, Bausch and Lomb Ferree-Rand Perimeter, Universal Slit Lamp, Vertometer and Keratometer.

---

**Belgard, Inc., Chicago, Ill.**
**Booths 44 and 45½**

Austin B. Belgard and J. Ralph Lowrey, representing the firm of Belgard, Inc., will exhibit the newest refracting room aids for the ophthalmologist. To mention just one of the outstanding items—the new "pocket-size" plastic prism bar. Among some of the other familiar Belgard products on exhibit will be the Lenscorometer, the illuminated Portable Visual Acuity Chart, and the Visual Acuity chart with illiterate "E" symbol for quantitative measurement of vision at the near point.

Of especial interest will be the long awaited ConAcor lens—regarded as an outstanding development in the field of optics. Messrs. Belgard and Lowrey will gladly explain the use for ConAcor.

The firm of Belgard, Inc. extends a cordial invitation to the members of the Academy and their guests to visit their complete display of ophthalmic equipment.

---

**Belgard-Spero, Inc. (The House of Vision), Chicago, Ill. Booth 15**

Our display will show continuous demonstrations of the Selinger Iontophoresis Apparatus, and also the premier showing of a multifocal head-mirror. In addition, we will show a new condensed contact lens fitting case, the House of Vision Distometer (for measuring vertex distances between the refracting lens and cornea), and also examples of unusual lens grinding in difficult prescriptions.

---

**Bell & Howell Co., Chicago, Ill.**
**Booth 62**

The Bell & Howell Company will exhibit motion picture cameras, projectors and accessories, and also 2 x 2 slide projectors and sound motion picture projectors.

---

**Camel Cigarettes, New York, N. Y.**
**Booths 51 and 52**

CAMEL Cigarettes will present a dramatic full color review of their recent medical research on smoking, as well as the details of the nationwide survey showing that "More Doctors Smoke Camels Than Any Other Cigarette." Another panel will illustrate the absorption of nicotine in the respiratory tract. Representatives will be present.

---

**Cameron Surgical Specialty Co., Chicago, Ill. Booth 56**

This year the new Lempert headlite with the large mirror will be on display for the first time in five years.

The new completely boilable, all stain-

less steel bronchoscopic outfit and the DeLuxe Omniangle gastroscope will be shown.

See the Telescopic Otoscope, the new "Day-Lite" ophthalmoscope and ask for a demonstration of the Electro-Surgical (Radio Frequency) machines which come in two sizes for eye, ear, nose and throat surgery.

**Clay-Adams Co., Inc., New York, N. Y. Booth 31**

Clay-Adams exhibit will feature visual aids in the field of ophthalmology and otolaryngology. A new series of three-dimensional Kodachromes on ophthalmology will be demonstrated. The exhibit will also include the certified Schiötz Tonometers.

**Contact Lens Service, New York, N. Y. Booth 89**

The exhibit by Contact Lens Service will demonstrate by means of plastic models the special uses of contact lenses in postoperative problem cases and in the cosmetic rehabilitation of cases of corneal blemishes. Contact Lens Service will also demonstrate the value of contact lenses in cases of keratoconus, aphakia and aniseikonia, and will exhibit original eye molds of some of the most interesting cases of the many thousands fitted by Contact Lens Service during the past six years. These molds will show the need for the many different types of lenses and methods of fitting used by Contact Lens Service, and will also demonstrate the technic of fitting the Naturform custom-made plastic eyes.

**DeVilbiss Co., Toledo, Ohio Booth 42**

The continuance of encouraging reports relative to spraying or the inhalation of penicillin, streptomycin and other substances, make the DeVilbiss Exhibit at Booth No. 42 of pertinent interest. Atomizers, nebulizers, vaporizers and powder blowers for home use as well as office treatment, will be displayed. There are types for any treatment where spray application or inhalation is to be employed.

**Doho Chemical Corp., New York, N. Y. Booth 57**

The Doho Chemical Corporation, makers of AURALGAN—O-TOS-MO-SAN, ethical ear medications, are displaying their exhibit depicting the many pathologic conditions of the ear drum, made in rubber moulage. Also, they are distributing copies of the AURAL ATLAS which are now available to all ear, nose and throat specialists, gratis.

**Edwards Optical Co., Chicago, Ill. Booth 11**

At the Edwards Optical Company exhibit in Booth No. 11 slides on peripheral astigmatism and bifocal lenses will be shown.

**Feinbloom Contact Lenses, Inc., New York, N. Y. Booth 103**

Members of our technical staff will be available at our booth for discussion of any problems in the fitting of contact lenses. The fitting characteristics of the FEINCONE contact lens (trial case method) will be demonstrated by actual fitting to those who wish it. Local adjustment with heated FEINCONE Forceps will also be shown.

**General Electric X-ray Corp., Chicago, Ill. Booth 86**

The Berman Metal Locator is a simple and effective device for detecting and accurately locating metallic foreign bodies embedded in tissue. To the ophthalmologist, the Locator offers a means of determining whether the foreign body is intraocular or extraocular; also whether it is magnetic, thus guiding him in the use of the magnetic extractor.

**Grune & Stratton, Inc., New York, N. Y. Booth 59**

Grune & Stratton will exhibit such well known standard works as Spiegel and Sommer: NEUROLOGY OF THE EYE, EAR, NOSE AND THROAT; and Kestenbaum: CLINICAL METHODS OF NEURO-OPHTHALMOLOGY. New surgical books of interest include Brown: MODERN PLASTIC SURGICAL PROSTHETICS; and Berson: ATLAS OF PLASTIC SURGERY. A number of new French and German language books will also be exhibited for the first time.

**Guild of Prescription Opticians of America, Inc., Philadelphia, Pa. Booths 109 and 110**

In addition to a collection of old spectacles, and a map showing the locations

of the various Guild members throughout the United States and Canada, the exhibit will illustrate the essential services offered to the patient of the ophthalmologist by the Guild Optician. They include the following:

(1) Accurate interpretation of your prescription; measurement of pupillary distances, etc.
(2) Expert skill in compounding your prescription.
(3) Painstaking care in fitting glasses to your patient.
(4) Periodic service in keeping the glasses properly fitted.

---

**Hollister-Stier Laboratories, Wilkinsburg, Pa. Booth 83**

In the opinion of many leading otolaryngologists, it is difficult to practice in this field without facilities to handle the allergic patient. Hollister-Stier Laboratories, specializing exclusively in allergy, are well equipped to assist you with allergic problems. Well-informed representatives will be in attendance at the annual meeting to discuss your problems and supply information concerning products. You are cordially invited to visit the booth.

---

**Invisible Lens Service, Buffalo and Syracuse, N. Y.; Stroudsburg and Scranton, Pa.; Cleveland, Ohio; Indianapolis, Ind. Booth 65**

Our services on the molding, fitting, and technical work on Invisible Lenses gives the ophthalmologist a complete service for his patients.

We will display complete equipment, and our technician in charge will explain in detail our services, or will call at your office should you be located in Ohio, New York, Pennsylvania or Indiana.

Vision has been restored on conical corneas, corneal scars, high myopes, and cataract cases. Consult us on your difficult refraction cases.

Our services are recommended by all the leading ophthalmologists.

---

**Kelley-Koett Manufacturing Co., Covington, Ky. Booths 9 and 10**

The American Electric Co., a division of The Kelley-Koett Mfg. Co., is exhibiting an X-Ray Head Unit at Booths 9 and 10. The American Electric Head Unit is especially designed for ENT work, and affords radiography in every position for sinus and skull work. It is quickly and accurately positioned for every technic, and affords the maximum of convenience for both the doctor and the patient.

---

**Keystone View Co., Meadville, Pa. Booths 49 and 50**

The Keystone BSM Stereo-Motivator is Keystone View Company's latest development for squint training, developed on the principle that strabismics reject macular fusion. This new technic begins fusion training in the peripheral area and then progresses toward the macula. See this new technic demonstrated at Booths 49 and 50.

---

**Lea & Febiger, Philadelphia, Pa. Booth 81**

At Booth 81 Lea and Febiger will exhibit among their new works and new editions Gifford's "Ocular Therapeutics," Spaeth's "Principles and Practice of Ophthalmic Surgery," Ballenger on the "Ear, Nose and Throat," Ballenger's "Manual of Otology, Rhinology and Laryngology," Cowan's "Refraction of the Eye," Stimson's "Common Contagious Diseases," Bell's "Text-book of Pathology," Cushny's "Pharmacology and Therapeutics," Joslin's "Treatment of Diabetes Mellitus," Wintrobe's "Hematology," Katz's "Electrocardiography," Levinson and MacFate's "Clinical Laboratory Diagnosis" and other standard works.

---

**Mager & Gougelman, Inc., Chicago, Ill. Paul Gougelman Co., Chicago, Ill. Booths 45½ and 46**

The exhibit will consist of a display of reform and shell artificial eyes in both glass and plastic, a complete line of conformers, drains, shields and the latest type of implants. A demonstration will be given showing how glass and plastic artificial eyes are made. In addition, a clinical movie will be shown concerning the Cutler Ball and Ring Implant.

---

**Maico Co. Inc., Minneapolis, Minn. Booths 74 and 75**

The Maico exhibit will contain the Maico D9 Audiometer, the "Atomeer" hearing aid, and will feature the sensational new Audiometer, the Maico El. The El contains all the features of the

D9, plus many new developments made possible by war research in the field of electronics and hearing and will open many new possibilities in hearing testing. Every otologist will profit by seeing this fine new precision instrument.

**Medical Bureau, Chicago, Ill.**
**Booth 104**

**Medical Case History Bureau, New York, N. Y.** **Booth 63**

Simplifying the doctor's history record and bookkeeping system with the Info-Dex Record Control system. Maintenance of accurate, informative data on both history and financial records is essential in the modern doctor's practice. The Info-Dex Record Control System helps to keep a constant finger on the physical and financial pulse of the patient. This system correlates information almost automatically for instant reference and research work. Its method of cross-indexing interesting cases according to the disease is unique and exclusive.

**Medical Film Guild, New York, N. Y.**
**Booth 33**

Medical Film Guild, through their "Medical Films That Teach," presents a refresher course in fundamental medical problems. They review such subjects as Parkinson's disease, major neuralgias, cervicitis, otolaryngological diseases, contagious diseases, arterial blood pressure, hypothyroidism, industrial medicine. These are available to medical institutions including projection service at no charge, through grants for postgraduate instruction.

**E. B. Meyrowitz Surgical Instruments Co., Inc., New York, N. Y.**
**Booth 108**

**C. V. Mosby Co., St. Louis, Mo.**
**Booth 58**

A cordial invitation is extended to those attending the American Academy of Ophthalmology and Otolaryngology convention to visit Booth No. 58 where the C. V. Mosby Company will display its complete line of medical publications. In addition to such well-known texts as Duke-Elder's "Textbook of Ophthalmology," Tassman's "Eye Manifestations of Internal Diseases," and Traquair's "Introduction to Clinical Perimetry," two new books in the eye field will be shown, namely, Scobee's "The Oculorotary Muscles" and Wright's "Researches on Normal and Defective Colour Vision." Whatever your book requirements, our representatives will be glad to discuss them with you.

**V. Mueller & Co., Chicago, Ill.**
**Booths 1 thru 6**

You are invited to inspect the new Mueller Electronic Tonometer, which will be demonstrated in the V. Mueller & Co. exhibit. This remarkable adaptation of the principles of electronics brings the ophthalmologist, at last, an instrument of precise accuracy, free from the disadvantages and shortcomings of earlier mechanical tonometers. The display will also include selected special and standard instruments for surgery of the eye, ear, nose and throat.

**National Contact Lens Corp., New York, N. Y.** **Booth 82**

**New Jersey Optical Co., Irvington, N. J.**
**Booth 76**

The New Jersey Optical Company will present their complete line of Quality Frames and Mountings—all guaranteed invariably, 50/1000 Fine Gold.

Wils-edge, which is New Jersey Optical's unique mounting—suspending lenses from above without holes or screws—will be featured, with especial emphasis on its easy adaptability to individual demands and true custom-fitting.

**Novocol Chemical Mfg. Co., Inc., Brooklyn, N. Y.** **Booth 12**

NOVOCOL CHEM. MFG. CO., INC., Booth 12, extends a cordial invitation to the attending physicians to visit with us. There will be on display—three MONOCAINE HCl solutions—the safe, more potent local anesthetic agent—designed for use in Ophthalmology, Otorhinolaryngology and for Plastic Surgery. The popular Anestube and Novampul (Cartridge systems) will be demonstrated also. Make BOOTH 12 a "MUST SEE" in your convention memo book.

**Obrig Laboratories, Inc., New York, N. Y.** **Booth 64**

The ever increasing popularity of contact lenses makes it imperative that every ophthalmologist be conversant with

the facts regarding this modern method of visual correction. A feature of the Obrig Laboratories exhibit, pioneers in the manufacture of plastic contact lenses, is an automatic slide film showing the use of the contact lenses in various eye conditions.

**Otarion Hearing Aids, Chicago, Ill.**
**Booth 107**

**Philip Morris & Co. Ltd., Inc., New York, N. Y.** **Booth 16**

Philip Morris & Company will demonstrate the method by which it was found that Philip Morris Cigarettes, in which diethylene glycol is used as the hygroscopic agent, are less irritating than other cigarettes. Their representative will be happy to discuss researches on this subject, and problems on the physiological effects of smoking.

**George P. Pilling & Son Co., Philadelphia, Pa.** **Booths 77 thru 79**

At the Academy Meeting in October for the first time in some years, we expect to show some ear, nose and tonsil punches including those developed by Dr. J. M. Robison of Houston, Texas, on which we are planning to make reasonably prompt deliveries; also some interesting developments in items made of plastic material of particular interest to the eye men. There will be a substantial addition to our showing of nasal plastic instruments.

Other items are Beck's turbinotome, asbestos packed ear syringe, the new Daniel's tonsillectome, and the popular folding laryngoscopes.

**Precision Contacts, Minneapolis, Minn.**
**Booth 29**

Newer developments in contact lenses and technics of fitting these visual aids will be shown. The 100 per cent acrylic Naturform plastic eye prosthesis will be exhibited.

**Premier Optical Products, Inc., New York, N. Y.** **Booth 41**

Premier Optical Products will have on display their visual training instruments, the Vectometer and Vectoluminator. They will also display Lenscoat, a new multifocus head mirror and other optical specialties which have already received wide acceptance in the optical professions.

**Professional Press, Chicago, Ill.**
**Booth 80**

Current and previous issues of the "Eye, Ear, Nose and Throat Monthly" will be shown. The "Monthly" is intended as a **practical** journal for the eye, ear, nose and throat specialist. The "Monthly" is now in its 26th year of publication.

**Radium Chemical Co. Inc., New York, N. Y.** **Booths 54 and 55**

Radium Chemical Company, Inc., will display the monel metal radium applicator for the treatment of lymphoid tissue in the nasopharynx. This is the original applicator designed by Radium Chemical Company according to specifications of pioneer otologists in this field.

Radon equipment will also be displayed.

**Ritter Co., Inc., Rochester, N. Y.**
**Booths 17 thru 21**

Ritter Company, Incorporated, will have on display in Booths No. 17, 18, 19, 20 and 21, the Ritter Ear, Nose and Throat Unit, Ritter Ear, Nose and Throat Motor Chair, Ritter Ear, Nose and Throat Shockproof X-Ray, the new Ritter Deluxe Hydromatic Sterilizer with Synchronous Timer, Ritter Motor Driven Multi-Purpose Table, Ritter Compressor and Ritter Rest and Relief Stool.

**Smith, Kline & French Laboratories, Philadelphia, Pa.** **Booth 72**

Eskay's Oralator is featured at this exhibit. Eskay's Oralator provides a revolutionary method of cough control. Inhaled by MOUTH, the Oralator's anesthetic-analgesic vapor (2-amino-6-methylheptane) is delivered directly to the nerve endings in the trachea and larynx, where it controls cough within a matter of seconds. Safe and effective, the Oralator is indicated in those types of coughs for which codeine would ordinarily be prescribed. Unlike sedatives and narcotics, however, the Oralator produces no appreciable systemic effects.

Our specially trained professional representatives will be glad to answer questions concerning the possible uses of our products in your practice.

**Soft-Lite Lens Co., Inc., New York, N. Y.**
**Booths 70 and 71**

Weary delegates at the annual convention of the American Academy of Oph-

thalmology and Otolaryngology, October 12th—17th, will find a pleasant spot to relax in the Soft-Lite Lens Company exhibit. Designed by one of America's foremost designers, the exhibit presents a pleasant Swedish Modern aspect that invites relaxation and comfort. On hand to discuss any problems concerning Soft-Lite will be Nathaniel Singer, President; Raymond G. Landis, Vice President and General Manager; Charles Ladew and George Pearce.

**Sonotone Corp., New York, N. Y.**
**Booth 30**

Sonotone Doctor's Audiometer—a new model, incorporating many new features such as masking for both bone and air conduction measurements, and improved switching arrangements, including the tone interrupter switch.

Sonotone Hearing Aids and accessories.

"The Human Ear in Anatomical Transparencies."

**C. M. Sorensen Co., Inc., New York, N. Y.**
**Booth 66**

Sorensen Company will have on display latest models of ear, nose and throat equipment (treatment as well as surgical units). The DeLuxe will be furnished with coagulator and cutting current. Operating room equipment has explosion-proof features.

**Frederick Stearns & Co., Detroit, Mich.**
**Booths 27 and 28**

Agents whose efficiency have been time proven in the practice of otolaryngology and ophthalmology will be on exhibit. Neo-Synephrine Hydrochloride and Sulfathiazolate Solutions, and Neo-Synephrine Penicillin combination for topical use in the nose offer distinct advantages by being effective on repeated dosage and by being virtually free of side reactions. Neo-Synephrine Hydrochloride preparations for ophthalmic use are offered in different percentage strength with effects varying from mild decongestive action to mydriatic potency of sufficient two-fold action to break up the posterior synechiae of secondary glaucoma.

**Storz Instrument Co., St. Louis, Mo.**
**Booths 90 and 91**

Storz Surgical Instruments feature stainless materials, fine workmanship, correct design and balanced smooth operation.

Inspect our uniquely designed eye scissors, delicate forceps, excellent eye knives and accessory eye instruments and equipment.

For ear, nose and throat the Storz La Force adenotome, Sage tonsil snare, a new model entirely rustless Beck type snare and nasoplastic instruments. Complete assortment of instruments, diagnostic and supply items.

Several new items not shown previously.

**Uhlemann Optical Co., Chicago, Ill.**
**Booths 7 and 8**

Exhibit — PHYSICIAN'S QUALITY GLASSES AND EQUIPMENT. First to serve the ophthalmologist exclusively. Let us tell you of our service.

**Univis Lens Co., Dayton, Ohio**
**Booths 13 and 14**

**Washington Institute of Medicine, Washington, D. C. Booth 101**

In Booth 101 the world-famous **Quarterly Reviews** of the WASHINGTON INSTITUTE OF MEDICINE will be exhibited. Of especial interest is the **Quarterly Review of Otorhinolaryngology and Broncho-Esophagology** edited by Dr. Chevalier L. Jackson and other distinguished teachers and clinicians. The **Quarterly Review of Ophthalmology**, edited by Dr. Conrad Berens and an Editorial Board composed of internationally recognized authorities, is the companion publication, which upholds the tradition and high standards of all the **Quarterly Reviews.** At this time will also be exhibited the first volumes of an outstanding accomplishment in medical bibliographic work, the first set of "Abstracts on Military and Aviation Ophthalmology and Visual Sciences through 1940". Under the direction of Dr. Conrad Berens, this great work, embracing more than 4,000 annotated references, was made possible through the co-operation of institutions and individuals, so that all interested in ophthalmologic progress may have ready access to an authoritative chronological record.

**Western Electric Co., New York, N. Y.**
**Booths 84 and 85**

Featured at the Western Electric booth will be a display showing Bell Telephone Laboratory designed Western Electric

Hearing Aids, Group Hearing Aids, and Diagnostic and School Type Audiometers.

Included in the display will be Western Electric's new "All-In-One" Hearing Aids and the new 4CA Group Audiometer.

Representatives of the Western Electric Company of 120 Broadway, New York, N. Y., will be in attendance at the booth to answer your questions concerning the products displayed.

**White-Haines Optical Co., Columbus, Ohio Booth 43**

The White-Haines Optical Company will display a number of ophthalmic spectacles incorporating unusual and difficult prescriptions interpreted into these ophthalmic spectacles.

**White Laboratories, Inc., Newark, N. J. Booth No. 105**

**Williams & Wilkins Co., Baltimore, Md. Booth 60**

The Williams & Wilkins Company will have copies of "Histopathology of the Ear, Nose, and Throat" by Eggston and Wolff; "Neuro-Ophthalmology" by Frank B. Walsh; a new edition of Grant's "Atlas of Anatomy" with more than 200 additional illustrations. The new world-wide series of 15 abstract journals, published under the general title, "Excerpta Medica," will be available for inspection and subscription.

**Max Wocher & Son Co., Cincinnati, Ohio Booths 22 thru 26**

An unusually complete line of instruments will be the feature of the exhibit of The Max Wocher & Son Company. The Wocher firm is not only a dealer in surgical instruments but manufactures many of its own models as well. It operates its own large factory for the production of hospital and physicians' metal furniture and is well known as the originator of many of the most popular models of ear, eye, nose and throat operating chairs and tables.

**Wottring Instrument Co., Columbus, Ohio Booth 73**

Here you will see demonstrated the 45B Troposcope, basically a Major Amblyoscope but with many modern improvements. Approved by the American Orthoptic Council, the 45B Troposcope provides hairline precision measurement of squint which is vital to successful operation. It is also excellent for general orthoptic training, both pre- and postoperative, with a unique method of visual stimulus without rocking the entire eyepiece assembly, thus preserving original settings. The Royal "50" Rotoscope is also well known in the orthoptic field, being used successfully for versatile training when glasses alone do not give complete comfort. Both the Troposcope and Rotoscope are manufactured here in America for The Wottring Instrument Company, being widely used in ophthalmologists' offices, hospitals, clinics and orthoptic schools.

**Year Book Publishers, Inc., Chicago, Ill. Booth 102**

**Zenith Radio Corp., Chicago, Ill. Booths 87 and 88**

The feature of the exhibit of Zenith Radio Corporation is the new Zenith "75" Hearing Aid. This is an entirely new type of compact instrument. With it the battery and amplifier may be worn in one case, or, for convenience, as two separate units. Outstanding performance is made possible by new acoustical, electrical and mechanical developments that have been incorporated in the instrument.

Another feature of the display will be the new Zenith radios and radio-phonographs featuring the Cobra Tone Arm.

## PAST OFFICERS OF THE ACADEMY

| Date | President | First Vice-President | Treasurer | Secretary | Meeting Place |
|---|---|---|---|---|---|
| **1896**— | Adolph Alt, M.D.<br>St. Louis, Mo. | W. C. Pipino, M.D.<br>Des Moines, Iowa | W. L. Dayton, M.D.<br>Lincoln, Nebr. | Hal Foster, M.D.<br>Kansas City, Mo. | Kansas City |
| **1897**— | Adolph Alt, M.D.<br>St. Louis, Mo. | W. C. Pipino, M.D.<br>Des Moines, Iowa | W. L. Dayton, M.D.<br>Lincoln, Nebr. | Hal Foster, M.D.<br>Kansas City, Mo. | St. Louis |
| **1898**— | B. E. Fryer, M.D.<br>Kansas City, Mo. | J. E. Colburn, M.D.<br>Chicago, Ill. | W. L. Dayton, M.D.<br>Lincoln, Nebr. | Hal Foster, M.D.<br>Kansas City, Mo. | Chicago |
| **1899**— | J. E. Colburn, M.D.<br>Chicago, Ill. | W. Scheppegrell, M.D.<br>St. Louis, Mo. | W. L. Dayton, M.D.<br>Lincoln, Nebr. | Thomas A. Woodruff, M.D.<br>Chicago, Ill. | New Orleans |
| **1900**— | W. Scheppegrell, M.D.<br>St. Louis, Mo. | M. A. Goldstein, M.D.<br>St. Louis, Mo. | W. L. Dayton, M.D.<br>Lincoln, Nebr. | Fayette C. Ewing, M.D.<br>St. Louis, Mo. | St. Louis |
| **1901**— | M. A. Goldstein, M.D.<br>St. Louis, Mo. | H. W. Wurdemann, M.D.<br>Milwaukee, Wis. | W. L. Dayton, M.D.<br>Lincoln, Nebr. | W. L. Ballenger, M.D.<br>Chicago, Ill. | Cincinnati |
| **1902**— | C. R. Holmes, M.D.<br>Cincinnati, Ohio | W. L. Dayton, M.D.<br>Lincoln, Nebr. | Otto Stein, M.D.<br>Chicago, Ill. | W. L. Ballenger, M.D.<br>Chicago, Ill. | Chicago |
| **1903**— | W. L. Ballenger, M.D.<br>Chicago, Ill. | C. J. Stillson, M.D.<br>Indianapolis, Ind. | Otto Stein, M.D.<br>Chicago, Ill. | Derrick Vail, M.D.<br>Cincinnati, Ohio | Indianapolis |
| **1904**— | Edward Jackson, M.D.<br>Denver, Colo. | Dudley Reynolds, M.D.<br>Louisville, Ky. | Otto Stein, M.D.<br>Chicago, Ill. | Derrick Vail, M.D.<br>Cincinnati, Ohio | Denver |
| **1905**— | Hanau Loeb, M.D.<br>St. Louis, Mo. | Derrick Vail, M.D.<br>Cincinnati, Ohio | Otto Stein, M.D.<br>Chicago, Ill. | George F. Suker, M.D.<br>Chicago, Ill. | Buffalo |
| **1906**— | Casey Wood, M.D.<br>Chicago, Ill. | J. A. Stucky, M.D.<br>Lexington, Ky. | Otto Stein, M.D.<br>Chicago, Ill. | George F. Suker, M.D.<br>Chicago, Ill. | St. Clair |

## PAST OFFICERS OF THE ACADEMY

| Date | President | First Vice-President | Treasurer | Secretary | Meeting Place |
|---|---|---|---|---|---|
| **1907**— | J. A. Stucky, M.D. Lexington, Ky. | R. A. Reeve, M.D. Toronto, Canada | Otto Stein, M.D. Chicago, Ill. | George F. Suker, M.D. Chicago, Ill. | Louisville |
| **1908**— | Derrick Vail, M.D. Cincinnati, Ohio | Joseph Beck, M.D. Chicago, Ill. | Otto Stein, M.D. Chicago, Ill. | George F. Suker, M.D. Chicago, Ill. | Cleveland |
| **1909**— | Otto Stein, M.D. Chicago, Ill. | Percy Fridenberg, M.D. New York, N.Y. | S. H. Large, M.D. Cleveland, Ohio | George F. Suker, M.D. Chicago, Ill. | New York |
| **1910**— | Wendell Reber, M.D. Philadelphia, Pa. | L. B. Lockhard, M.D. Denver, Colo. | S. H. Large, M.D. Cleveland, Ohio | George F. Suker, M.D. Chicago, Ill. | Cincinnati |
| **1911**— | J. J. Kyle, M.D. Indianapolis, Ind. | F. Parke Lewis, M.D. Buffalo, N. Y. | S. H. Large, M.D. Cleveland, Ohio | George F. Suker, M.D. Chicago, Ill. | Indianapolis |
| **1912**— | George F. Suker, M.D. Chicago, Ill. | J. W. Murphy, M.D. Cincinnati, Ohio | S. H. Large, M.D. Cleveland, Ohio | L. M. Francis, M.D. Buffalo, N. Y. | Niagara Falls, Ont. |
| **1913**— | J. W. Murphy, M.D. Cincinnati, Ohio | J. O. McReynolds, M.D. Dallas, Texas | S. H. Large, M.D. Cleveland, Ohio | L. M. Francis, M.D. Buffalo, N. Y. | Chattanooga |
| **1914**— | J. M. Ray, M.D. Louisville, Ky. | J. M. Ingersoll, M.D. Cleveland, Ohio | S. H. Large, M.D. Cleveland, Ohio | L. M. Francis, M.D. Buffalo, N. Y. | Boston |
| **1915**— | Joseph Beck, M.D. Chicago, Ill. | Robert S. Lamb, M.D. Washington, D.C. | S. H. Large, M.D. Cleveland, Ohio | L. M. Francis, M.D. Buffalo, N. Y. | Chicago |
| **1916**— | J. E. Brown, M.D. Columbus, Ohio | T. E. Carmody, M.D. Denver, Colo. | S. H. Large, M.D. Cleveland, Ohio | L. M. Francis, M.D. Buffalo, N. Y. | Memphis |
| **1917**— | W. L. Dayton, M.D. Lincoln, Nebr. | Edward Stieren, M.D. Pittsburgh, Pa. | S. H. Large, M.D. Cleveland, Ohio | L. M. Francis, M.D. Buffalo, N. Y. | Pittsburgh |
| **1918**— | Allen Greenwood, M.D. Boston, Mass. | Virginius Dabney, M.D. Washington, D. C. | S. H. Large, M.D. Cleveland, Ohio | L. M. Francis, M.D. Buffalo, N. Y. | Denver |

# PAST OFFICERS OF THE ACADEMY

| Date | President | First Vice-President | Treasurer | Secretary | Meeting Place |
|---|---|---|---|---|---|
| 1919 | J. M. Ingersoll, M.D. Cleveland, Ohio | Melville M. Black, M.D. Denver, Colo. | S. H. Large, M.D. Cleveland, Ohio | Luther C. Peter, M.D. Philadelphia, Pa. | Cleveland |
| 1920 | L. M. Francis, M.D. Buffalo, N. Y. | Hal Foster, M.D. Kansas City, Mo. | S. H. Large, M.D. Cleveland, Ohio | Luther C. Peter, M.D. Philadelphia, Pa. | Kansas City |
| 1921 | Emil Mayer, M.D. New York, N. Y. | John R. Newcomb, M.D. Indianapolis, Ind. | S. H. Large, M.D. Cleveland, Ohio | Luther C. Peter, M.D. Philadelphia, Pa. | Philadelphia |
| 1922 | Walter R. Parker, M.D. Detroit, Mich. | Ross H. Skillern, M.D. Philadelphia, Pa. | S. H. Large, M.D. Cleveland, Ohio | Luther C. Peter, M.D. Philadelphia, Pa. | Minneapolis and St. Paul |
| 1923 | T. E. Carmody, M.D. Denver, Colo. | Hunter H. McGuire, M.D. Winchester, Va. | S. H. Large, M.D. Cleveland, Ohio | Luther C. Peter, M.D. Philadelphia, Pa. | Washington, D. C. |
| 1924 | W. B. Lancaster, M.D. Boston, Mass. | William P. Wherry, M.D. Omaha, Nebr. | S. H. Large, M.D. Cleveland, Ohio | Luther C. Peter, M.D. Philadelphia, Pa. | Montreal |
| 1925 | Horace Newhart, M.D. Minneapolis, Minn. | John M. Wheeler, M.D. New York, N. Y. | S. H. Large, M.D. Cleveland, Ohio | Luther C. Peter, M.D. Philadelphia, Pa. | Chicago |
| 1926 | E. C. Ellett, M.D. Memphis, Tenn. | Austin A. Hayden, M.D. Chicago, Ill. | S. H. Large, M.D. Cleveland, Ohio | Luther C. Peter, M.D. Philadelphia, Pa. | Colorado Springs |
| 1927 | Ross Hall Skillern, M.D. Philadelphia, Pa. | H. W. Woodruff, M.D. Joliet, Ill. | S. H. Large, M.D. Cleveland, Ohio | William P. Wherry, M.D. Omaha, Nebr. | Detroit |
| 1928 | Luther C. Peter, M.D. Philadelphia, Pa. | Frank L. Dennis, M.D. Colorado Springs, Colo. | S. H. Large, M.D. Cleveland, Ohio | William P. Wherry, M.D. Omaha, Nebr. | St. Louis |
| 1929 | Harris P. Mosher, M.D. Boston, Mass. | Frank E. Burch, M.D. St. Paul, Minn. | S. H. Large, M.D. Cleveland, Ohio | William P. Wherry, M.D. Omaha, Nebr. | Atlantic City |
| 1930 | William H. Wilder, M.D. Chicago, Ill. | Frank R. Spencer, M.D. Boulder, Colo. | S. H. Large, M.D. Cleveland, Ohio | William P. Wherry, M.D. Omaha, Nebr. | Chicago |

# PAST OFFICERS OF THE ACADEMY

| Date | President | First Vice-President | Ex. Sec'y-Treas. | Comptroller | Meeting Place |
|---|---|---|---|---|---|
| 1931— | John F. Barnhill, M.D. Miami Beach, Fla. | Meyer Wiener, M.D. Coronado, Calif. | William P. Wherry, M.D. Omaha, Nebr. | S. H. Large, M.D. Cleveland, Ohio | French Lick, Ind. |
| 1932— | S. Hanford McKee, M.D. Montreal, Canada | Gordon B. New, M.D. Rochester, Minn. | William P. Wherry, M.D. Omaha, Nebr. | S. H. Large, M.D. Cleveland, Ohio | Montreal |
| 1933— | Burt R. Shurly, M.D. Detroit, Mich. | Nelson Miles Black, M.D. Miami, Fla. | William P. Wherry, M.D. Omaha, Nebr. | S. H. Large, M.D. Cleveland, Ohio | Boston |
| 1934— | John M. Wheeler New York, N. Y. | LeRoy A. Schall, M.D. Boston, Mass. | William P. Wherry, M.D. Omaha, Nebr. | S. H. Large, M.D. Cleveland, Ohio | Chicago |
| 1935— | Wells P. Eagleton, M.D. Newark, N. J. | W. Thornwall Davis, M.D. Washington, D. C. | William P. Wherry, M.D. Omaha, Nebr. | S. H. Large, M.D. Cleveland, Ohio | Cincinnati |
| 1936— | Frank E. Burch, M.D. St. Paul, Minn. | Gordon F. Harkness, M.D. Davenport, Iowa | William P. Wherry, M.D. Omaha, Nebr. | S. H. Large, M.D. Cleveland, Ohio | New York |
| 1937— | Lee W. Dean, M.D. St. Louis, Mo. | Bernard Samuels, M.D. New York, N. Y. | William P. Wherry, M.D. Omaha, Nebr. | S. H. Large, M.D. Cleveland, Ohio | Chicago |
| 1938— | Harry S. Gradle, M.D. Chicago, Ill. | E. M. Seydell, M.D. Wichita, Kan. | William P. Wherry, M.D. Omaha, Nebr. | S. H. Large, M.D. Cleveland, Ohio | Washington, D. C. |
| 1939— | George M. Coates, M.D. Philadelphia, Pa. | William W. Pearson, M.D. Des Moines, Iowa | William P. Wherry, M.D. Omaha, Nebr. | S. H. Large, M.D. Cleveland, Ohio | Chicago |
| 1940— | Frank E. Brawley, M.D. Chicago, Ill. | Arthur W. Proetz, M.D. St. Louis, Mo. | William P. Wherry, M.D. Omaha, Nebr. | S. H. Large, M.D. Cleveland, Ohio | Cleveland |
| 1941— | Frank R. Spencer, M.D. Boulder, Colo. | Everett L. Goar, M.D. Houston, Texas | William P. Wherry, M.D. Omaha, Nebr. | S. H. Large, M.D. Cleveland, Ohio | Chicago |
| 1942— | Ralph I. Lloyd, M.D. Brooklyn, N. Y. | Walter H. Theobald, M.D. Chicago, Ill. | William L. Benedict, M.D. Rochester, Minn. | S. H. Large, M.D. Cleveland, Ohio | Chicago |
| 1943— | James A. Babbitt, M.D. Philadelphia, Pa. | Sanford R. Gifford, M.D. Chicago, Ill. | William L. Benedict, M.D. Rochester, Minn. | S. H. Large, M.D. Cleveland, Ohio | Chicago |

| Date | President | First Vice-President | Ex. Sec'y-Treas. | Meeting Place |
|---|---|---|---|---|
| 1944— | Lawrence T. Post, M.D St. Louis, Mo. | C. Stewart Nash, M.D. Rochester, New York | William L. Benedict, M.D. Rochester, Minn. | Chicago |
| 1945— | Gordon B. New, M.D. Rochester, Minn. | Edmund B. Spaeth, M.D. Philadelphia, Pa. | William L. Benedict, M.D. Rochester, Minn. | Meeting Cancelled |
| 1946— | Gordon B. New, M.D. Rochester, Minn. | Edmund B. Spaeth, M.D. Philadelphia, Pa. | William L. Benedict, M.D. Rochester, Minn. | Chicago |

Index photographed at the
beginning for the convenience
of the microfilm user.

SUPPLEMENT

*to the*

# TRANSACTIONS

*of the*

# American Academy of Ophthalmology and Otolaryngology

---

# DIRECTORY

**ALPHABETICAL**

**GEOGRAPHICAL**

---

**OFFICIAL ROSTER**

1947

Published by the Douglas Printing Company, 109 North 18th Street, Omaha, Nebraska, for the American Academy of Ophthalmology and Otolaryngology. Editorial and business office, 100 First Avenue Building, Rochester, Minnesota.

# American Academy of Ophthalmology and Otolaryngology

# DIRECTORY

Names and Addresses Corrected to March 1, 1947

**Number immediately following a name indicates the year in which the Fellow was elected.**

**Number in circle in front of a name indicates classification as:**
① LIFE FELLOW ② SENIOR FELLOW ③ HONORARY FELLOW

**All Fellows without number designation are classified as Junior Fellows.**

**Please report change of address immediately to the Executive Secretary-Treasurer, William L. Benedict, M.D., 100 First Avenue Bldg., Rochester, Minnesota.**

# *Alphabetical*

## A

Aagesen, Walter J., '46
615 Anderson Bank bldg., Anderson, Ind. ALR

**Aageson, Carl W., '44**....1 S. Pinckney, Madison 3, Wisc. OP

Abbot, Edward A., '41.....225 High St., Holyoke, Mass. ALR

**Abbott, Albert J., '39**....100 E. Main St., Nanticoke, Pa. OP

**Abbott, Henry B., '41**.....574 So. Main, Geneva, N. Y. OP

**Abell, Harry Davis, '42**.....333 Broadway, Paducah, Ky. OP

**Abraham, Samuel Victor, '39**
1930 Wilshire blvd., Los Angeles 5, Cal. OP

Abrahamson, Ira A., '27
925 Union Trust bldg., Cincinnati, Ohio OP

Abrams, Henry, '46.....195 Nassau Street, Princeton, N. J. OP

Abrams, Murray, '45...100 Woodruff Ave., Brooklyn, N. Y. ALR

**Ackerman, Walter Gabe, '39**
104 S. Michigan av., Chicago 3, Ill. OP

**①Adams, A. L., '06**...Professional bldg., Jacksonville, Ill. OALR

**Adams, Charles J., '28**
Armstrong-Landon bldg., Kokomo, Ind. OALR

②Adams, D. S., '20
520 Hume-Mansur bldg., Indianapolis 4, Ind. OALR

**③Adams, Eldridge S. '20, Col (M.C.)**
c/o C.A.A., 1500 4th St., Santa Monica, Calif. ALR

Adams, Frank M., '24, 122 Waterman, Providence 6, R. I. ALR

③Adams, Frederick Wm., '20.....Carmel, Calif. OALR

**Adams, John Goldthwaite, '38**....24 Winter, Salem, Mass. ALR

**Adamski, Michael S., '40**....408 North St., Logansport Ind. OP

Adelman, Benjamin B., '35..186 Clinton av., Newark 5, N.J. OP

Adelstein, Fred, '41.....Rose bldg., Cleveland 15, Ohio ALR

Adix, H. Victor, Jr., '43
425 Medical Dental bldg., Portland 5, Ore. ALR

**Adkins, George E., '29**
Jackson Infirmary, Jackson 27, Miss. ALR

**Adler, Francis Heed, '29.313 S. 17th, Philadelphia 3, Pa. OP**

**Aebli, Rudolf, '31**.....30 E. 40th, New York 16, N. Y. OP

Agatston, Howard James, '46
57 West 57th St., New York 19, N.Y. OP

Agnew, Hobart McVickar, '38
17 Plymouth, Montclair, N. J. OP

Aguero, Alexis, '41.....San Jose, Costa Rica OP

Albaugh, Clarence Henry, '41
727 W. Seventh (Suite 1131), Los Angeles 14, Calif. OP

**Albers, Edward C., '36**.....104 W. Clark, Champaign, Ill. OP

**Albright, George C., '31**....104 S. Clinton, Iowa City, Ia. ALR

②Albright, Paul M., '22, Rear Adm. (MC) USN
Naval Retiring Board, Navy Dept., Washington 25, D.C. OALR

②Alden, Arthur M., '21.....Frisco bldg., St. Louis 1, Mo. ALR

**①Alderdyce, Wm. W., '09, 513 Madison av., Toledo 4, Ohio OALR**

**Alexander, Charles S., '36**
Medical Arts bldg., Houston 2, Texas OP

Alexander, Fred William, '42
10515 Carnegie Ave., Cleveland 6, Ohio ALR

Alexander, Lucian W., '34
Maison Blanche bldg., New Orleans 16, La. ALR

**Alexander, Margaret H., '37**....720 Talbot, Taylor, Texas ALR

Alexander, Samuel A., '33
1830 Eye St. N. W., Washington 6, D. C. ALR

Alexander, S. L., '33..Medical Arts bldg., Toronto 5, Can. ALR

Alfaro, Victor R., '31
2007 Massachusetts av., N.W., Washington 6, D.C. ALR

Alger, L. James, '36.....Box 328, Grand Forks, N. D. OP

Allan, William B., '34..410 Park av., New York 22, N. Y. OALR

Allen, Benjamin Israel, '46.1070 Park Ave., New York, N.Y. ALR

Allen, Cary D., '36, 350 State st., Portsmouth, N. H. ALR

②Allen, Charles E., '20
Waldheim bldg., Kansas City 6, Mo. OALR

Allen, Donald G., '36.1021 Prospect av., Cleveland 15, Ohio OP

**Allen, James Harrill, '38,**
University Hospitals, Iowa City, Ia. **OP**
**Allen, Orris T., '28,**
Rose Dispensary bldg., Terre Haute, Ind. **OALR**
**Allen, Thomas Dyer, '28,**
122 South Michigan av., Chicago 3, Ill. **OP**
Allen, Wilmer L., '43.....201 N. 8th St., Las Vegas, Nev. **ALR**
**Alliband, George Thomas '44**
1020 Medical Arts Bldg., Omaha, Nebr. **OP**
**Allis, Paul M., '36.....6 N. Brown, Lewistown, Pa. ALR**
Allman, Charles H., '41,
520 Commonwealth av., Boston 15, Mass. **ALR**
Almour, Ralph, '25,...57 W. 57th st., New York 19, N. Y. **ALR**
②Alonzo, Justo M., '46.Mercedes 1233, Montevideo, Uruguay **ALR**
Alpert, David R., '44, Major (MC),
28 Verndale, Brookline, Mass. **OP**
Alpert, Max, '46.....881 Lafayette St., Bridgeport, Conn. **ALR**
Alter, Francis William, Jr., '46
23 Second Ave., San Mateo, Calif. **ALR**
Altmann, Franz, '45..10 East 85th St., New York 28, N. Y. ALR.
②Altringer, A. N., '20, Professional bldg.,
Kansas City 6, Mo. **ALR**
Alvaro, M. E., '39....1151 Consolacao, Sao Paulo, Brazil **OP**
Alvis, Bennett Y., '28.900 Carleton bldg., St. Louis 1, Mo. **OP**
**Alvis, Edmond O., '37**
320 Hume-Mansur bldg., Indianapolis 4, Ind. **OP**
Alvis, Edmund B., '40....308 N. 6th st., St. Louis 1, Mo. **OP**
Alvis, Raymond W., '40.....Ohio bldg., Sidney, Ohio **OALR**
①Amberg, Emil, '06, 1244 W. Boston blvd., Detroit 2, Mich. **ALR**
Ambrose, Elmer C., '27.....910 Main, Trenton, Mo. **OALR**
Amdur, Louis A., '46
2540 Boulevard, 15 Exchange Pl., Jersey City 4, N.J. **OP**
Anderson, Edwin Rudolph, '38...220 Liberty, Warren, Pa. **OALR**
Anderson, Ernest Richard V., '46
1052 W. Sixth St., Los Angeles 14, Calif. **ALR**
Anderson, George H., '40
111½ Strongs av., Stevens Point, Wis. **ALR**
Anderson, Orrin E., '41...17 E. 61st st., New York, N. Y. **ALR**
Anderson, Ross Epting, '41
Standard Life bldg., Jackson 27, Miss. **ALR**
Anderson, Ruskin G., '41
753 N. Church St., Spartanburg, S.C. **OALR**
Anderson, Wm. Banks, '35..Duke Hospital, Durham, N. C. **OALR**
Andrews, Albert H., Jr. '36
700 N. Michigan av., Chicago 11, Ill. **ALR**
①Andrews, H. D., '12, 429 Oakland av., East Aurora, N. Y. **OP**
①Andrews, M. P., '15.....826 S. 8th, Manitowoc, Wis. **OALR**
Anneberg, Adrian R., '39..1332 N. Carroll st., Carroll, Ia. **ALR**
Annon, W. T., '23.....235 S. 15th, Philadelphia 2, Pa. **ALR**
Anthony, Daniel H., '29..2968 Iroquois Rd., Memphis, Tenn. **OP**
Appelbaum, Alfred, '36,
5905 Pacific blvd., Huntington Park, Calif. **OP**
**Apple, Carl, '38.....55 East Washington, Chicago 2, Ill. OP**
①Appleman, L. F., '13...308 S. 16th, Philadelphia 2, Pa. **OP**
Arbuckle, David S., '31, Akron S. & L. bldg., Akron 8, Ohio **OALR**
①Arbuckle, Millard F., '15, Col. (MC),
7118 Washington, St. Louis 5, Mo. **ALR**
Armentrout, Lewis H., '42
Medical Arts Bldg., Charleston, W. Va. **ALR**
**Armstrong, E. Leslie, '32**
Medical Arts bldg., Duluth 2, Minn. **ALR**
Armstrong, Richard C., '44
623 First Trust bldg., Pasadena 1, Calif. **OP**
①Arnold, Francis J., '12.....182 Pearl, Burlington, Vt. **OALR**
Arnold, Leon M., '46
1235 Park Avenue, New York 28, N. Y. **ALR**
**Arnold, Morton, '38.....781 Main, Willimantic, Conn. ALR**
Arnold, Ralph A., '42.....Duke Hospital, Durham, N. C. **ALR**
Aronson, Sidney S., '29...23 E. Ohio St., Indianapolis, Ind. **ALR**
Asbill, David S., '33..1512 Marion st., Columbia 49, S. C. **OP**
Asbury, Mary K., '25, 3549 Holly Lane, Cincinnati 8, Ohio **OP**
Ash, Arthur F., '44.710 Boulevard East, Weehawken, N. J. **ALR**
Ashley, Byron J., '37....2315 W. 13th st., Topeka, Kans. **OALR**
Ashley, Rea, '38.....384 Post St., San Francisco, Calif. **ALR**
Ashley, Richard Warner, '45...5811 8th av., Kenosha, Wis. **OP**
①Ashley, T. W., '15....5711 Eighth av., Kenosha, Wis. **OALR**
**Athens, Alvin G., '32..Medical Arts bldg., Duluth 2, Minn. OP**
**Atkins, John Leon, '39**
345 Bloor St. West, Toronto 5, Ont., Can **ALR**
Atkins, Joseph Preston, '45
250 S. 18th St., Philadelphia, Pa. **ALR**
②Atkinson, D. T., '18
Medical Arts bldg., San Antonio 5, Texas **OALR**
**Atkinson, John H., '36....168 Sterling, Watertown, N. Y. ALR**
Atkinson, Miles, '38..
310 Lexington av., New York 16, N.Y. **ALR**
**Atkinson, Thomas E., '29...Coronado bldg., Greeley, Colo. ALR**
**Atkinson, Walter S., '25....129 Clinton, Watertown, N. Y. OP**
**Atwood, Edward A., '32...360 Park av., Paterson 4, N. J. ALR**
②Auerbach, Julius, '18..897 Park av., New York 21, N. Y. **ALR**
Auslander, Milton Manuel, '45
1422 W. Erie Ave., Philadelphia 40, Pa. **ALR**
Austen, Willard Emerson, '40...10 Park av., New York, N.Y **ALR**
Austin, M. Fletcher, '42
206 Central Trust bldg., Sterling, Ill. **ALR**
Auten, Hanford Louis, Jr., '46
Dartmouth Eye Institute, Hanover, N. H. **OP**
**Avery, Elroy J., '34...11 N. Goodman, Rochester 7, N. Y. ALR**
Awtrey, Hugh H., '39..533 Patterson Bldg., Fresno, Calif. **OALR**
①Aynesworth, H. T., '12.....Box 1491, Waco, Texas **OALR**

## B

Bach, Edwin Clarence, '40
324 E. Wisconsin av., Milwaukee 2, Wis. **ALR**
Bacon, Lewis H., '42...300 Mahantongo St.,-Pottsville, Pa. **OP**
Baer, Alvin J., '42..1404 Bryant bldg., Kansas City 6, Mo. **OP**
Baeseman, R. W., '29....112 Main St., Allenhurst, N. J. **OALR**
**Bagley, Cecil Hopkins, '41, 2 East Read, Baltimore 2, Md. OP**
②Bahn, Charles A., '21
1028 Maison Blanche bldg., New Orleans 16, La. **OP**
①Bailey, Harold, '08.....Landers bldg., Springfield, Mo. **OALR**
Bailey, Paul, '37...1515 S.W. Myrtle st., Portland 1, Ore. **ALR**
**Bair, Hugo L., '43.....Mayo Clinic, Rochester, Minn. OP**
②Baird, Charles G., '20, Col. (MC)
AAG Nanking, China, APO 909 c/o PM San Francisco, Cal. **OALR**
Baird, James Mason, '38
511 Medical Arts bldg., Atlanta, Ga. **OP**
Baker, Daniel C., Jr., '39.903 Park Ave., New York 21, N.Y. **ALR**
**Baker, Frederick A., '44,**
4575 Motorway Dr., Pontiac 15, Mich. **OP**
**Baker, John Percy, '40...51 S. Oxford, Brooklyn 17, N. Y. ALR**
Baker, Lauchlin J., '16..310 Franklin St., Ogdensburg, N.Y **ALR**
Baker, Melvin C., '40....Heyburn bldg., Louisville 2, Ky. **OP**
Balding, Laurence Grant, '41
93 S. Madison av., Pasadena 5, Calif. **OP**
Baldwin, Gertrude Werder, '42 Carle Hospital Clinic
602 W. University Ave., Urbana, Ill. **OP**
**Baldwin, H. E., '30.....139 N. Vermilion, Danville, Ill. OALR**
Baldwin, Stephen Glidden, '42, Adams bldg., Danville, Ill. **ALR**

Baldwin, Thomas M., '43
222 Brodhead bldg., Beaver Falls, Pa. ALR
①Balentine, P. L., '15..1524 Chestnut, Philadelphia 2, Pa. OP
①Ballenger, H. C., '15.....723 Elm, Winnetka, Ill. ALR
①Ballin, M. J., '08.....25 E. 77, New York 21, N. Y. ALR
Ballon, David H., '24,
1022 Medical Arts bldg., Montreal 25, Quebec ALR
Bancks, Howard Raymond, '45
75 Lenox Rd., Brooklyn 26, N. Y. ALR
②Bane, W. M., '20..1612 Tremont Place, Denver 2, Colo. OALR
Bankes, Claude Webster, '38.....212 N. 6th, Reading, Pa. OP
①Barber, F., '13.....75 S. Fitzhugh, Rochester 8, N. Y. OP
Barbour, Fleming A., '41....342 Bradley av., Flint, Mich. OP
①Bardenheier, F. G. A., '14, Frisco bldg., St. Louis 1, Mo. ALR
②Barkan, Hans, '18, 2351 Clay st., San Francisco 15, Calif. OP
Barkan Otto, '29.....490 Post, San Francisco 2, Calif. OP
Barkhorn, Charles W., '33..223 Roseville Av., Newark, N.J. ALR
②Barlow, Roy, '20, R. F. D. No. 2,
Charles Town, Jeff County, West Va. ALR
Barnaby, Henry C., '16, Capt. (MC)
Walter Reed Gen. Hosp., Washington, D.C. ALR
Barnes, Lancelot, '35.....170 St. George, Toronto 5, Can. OP
Barnes, Mark Hopkins, '42
324 E. 41st st., New York 17, N. Y. OALR
Barnet, Emmanuel, '46
214 W. Grand Av., Wisconsin Rapids, Wisc. ALR
Barnett, Irving F., '33, 30 N. Michigan av., Chicago 2, Ill. OALR
Barnett, Saul E., '29.....10 Peterboro, Detroit, Mich. ALR
Baron, Shirley Harold, '35
516 Sutter St., San Francisco 2, Calif. ALR
Barone, Michael Horace, '42
468 Delaware Ave, Buffalo 2, N.Y. ALR
Barr, A. S., '32...1st Nat'l Bank bldg., Ann Arbor, Mich. OP
Barr, William T., '38
1327 Medical Arts bldg., Dallas 1, Texas ALR
Barrett, John H., '43...1304 Walker av., Houston 2, Texas ALR
Barry, Wm. B., '42..441 Huntington rd., Kansas City, Mo. ALR
Barton, John F., '31
1346 Fourteenth av., Longview, Wash. ALR
Barton, William C., '32
813 Don Gaspar av., Sante Fe, N. M. OALR
Bass, A. L., '24.....Heyburn bldg., Louisville 2, Ky. OALR
Bassen, Edward J., '30..70 E. 66th st., New York 21, N. Y. OP
Bassett, David I., '40
1075 Grand Concourse, New York 52, N. Y. ALR
Batson, Oscar V., '34.....133 S. 36, Philadelphia 4, Pa. ALR
Battles, Newell H., '37..P.O. Box 1180, Idaho Falls, Idaho OALR
Bau, Hyman William, '45
3216 W. Roosevelt Rd., Chicago 24, Ill. ALR
Bauer, Arthur S., '44,
Suite No. 812, 1801 Eye st. NW, Washington 6, D. C. ALR
Bauer, Charles E. '46..11 Cannon St., Poughkeepsie, N. Y. OP
①Baum, H. L., '17.....Republic bldg., Denver 2, Colo. ALR
Baum, William W., '30....206 Guardian bldg., Salem, Ore. OALR
Bausch, Carl Philipp, '38..36 Pearl st., Hartford 3, Conn. OP
①Beach, S. Judd, '14....704 Congress, Portland 4, Maine OP
Beal, Homer A., '26,
1000 Professional bldg., Kansas City, Mo. ALR
Beale, Allen W., '40.....308 N. Park av., Warren, Ohio ALR
Beall, John G., '44
118½ E. Front st., Traverse City, Mich. OP
Beals, C. Wearne, '30....28 W. Scribner av., DuBois, Pa. ALR
Beam, A. Duane, '39
85 Kercheval av., Grosse Point 30, Mich. OP
①Beattie, Robert, '06
David Whitney bldg., Detroit 26, Mich. OALR
②Beattie, William, '21...5 Derbyshire pl., Utica 3, N. Y. OALR
①Beatty, H. G., '16..150 E. Broad, Columbus 15, Ohio PI S-ALR
Beauchamp, Mark L., '44,
1136 W. Sixth st., Los Angeles 14, Calif. ALR
Beavis, James Olin, '36
900 Fidelity Medical bldg., Dayton 2, Ohio ALR
Beck, A. L., '27.....421 Huguenot, New Rochelle, N. Y. OALR
Beck, Frederick W., '40
218 First Security Bank bldg., Ogden, Utah ALR
Beck, Robert J., '35....19 Hope St., Saranac Lake, N.Y. OP
Becker, Oscar J., '41..30 N. Michigan av., Chicago 2, Ill. ALR
Beckner, William Forrest, '38
West Virginia bldg., Huntington 1, W. Va. ALR
①Bedell, Arthur J., '12.....344 State, Albany 6, N. Y. OP
①Beem, Ione F., '16
30 N. Michigan Avenue, Chicago 2, Ill. OALR
Beetham, William P., '45..108 Bay State Rd., Boston, Mass. OP
Begle, Howell L., '28
2730 E. Jefferson av., Detroit 7, Mich. OP
Behen, William C., '33.535 S. Capitol av., Lansing 15, Mich. ALR
Behrens, Alvin, '33
9 Prospect Park West, Brooklyn 15, N. Y. OP
Beideman, J. E., '29
Montgomery Trust Arcade, Norristown, Pa. OP
①Beil, J. Wallace (Retired) '18
1205 W. 59th St., Kansas City, Mo. OALR
Beil, Wallace C., '43
912 American Bank bldg., New Orleans, La. OP
Beinfield, Henry Harold, '38
760 Eastern Parkway, Brooklyn 13, N. Y. ALR
Beisbarth, Carl, '31
3720 Washington blvd., St. Louis 8, Mo. OP
Bell, John Gordon, '40
1835 Eye Street, N. W., Washington 6, D.C. ALR
Bell, Julius W., '36....200 E. 16th St., New York 3, N.Y. ALR
Bellows, John G., '35,
Rm. 1729, 30 N. Michigan, Chicago 2, Ill. OP
Bellucci, Richard John, '46
121 East Sixtieth St., New York 22, N.Y. ALR
Bender, H. A., '30.....Black bldg., Waterloo, Iowa ALR
Bender, M. S., '30.....88-05 150th st., Jamaica 2, N. Y. ALR
Also: 121 E. 60th, New York 22, N.Y.
Bender, Rosarie Rossell, '45
195 Linwood Ave., Buffalo, N. Y. ALR
②Benedict, William L., '19, Mayo Clinic, Rochester, Minn. OP
Benkwith, Karl B., '42,
209 Lexington rd., Montgomery, Ala. OP
Benson, C. E., '40.....515 6th St., Bremerton, Wash. OALR
Benson, Kenneth H., '44
1114 North 2nd St., Harrisburg, Pa. ALR
②Bentley, Neil I., '22
David Whitney bldg., Detroit 26, Mich. OALR
Benz, Alvin H., '41
Medical Arts Bldg., Chattanooga, Tenn. OP
②Berens, Conrad, '22..35 E. 70th, New York 21, N.Y. PI S-OP
Berger, Isidore, '40
888 Grand Concourse, Bronx, New York 51, N. Y. ALR
Berger, Louis, '40.....30 N. Michigan Ave., Chicago 2, Ill. OP
Berger, Meyer D., '44..681 Ocean av., Brooklyn 26, N. Y. ALR
Berger, Murray, '38
101 Central Park West, New York 24, N.Y. ALR
Bergida, Jerome, '40..1615 Dorchester Road, Brooklyn, N.Y. ALR
Bergmann, M. B., '32
959 Bushwick av., Brooklyn 21, N. Y. OP

**Berke, Raynold N., '34**.....430 Union, Hackensack, N. J. OP
Berkley, William Leneave, '46, Capt. (MC) USN
U.S. Naval Hospital, St. Albans, N.Y. OP
**Berliner, Milton L., '31**...57 W. 57th, New York 19, N. Y. OP
Berman, Harry, '46.128 Lincoln St., Salt Lake City 2, Utah ALR
**Berman, Harry L., '43**..1142 Chapel, New Haven 11, Conn. ALR
**Berman, Philip G., '35**.....174 Central, Lowell, Mass. OP
Bernasconi, Ezio Joseph, '45
716 River Av., Providence, R. I. OP
①Bernatz, C. F., '17.....Park bldg., Pittsburgh 22, Pa. OP
**Berner, George E., '37**..5430 Greene, Philadelphia 44, Pa. OP
Berney, Irving Vincent, '43
31 Lincoln Park, Newark 2, N. J. ALR
**Bernheimer, L. Benno, '30**
104 S. Michigan av., Chicago 3, Ill. ALR
Berns, Charles, '35
11408 Whitmore av., Cleveland 8, Ohio ALR
**Bernstein, David, '44**.....1342 51st St., Brooklyn, N. Y. ALR
①**Berry, D. F., '11**
Bankers Trust bldg., Indianapolis 4, Ind. ALR
**Berry, Gordon, '30**.....36 Pleasant, Worcester 8, Mass. ALR
**Bertolet, John A., '37**....1530 Locust, Philadelphia 2, Pa. ALR
Bertrand, George Joseph, '42, 73 Main St., Montpelier, Vt. OP
Best, L. Kent, '35
107 G. Ashley av., Charleston 16, S. C. OP
**Beyer, A. G., '25**.....Carew Tower, Cincinnati 2, Ohio OALR
②Beyer, Louis J., '18...449 Delaware av., Buffalo 2, N. Y. ALR
**Beyer, Theodore E., '31**..227 Sixteenth st., Denver 2, Colo. ALR
**Biehn, J. Telford, '37,**
282 Wellington St., Sarnia, Ont., Canada OALR
Bilchick, Edwin B., '35
133 E. 58th St., New York 22, N.Y. ALR
**Billings, Gilbert M., '39**
110 S. Sterling, Morganton, N. C. ALR
**Billings, Robert J., '35**...500 Penn av., Pittsburgh 22, Pa. OP
Billingsley, J. A., '38, Huron bldg., Kansas City 10, Kans. OP
①Binger, H. E., '17, Lowry Med. bldg., St. Paul 2, Minn. OALR
①Binkley, Roy S., '17....Fidelity bldg., Dayton 2, Ohio OALR
Birch, John R., '39
1010 Maccabees bldg., Detroit 2, Mich. ALR
**Birdsall, Clarence H., '37**....30 Summer, Haverhill, Mass. ALR
Birge, Henry L., '39.....179 Allyn st., Hartford, Conn. OP
Birsner, Louis J., '26..1232 Missouri bldg., St. Louis, Mo. ALR
**Bishop, Frederick J., '36**
Medical Arts bldg., Scranton 3, Pa. ALR
**Bisno, Daniel, '43**.....4500 Olive, St. Louis 8, Mo. OP
Bizzell, Marcus Edward, '46
139 W. Walnut Street, Goldsboro, N.C. ALR
①Black, Nelson Miles, '03, Huntington bldg.,
Miami 32, Fla. OP
**Black, Paul, '27**.....Stuart bldg., Lincoln 8, Nebr. OALR
Black, Vernon Anderson, '45..105 Hawes St., Wharton, Tex. OP
**Black, W. Byron, '31**.Professional bldg., Kansas City 6, Mo. ALR
①Black, W. D., 1900..Metropolitan bldg., St. Louis 3, Mo. ALR
Blackford, Roger W., '44,
503 Hume-Mansur bldg., Indianapolis 4, Ind. ALR
**Blair, M. W., '28**
N. E. cor. 20th and Chestnut, Philadelphia 3, Pa. OP
①Blair, W. W., '17.....Woodland rd., Pittsburgh 6, Pa. OP
**Blaisdell, Irl Hazard, '41**
Medical Arts bldg., Syracuse 2, N. Y. ALR
**Blankfeld, Nathan, '36**
538 9th St., Brooklyn 15, N. Y. ALR
Blankstein, Samuel S., '42,
208 E. Wisconsin av., Milwaukee 2, Wisc. OP
**Blasdel, Tyler David, '40**
110½ S. 18th st., Parsons, Kan. ALR
**Blassingame, C. D., '23**
899 Madison av., Memphis 3, Tenn. ALR
**Blaydes, J. E., '43**
First Nat'l Bank bldg., Bluefield, W. Va. OP
①Bledsoe, R. W., '07
First Nat'l Bank bldg., Covington, Ky. OALR
②Blickenstaff, A. J., '22
First Nat'l Bank bldg., Peoria 2, Ill. OALR
**Bloch, Charles L., '43**....2905 Dundee rd., Louisville, Ky. OP
**Blomberg, Thorsten E., '33**.501 Seventh st., Rockford, Ill. ALR
Blonder, Edwin J., '34, 25 E. Washington st., Chicago 2, Ill. ALR
**Bloom, C. Hugh, '32**.....65 N. 3rd, Easton, Pa. ALR
**Bloom, Samuel M., '46**.121 E. 60th St., New York 22, N.Y. ALR
①Blue, J. B., '16.....62 N. Main, Memphis 3, Tenn. OALR
**Blumenfeld, Louis, '39**
221 Brooklyn av., Brooklyn 13, N. Y. ALR
Blumklotz, Bruno, '43
25 E. Washington st., Chicago 2, Ill. ALR
**Boaz, Emmett Daniel, '38**
3710 Fifth av., Pittsburgh 13, Pa. ALR
**Bochner, Maxwell K., '33**..329 Bloor st. W., Toronto, Can. OP
**Bockoven, Sterling, '38**
1752 Mass. av. N.W., Washington 6, D. C. OP
Boden, Worthey C., '42, 6537 Kenwood, Chicago 37, Ill. ALR
**Bodenheimer, Ernst, '43**.1212 Eutaw pl., Baltimore 17, Md. OP
②Boeckmann, Egil, '18
Lowry Medical bldg., St. Paul 2, Minn. OALR
**Boehs, Charles J., '37**
Medical Arts bldg., San Antonio 5, Texas ALR
**Boemer, L. C., '30,**
3720 Washington blvd., St. Louis 8, Mo. ALR
**Bogart, Donald Weeks, '40**
20 East 53rd st., New York 22, N. Y. OP
Bogdasarian, Robert Michael, '45
108 Oak St., Binghamton, N.Y. ALR
**Boggs, Frank Clinton, '38**.....Mills bldg., Topeka, Kan. OP
**Boice, Ralph E., '35**.....316 Michigan, Toledo 2, Ohio OP
Boies, Lawrence R., '36
Medical Arts bldg., Minneapolis 2, Minn. ALR
①Boiler, William F., '12, Major (MC),
206 Dey bldg., Iowa City, Iowa OALR
Boland, Wm. T., '41....378 W. Church st., Elmira, N. Y. OP
**Boles, Wm. M., '41,**
4132 St. Charles Ave., New Orleans 15, La. OP
Bolman, Harold Robert, '38
6½ S. Main, Monroeville, Ohio OP
Bolotow, Nathan A., '29.126 Waterman, Providence 6, R. I. ALR
Bolton, Wilbur Minnis, '46
1020 S.W. Taylor St., Portland 1, Ore. ALR
Bonaccolto, Girolamo, '37
123 East 61st st., New York 21, N. Y. OP
**Bond, Floyd M., '41**...625 Broadway, San Diego 1, Calif. OP
③**Bone, Herman D., '18**.....19 Pleasant, Gardner, Mass. OALR
Bonham, William Lawrence, '39
Medical Arts bldg., Oklahoma City 2, Okla. ALR
Bonner, Wm. F., '26....McCloskey V.A.H., Temple, Tex. OALR
**Bonner, William Richard, '42**
3 W. White st., Summit Hill, Pa. OP
②Bordley, James Jr., '18
2630 Guilford Ave., Baltimore 18, Md. OALR
**Borgeson, E. J., '30**
Medical Arts bldg., Minneapolis 2, Minn. OP
Borley, Wm. E., '39
450 Sutter Bldg., San Francisco, Calif. OP

Bornstein, Max, '42....308 Will Sales bldg., Louisville, Ky. ALR

Boss, Eugene George, '33..1537 Main, Springfield 3, Mass. OALR

Bostwick, D. S., '34.....106 Linwood av., Ardmore, Pa. ALR

Boswell, Robert Echo, '41..Fidelity bldg., Dayton 2, Ohio ALR

Bothman, Louis, '30..30 N. Michigan av., Chicago 2, Ill. OP

②Boucher, R. B., '19
Medical-Dental bldg., Vancouver, B. C., Can. OALR

Bourne, Malcolm G., '37...110 W. 3rd, Jamestown, N. Y. OP

②Bouvy, Harry M., '20
800 Pittock blk., 10th and Washington, Portland, Ore. OALR

Bouvy, L. B., '27.....Foley bldg., La Grande, Ore. OALR

Bower, Richard L., '43.1103 Grand av., Kansas City 6, Mo. OALR

Bowers, Wesley C., '36...17 E. 61st, New York 21, N. Y. ALR

①Boyce, G. H., '12
1st Nat'l Bank bldg., Iron Mountain, Mich. OALR

Boyce, Wm. A., '31.Roosevelt bldg., Los Angeles 14, Calif. OP

Boyd, Charles Wilson, '40
111 W. Adams St., Jacksonville, Fla. OP

Boyd, Greydon G., '39, Capt. (MC) USN,
307 East 44th st., New York, N. Y. ALR

Boyd, Harold M. E., '44
1136 W. 6th St., Los Angeles 14, Calif. ALR

Boyden, Guy Lee, '39.1735 N. Wheeler, Portland 12, Ore. ALR

Boyes, T. L., '32....654 Madison av., New York 21, N. Y. OP

Bozer, Herrmann E., '23
468 Delaware av., Buffalo 2, N. Y. ALR

Bradford, George Edwin, '41
Nissen bldg., Winston-Salem, N. C. ALR

Bradish, Robert F., '42, Col. (M.C.),....Address Unknown OP

Bradley, Jeter C., '34
900 17th st., N.W., Washington 6, D. C. ALR

Bradley, John W., '37..Burns bldg., Colorado Springs, Colo. ALR

Brady, Charles Henry, 43..205 Ilgenfritz bldg., Sedalia, Mo. ALR

Braley, A. E., '39..635 W. 165th St., New York 32, N.Y. OP

Brandenburg, Kenneth C., '37
110 Pine av., Long Beach 2, Calif. OALR

Brandenburg, Nora B., '36
700 N. Michigan av., Chicago 11, Ill. ALR

Brandfield, Ira Irvin, '40..305 W. 2nd St., Frederick, Md. ALR

Brandon, James R., '44,
800 First Nat'l Bank bldg., Canton, Ohio ALR

Brandwan, Samuel R., '37....Rose bldg., Cleveland 5, Ohio OP

Branigan, C. Hugh, '33....17 Kraft av., Bronxville, N. Y. OP

Brannin, Daniel, '36...Medical Arts bldg., Dallas 1, Texas OP

①Braunlin, Wm. H., '20
Marion Nat'l Bank bldg., Marion, Ind. OALR

Brav, Solomon S., '46
2027 Spruce St., Philadelphia 41, Pa. OP

①Brawley, F. E., '07...30 N. Michigan av., Chicago 2, Ill. OP

Brawner, Leon E., '29
Thelon "By the Sea", St. Simon's Island, Ga. ALR

Bredlau, Ernest A., '29..2753 W. North, Chicago 47, Ill. ALR

Breeding, Earl G., '32
1801 Eye st. N.W., Washington 6, D. C. ALR

②Brehmer, Harrison L., '20
First Nat'l Bank bldg., Albuquerque, N. M. OALR

Breisacher, Carl F., '44
432 Medical Arts Bldg., Charleston 1, W. Va. OP

Breitstein, M. Lewis, '37
1213 Eutaw pl., Baltimore 17, Md. ALR

Brennan, James William, '45.11 Horton Place, Buffalo, N.Y. OP

Bressler, J. L., '34...Medical Arts bldg., Houston 2, Texas OP

Breuning, Paul H., '32...133 E. 58th, New York 22, N. Y. ALR

②Brewer, John D., '20
Baird Brewer Gen. Hosp., Dyersburg, Tenn. OALR

Brewster, H. F., '32.913 Richards bldg., New Orleans, La. ALR

Briant, Thomas E., '29...28 E. Main, Welland, Ont., Can. ALR

②Bribach, Eugene J., '19
Professional bldg., Atchison, Kan. OALR

Bricker, Sacks, '31.1101 W. Wyoming, Philadelphia 40, Pa. ALR

②Brickley, Daniel W., '21
Uhler-Phillips bldg., Marion, Ohio OALR

Brickley, Daniel W., Jr., '38
401 Uhler Phillips bldg., Marion, Ohio ALR

Bridgett, Frank Aloysius, '42
3332 Chestnut st., Philadelphia 4, Pa. ALR

Briggs, Henry Harrison, Jr., '41
611 City bldg., Asheville, N.C. OP

Brighton, George R., '36
580 Park av., New York 21, N. Y. ALR

Briglia, Frank J., '36..819 Wharton, Philadelphia 47, Pa. ALR

Brikates, Peter, '45.....140 E. 54th St., New York, N.Y. OP

Brindley, Benjamin, '40,
304 W. Washington, Madison 3, Wis. OP

Brinkerhoff, David E., '39....Grunow Clinic, Phoenix, Ariz. ALR

Bristow, Walter J., '35
1517 Hampton, Columbia 29, S. C. OALR

Broadwell, Stuart, Jr., '37...103½ N. 5th, Springfield, Ill. ALR

Brobeck, Von Haller, '32
Ferguson bldg., Colorado Springs, Colo. OP

Brodnitz, Friedrich S., '44,
667 Madison av., New York 21, N. Y. ALR

①Brodrick, Frank W., '11.....302 First av., Sterling, Ill. OALR

Brody, Yale, '46
919 Bank of America Bldg., Stockton, Calif. OP

Brook, Jeffrey J., '34
231 W. Wisconsin av., Milwaukee 3, Wis. ALR

Brooks, Clyde M., '45,
105 S. University ave., Carbondale, Ill. ALR

①Brooks, E. H., '15.....Zuelke bldg., Appleton, Wis. OALR

②Brooks, Earl B., '19....128 N. 13th st., Lincoln 8, Nebr. OALR

Brooks, Jack E., '43
c/o P. O. Box 1539, Santa Fe, N. Mex. ALR

Brooks, Morris J., '34...455 Ocean av., Brooklyn 26, N. Y. ALR

Brooks, Richmond C. A., '39
1150 Connecticut av., Washington 6, D. C. ALR

Broones, Otto, '36.....302 W. 87th, New York 24, N. Y. ALR

Brown, Albert L., '26.....Carew Tower, Cincinnati 2, Ohio OP

Brown, Andrew George, '42.717 Dul'ont bldg., Miami, Fla. OP

Brown, Audrey O., '27.5057 Woodward av., Detroit 2, Mich. OALR

Brown, Charles Lafayette, '38
713-15 Richards bldg., New Orleans 12, La. ALR

Brown, Charles Wm., '27
Bank of America bldg., San Diego 1, Calif. ALR

Brown, C. J., '34, Capt. (MC) USN
U.S. Naval Hospital, St. Albans, L.I., N.Y. ALR

①Brown, Clayton M., '12
510 Delaware av., Buffalo 2, N. Y. ALR

Brown, Clyde O., '28
3720 Washington blvd., St. Louis 8, Mo. ALR

Brown, David Edward, '41
520 Hume-Mansur, Indianapolis 4, Ind. ALR

Brown, Earl Henderson, '39...130 S. Scott, Tucson, Ariz. ALR

Brown, Harold Ethan, '46..581 Bulen av., Columbus 9, Ohio OP

Brown, Harold W., '44..15 Park av., New York 16, N. Y. OP

Brown, Henry A., '43
1522 Damon Ct. S.E., Rochester, Minn. ALR

①**Brown, J. M., '11...1136 W. 6th, Los Angeles 14, Calif. ALR**
②Brown, John C., '20, Medical Arts bldg.,
St. Paul 2, Minn. **OALR**
①Brown, John E., '01..135 Preston rd., Columbus 9, Ohio **OALR**
Brown, Lester A., '39....815 Doctors bldg., Atlanta, Ga. **ALR**
②Brown, Mortimer G., '21
State Tower bldg., Syracuse 2, N. Y. **ALR**
①Brown, W. Harold, '12
925 W. Georgia st., Vancouver, B. C., Can. **OALR**
Brown, Wilbur Max, '43..49 W. 2nd St., Mansfield, Ohio **OP**
Brownell, Durwin H., '40,
3520 Third av., San Diego 3, Calif. **ALR**
**Brownell, Morton E., '28.106 W. Douglas, Wichita 2, Kan. OP**
Browning, Andrew J., '35....Mayer bldg., Portland 5, Ore. **OP**
**Browning, Birt Lee, '35.....208 Grand, Newburgh, N. Y. ALR**
Brownsberger, Sidney B., '38
727 W. 7th st., Los Angeles 14, Calif. **OP**
**Bruce, G. M., '30....635 W. 165th st., New York 32, N.Y. OP**
①Bruder, Joseph, '08....57 W. 57th, New York 19, N. Y. **OALR**
Brumbaugh, John D., '34,
75 North Wheaton Road, Akron 13, Ohio **OALR**
Brumm, Lewis Patrick, '46
726 Carew Tower, Cincinnati, Ohio **OP**
②Brumm, Seth A., '20
Medical Arts bldg.,Philadelphia 3, Pa. **OALR**
**Bruner, Abram B., '23..Guardian bldg., Cleveland 14, Ohio OP**
Bruner, Claude R., '41
Exchange Nat'l Bank bldg., Columbia, Mo. **OALR**
①**Bruner, Wm. E., '05..Guardian bldg., Cleveland 14, Ohio OP**
**Brunk, Clifford F., '39**
7815 E. Jefferson av., Detroit 14, Mich. **ALR**
Brunner, George Harmon, '40
603 Navarro st., San Antonio, Texas **OP**
③**Brunner, Hans, '35.307 N. Michigan av., Chicago 1, Ill. ALR**
**Brusegard, James F., '33.....414 Main, Red Wing, Minn. OP**
①Brust, H. O., '13..820 S. Crouse av., Syracuse 1, N. Y. **ALR**
Bryan, James H., '43
242 Bristol rd., Webster Groves, Mo. **OP**
Bryan, James L., '28
Bennie-Dillon bldg., Nashville 3, Tenn. **OALR**
**Bryan, William T. K., '42**
3720 Washington blvd., St. Louis 8, Mo. **ALR**
Brant, Ben L., '31
3875 Wilshire blvd., Los Angeles 5, Calif. **ALR**
Brant, Frank L., '37.....2622 Greenwood, Shreveport, La. **ALR**
**Buchanan, Norman D., '25**
129 Murray, Peterborough, Ont., Can. **OALR**
Buchbinder, Murray W., '43
94-12 109th Ave., Ozone Park, N.Y. **ALR**
**Bucher, Walter M., '34..4187 Pearl rd., Cleveland 9, Ohio ALR**
**Buck, Clair A., '31.....191 Glen st., Glen Falls, N.Y. ALR**
**Buckingham, Tracy W., '31**
405 Broadway, Bismarck, N. D. **ALR**
**Buckman, Lewis T., '32..83 S. Franklin, Wilkes-Barre, Pa. OALR**
**Buckman, Samuel T., '40.70 S. Franklin, Wilkes-Barre, Pa. OALR**
**Budetti, Joseph A., '43..90 Sherman Ave., Mt. Vernon, N.Y. ALR**
**Buffington, Wiley R., '35**
Hibernia Bank bldg., New Orleans 12, La. **OP**
**Buffum, Edward S., '36....40 W. North, Buffalo 2, N. Y. ALR**
**Bullard, Carleton Wm., '32...120 Genesee, Auburn, N. Y. ALR**
**Bullis, John A. E., '39**
3875 Wilshire blvd., Los Angeles 5, Calif. **OALR**
**Bullwinkel, H. Griffin, '31, Capt. (MC), USNR**
Glenwood Gardens, Yonkers 2, N. Y. **ALR**
**Bulson, Eugene L., '28..347 W. Berry, Ft. Wayne 2, Ind. OALR**
**Bunker, Paul G., '37.....Newberry bldg., Aberdeen, S. D. ALR**
Bunnell, Chester Wm., '35
1316-27th Ave., San Francisco, Calif. **ALR**
Buonaguro, Michael J., '31
589 Lorimer, Brooklyn 11, N. Y. **OP**
Burch, Edward P., '39.....Hamm bldg., St. Paul, Minn. **OP**
①**Burch, Frank E., '07..754 Linwood pl., St. Paul 5, Minn. OP**
③Burchell, Edgar B., '44, New York Eye and
Ear Infirmary, 2nd av., cor. 13th, New York 3, N.Y.
**Burchfield, George W., '36....309 Court, Maryville, Tenn. ALR**
**Burdick, Austin F., '25**
P. O. Drawer 1378, Lansing 4, Mich. **OALR**
Burgess, John L., '40
904 Medical Arts bldg., Waco, Tex. **ALR**
Burgess, Taylor S., '39
310 Medical Arts bldg., Atlanta 3, Ga. **ALR**
**Burgman, James Everette, '38**
8 W. Main, Springfield, Ohio **ALR**
Burian, Hermann M., '38
520 Commonwealth av., Boston 15, Mass. **OP**
Burke, B. Russell, '35
478 Peachtree St., N.E., Atlanta 3, Ga. **ALR**
**Burke, John R., '27.....207 Elm, Holyoke, Mass. OALR**
①**Burke, T. A., '06.....Rose bldg., Cleveland 15, Ohio OALR**
Burman, Daniel, '45.....121 1st St., Newburgh, N.Y. **ALR**
**Burman, Herman J., '35**
1235 Grand Concourse, New York 52, N. Y. **ALR**
**Burnham, Howard H., '37**
Medical Arts bldg., Toronto 5, Can. **ALR**
Burns, Coleman Craig, '46
Medical Arts bldg., Abilene, Tex. **ALR**
②**Burns, S. S., '19.Missouri Theatre bldg., St. Louis 3, Mo. ALR**
**Burritt, N. W., '30....30 Beechwood road, Summit, N. J. ALR**
**Burston, Herschel, '43,**
1917 Wilshire blvd., Los Angeles 5, Calif. **ALR**
Burtoff, Samuel, '44
2025 Eye St., N.W., Washington 6, D.C. **ALR**
Burton, Edwin W., '37.307 E. Market, Charlottesville, Va. **OP**
Busby, William, Jr., '45
Sect. 5, Madigan Gen. Hosp., Tacoma, Wash. **ALR**
**Bushman, L. B., '27**
City National Bank bldg., Omaha 2, Nebr. **OALR**
Butler, Edmund John, '42, 25 Garden st.,
Cambridge 38, Mass. **ALR**
Butler, Harry, '37.....77 Broadway, Bangor, Maine **ALR**
①Butt, Miriam M., '08,
333 Seymore st., Philadelphia 44, Pa. **OP**
③**Buvinger, Charles W., '21**
50 Washington, East Orange, N. J. **OALR**
**Buzzard, Josiah F., '31..1110 Thirteenth av., Altoona, Pa. OALR**
**Byrne, Harry Vincent, '38.....301 Essex, Lawrence, Mass. OP**
**Byrne, J. Arthur, '41.....16 Elm, Morristown, N. J. ALR**
①**Byrnes, Harry F., '10..67 Chestnut, Springfield 5, Mass. OALR**
Byrnes, Victor A., '43 Col. (MC)
City Hospital, 1515 Lafayette St., St. Louis, Mo. **OP**

## C

Cabot, Clyde M., '38
5629 - 12th av. S., Minneapolis, Minn. **ALR**
**Cady, D. W., '30....65 N. Madison av., Pasadena 1, Calif. OP**
**Cady, F. J., '32.....402 S. Jefferson av., Saginaw, Mich. OALR**
**Cahill, James F., '37.....428 S. Salina, Syracuse 2, N. Y. OP**

Calcote, Royal J., '36
541 Donaghey bldg., Little Rock, Ark. OP
①Caldwell, Robert, '12
New Donaghey bldg., Little Rock, Ark. OALR
①Calhoun, F. Phinizy, '08
478 Peachtree st. N.E., Atlanta 3, Ga. OP
Calhoun, Ferdinand Phinizy, Jr., '46
478 Peachtree St., Atlanta, Ga. OP
Callahan, Alston, '38
Medical College of Alabama, Birmingham 5, Ala. OP
Calvert, Raymond R., '36...314 N. 6th st., Lafayette, Ind. OP
Cameron, Walter Courtney, '46
1103 Medical Arts Bldg., Tacoma 2, Wash. OP
Cammisa, James J. Vincent, '43, Comdr. (MC), USN,
44 Leamington rd., Brighton 35, Mass. OALR
②Camp, W. E., '19
Medical Arts bldg., Minneapolis 2, Minn. OALR
Campbell, A. A., '80.....86 Bloor st. W., Toronto, Can. ALR
②Campbell, C. H., '18.2514½ Thirteenth, Columbus, Nebr. OALR
③Campbell, Duncan A., '19
David Whitney bldg., Detroit 26, Mich. OALR
Campbell, Edward H., '38
Spruce St. Med. bldg., 19th & Spruce, Philadelphia, Pa. ALR
Campbell, Glenn S., '37
340 No. Oxford av., Los Angeles 4, Calif. ALR
Campbell, Mac D., '27
Washington Square Bldg., Royal Oak, Mich. OALR
Campbell, Paul A., '39
Suite 314, 700 N. Michigan av., Chicago, Ill. ALR
Campbell, Sherburne, '36, 270 Center St., Wallingford, Conn. OALR
Campbell, William, '39.144 S. Harrison, E. Orange, N. J. ALR
Campbell, Wm. E., Jr., '36
Medical Arts bldg., Atlanta 3, Ga. OP
Canfield, Norton, '33...789 Howard av., New Haven 4, Conn. ALR
Cannon, Harold J., '43
536 W. Wisconsin av., Milwaukee 3, Wis. ALR
Capeles, Thomas Francis, '42
191 Merrimack St., Haverhill, Mass. OALR
Cappeta, Domenico, '46
125 East 84th St., New York 28, N.Y. OP
Cardwell, Edgar P., '29.....965 Broad st., Newark, N. J. ALR
Carelli, Paul Vincent, '41
3626 W. Chicago av., Chicago 51, Ill. OP
Carey, Edward Thomas, '45...415 Howes bldg., Clinton, Ia. ALR
Carl, Evan Elsworth, '40
606 W. Wisconsin av., Milwaukee 3, Wis. OP
Carmody, Raymond F., '37.....504 Broadway, Gary, Ind. OP
Caron, Armand L., '39....36 Pleasant, Worcester 8, Mass. ALR
Carpenter, Chapin, '30..1930 Chestnut, Philadelphia 3, Pa. OP
Carrasquillo, Honorio Fernando, '41
P. O. Box 128, Utuado, Puerto Rico OP
Carroll, Frank D., '37.635 W. 165th, New York, 32, N. Y. OP
Also: Blind Brook Lodge, Rye, N. Y.
Carroll, Walter J. E., '37..5 Chestnut, Arlington 74, Mass. ALR
Carruth, Howard E., '28
Medical Arts bldg., Portland 5, Ore. OALR
Carter, Albert A., '37.....419 Boylston, Boston 16, Mass. OP
Carter, E. D., '30.....Central bldg., Wichita 2, Kan. ALR
Carter, John M., '29.David Whitney bldg., Detroit 26, Mich. ALR
Carter, Leland F., '37
David Whitney bldg., Detroit 26, Mich. OP
①Carter, W. W., '08.....6 Chestnut av., Bronxville, N. Y. ALR
Caruthers, Samuel B., '37..Grenada Clinic, Grenada, Miss. OALR
①Cary, E. H., '12.....Medical Arts bldg., Dallas 1, Texas OALR
②Cary, G. C., '18.....Canon Block, Grand Junction, Colo. OALR
Case, Paul Henry, '39.....15 E. Monroe, Phoenix, Ariz. OP
Cassady, J. Vernal, '28
527 Sherland bldg., South Bend 9, Ind. OALR
Cassidy, Waldron A., '31
Medical Arts bldg., Omaha 2, Nebr. ALR
Casten, Virgil, '32.....412 Beacon st., Boston, Mass. OP
Castroviejo, Ramon, '31
635 W. 165th, New York 32, N. Y. OP-PI S
Cavanor, Frank Tracy, '46
1818 Medical Arts bldg., Minneapolis 2, Minn. ALR
Cave, Linus S., '29...State Tower bldg., Syracuse 2, N. Y. ALR
Cawthorne, Harold Jesse, '41
239 Pipestone, Benton Harbor, Mich. ALR
Cecha, Albin H., '35, Capt. (MC) USN
U. S. Naval Hospital, Memphis 15, Tenn. OP
Cerny, Frank Joseph, '45
160 Bischoff st., Fond du Lac, Wis. OP
Cetner, John A., '43.....365 State St., Albany, N. Y. OP
Chace, Robert R., '44,
635 W. 165th, New York 32, N. Y. OP
②Chadwick, Ira B., '20..717½ Walnut, Coffeyville, Kan. OALR
Chaffers, Wm. H., '31.....190 Bates, Lewiston, Maine ALR
Chainski, Edward L., '32.....130 N. Wells, Chicago 6, Ill. ALR
Chait, Robert A., '46.....20 Plaza st., Brooklyn, N.Y. ALR
Chamberlain, Webb P., '31
7405 Detroit av., Cleveland 2, Ohio OP
Chamberlain, Webb P. Jr., '44
7405 Detroit av., Cleveland, Ohio OP
Chamberlin, Clyde, '46
300 Rentschler Bldg., Hamilton, Ohio ALR
Chamberlin, Ira Iowa, '45
4 Chestnut St., Springfield, Mass. ALR
Chamlin, Max, '44,
1840 Grand Concourse, New York, N. Y. OP
Champion, Albert N., '36
Medical Arts bldg., San Antonio 5, Texas ALR
Chandler, Arthur C., '42
1562 Virginia st. E., Charleston 11, W. Va. OP
Chandler, G. E., '30..502 Huntington bldg., Miami 32, Fla. ALR
Chandler, Paul Austin, '38
5 Bay State Road, Boston 15, Mass. OP
Chapman, E. J., '43...331 Haywood bldg., Asheville, N. C. ALR
Chapman, Katharine H., '36
304 Burns bldg., Colorado Springs, Colo. OP
Chapman, S. Jefferson, '26
315 No. Cascade av., Colorado Springs, Colo. ALR
①Chapman, Vernon A., '15
435 N. Palm Canyon drive, Palm Springs, Calif. OALR
②Charles, Joseph W., '22..Humboldt bldg., St. Louis 3, Mo. OP
①Charlton, C. Coulter, '16
1616 Pacific av., Atlantic City, N. J. ALR
Chase, Carl Clarence, '39....121 Main, Middletown, Conn. OP
②Chase, E. Frank, '18.....Cobb bldg., Seattle 1, Wash. OALR
Chase, S. B., '25.....Carver bldg., Ft. Dodge, Iowa OALR
Chase, W. D., '24.....230 E. Broad, Bethlehem, Pa. ALR
Cheek, Carey, '38.....Medical Arts bldg., Springfield, Mo. OP
①Chenery, W. E., 377 Commonwealth av., Boston 15, Mass. ALR
③Cheney, James W., '20..Schweiter bldg., Wichita 2, Kan. OALR
Cheney, Ralph E., '36.....United bldg., Salina, Kans. OP
Chenoweth, Charlie Everette, '42
Forester's bldg., Mason City, Iowa ALR
Chessen, James, '46.....510 Republic bldg., Denver, Colo. ALR
Chester, Lewis, '45.....179 Allyn st., Hartford 3, Conn. ALR

Chevigny, Julius James, '45.....504 Broadway, Gary, Ind. ALR
Childers, Robert J., '29.....604 Park av., Plainfield, N. J. ALR
①Childs, H. A., '15....1016 W. Adams, Creston, Iowa OALR
Chinn, Chester W., '46..209 W. 125 st., New York, N.Y. OALR
Chipman, L. DeV., '25
44 Westwood ave., Wolfville, Nova Scotia, Canada OALR
Chisholm, Julian F., Jr., '36,
20 W. Cedar, c/o Mrs. Bunting, Boston 8, Mass. OP
Christensen, Eugene L., '44,
727 W. 7th, Los Angeles 14, Calif. OP
Christenberry, Henry Edward, Jr., '46
501 W. Church st, Knoxville, Tenn. ALR
Christoph, Carl H., '28.30 N. Michigan av., Chicago 2, Ill. OALR
Cinelli, Albert A., '36..1021 Park av., New York 28, N. Y. ALR
Cirillo, Anchise Anthony, '46
89-21 153rd St., Jamaica, N.Y. ALR
Clapp, C. A., '43.....513 N. Charles, Baltimore 1, Md. OP
Clark, Cecil P., '31.922 Hume-Mansur, Indianapolis 4, Ind. OP
Clark, Howard Elton, '39, 576 Hartnell st., Monterey, Calif. OALR
②Clark, I. G., '20.....188 E. State, Columbus 15, Ohio OALR
①Clark, J. Sheldon, '14...State Bank bldg., Freeport, Ill. OALR
Clark, James W., '37....2250 W. 91st St., Chicago 20, Ill. OP
①Clark, L. H., '15..337 Monroe av., Rochester 7, N. Y. OALR
Clark, Willard C., '43....333 Rockwood Av., Dayton, Ohio OP
Clark, William B., '34
American Bank bldg., New Orleans 12, La. OP
Clark, William H., '46
120 N. Lafayette Blvd., South Bend, Ind. ALR
Clarke, Clement C., '39
Amity Rd., Woodbridge, New Haven, Conn. OP
Clarke, Samuel Tracy, '41, Medical Dental Bldg., Reno, Nev. OP
Clayton, Milton Burns, '42
Southern R. R. Office bldg., Washington 13, D. C. ALR
Cleary, James A., '40
804 Boston bldg., Salt Lake City, Utah ALR
Cleff, Oscar, '26.....55 E. Washington, Chicago 2, Ill. OALR
②Clement, C. C., '18...30 N. Michigan av., Chicago 2, Ill. OALR
Clement, L. O., '30.....Guardian bldg., Salem, Ore. OALR
Clements, Frank Hartman, '44,
806 Medical Arts bldg., Birmingham, Ala. OP
Clerf, Louis H., '23.....1530 Locust, Philadelphia 2, Pa. ALR
Clifford, T. P., '27.David Whitney bldg., Detroit 26, Mich. OALR
Cline, Samuel, '32.....416 Marlborough, Boston 15, Mass. ALR
①Clothier, J. V., '06.....Kane bldg., Pocatello, Idaho OALR
Clothier, William L., '43
407-11 Kane bldg., Pocatello, Idaho OP
Coachman, Edwin H., '39
Manhattan bldg., Muskogee, Okla. ALR
Coakley, Leo P., '41...Medical Arts bldg., Omaha 2, Nebr. ALR
③Coates, George M., '21....1721 Pine, Philadelphia 3, Pa. ALR
①Cobb, Edwin, '12, 1680 N. Vine St., Hollywood 28, Calif. OALR
Cobey, James Francis, '46
1210 Chapel St., New Haven 11, Conn. OALR
Cockrum, William Marion, '45
908 Hulman bldg., Evansville, Ind. OP
Cody, Claude C., Jr., '28
1304 Walker av., Houston 2, Texas ALR
Cody, Claude Carr III, '46
1304 Walker av., Houston, Tex. ALR
Cogan, David G., '39.....243 Charles, Boston, Mass. OP
Cohen, Bernard M., '38
25 E. Washington st., Chicago 2, Ill. ALR
Cohen, Herman B., '27...255 S. 17th, Philadelphia 3, Pa. ALR
Cohen, Jerome H., '42
604 Johnstown Trust bldg., Johnstown, Pa. OP
Cohen, Louis B., '36....1320 Arrott, Philadelphia 24, Pa. OP
①Cohen, Martin, '10.....29 E. 64th, New York 21, N. Y. OP
②Cohen, Samuel, '21,
Spruce St. Medical bldg., Philadelphia 3, Pa. ALR-PLS
Cohoe, Don A., '30.13535 Woodward av., Detroit 3, Mich. ALR
Cole, John Gordon, '45.....780 Park av., New York, N.Y. OP
Cole, Nathan B., '35.....104 Market, Perth Amboy, N.J. OALR
Coleman, Benjamin, '41,
36-40 Bowne, Flushing, L. I., N. Y. ALR
Coleman, Joseph A., '40.5051 Iselin Av., New York 63, N.Y. ALR
Coleman, Lester Laudy, '41
162 E. 80th, New York 21, N. Y. ALR
Collins, Arthur D., '45
1007 Guardian Bldg., Cleveland 14, Ohio OP
Collins, Braswell E., '42, 701 Elizabeth St., Waycross, Ga. ALR
①Collins, E. W., '10.....1578 Humboldt, Denver 6, Colo. ALR
①Collins, J. D., '14.....187 Main, Northampton, Mass. ALR
Collins, James T., '40...Republic bldg., Cleveland 15, Ohio ALR
Colson, Z. William, '44....301 Essex St., Lawrence, Mass. OP
①Colver, B. N., '13.436 S. Boyle av., Los Angeles 33, Calif. ALR
Combacker, Leon C., '35
101½ Lincoln av., Fergus Falls, Minn. OP
Combs, Arnold B., '42.....Northwest Clinic, Minot, N. D. ALR
Combs, Fielding, '42.Nissen bldg., Winston Salem 3, N. C. ALR
Conboy, John E., '35
2265 University av., New York 53, N.Y. OP
Cone, Alfred J., '29.....453 N. Taylor, St. Louis 8, Mo. ALR
Coniey, John J., '43....425 E. 51st St., New York, N. Y. ALR
Connell, Evan Shelby, '40
Commerce bldg., Kansas City 6, Mo. OP
Connelly, Edgar J., '42.....Roshek bldg., Dubuque, Iowa ALR
Connole, J. V., '32
Miners Nat'l Bank bldg., Wilkes-Barre, Pa. OP
Connor, Arthur B., '42.....P.O. Box 398, Dania, Fla. ALR
②Connor, C. E., '20..Lowry Med. bldg., St. Paul 2, Minn. ALR
Connor, Joseph Joyce, '42....750 Main, Hartford 3, Conn. OP
Constans, George M., '29....Clinic bldg., Bismarck, N. D. OP
Constantine, Elizabeth Frederick, '43
340 E. 63rd, New York, 21, N. Y. OP
Constantine, K. W., '36..229 Barton av., Palm Beach, Fla. OP
Constantinople, P. S., '29
1801 Eye St. N.W., Washington 6, D. C. ALR
Canty, Anthony J., '37
318 Forty-eighth st., Union City, N. J. ALR
Conway, J. M., '30....Jenkins Arcade, Pittsburgh 22, Pa. ALR
Conway, Wm. S., '35..314½ Howard St., Petoskey, Mich. OALR
Cook, Raymond C., '36,
1005 Donaghey Bldg., Little Rock, Ark. OP
Cook, W. Albert, '24....Medical Arts bldg., Tulsa 3, Okla. OALR
Cooley, Carl C., '37, Medical Arts bldg., Norfolk 10, Va. OALR
Coombs, A. J., '32....30 N. Michigan av., Chicago 2, Ill. ALR
Coon, Eugene H., '35...131 Fulton av., Hempstead, N. Y. ALR
Cooper, Edmond L., '39
414 David Whitney bldg., Detroit 26, Mich. OP
Cooper, F. Maxey, '41
Medical Arts bldg., Oklahoma City 2, Okla. OP
Cooper, James H., '43, 316 Michigan st., Toledo 2, Ohio OP
Cooper, Kemp G., '39..Metropolitan bldg., Denver 2, Colo. ALR
Cooper, Roy C., '32..6929 Yorkshire rd., Pittsburgh 8, Pa. ALR
Cooperman, Harold O., '37
416 No. Bedford, Beverly Hills, Calif. OP
Copps, Lyman A., '23..Marshfield Clinic, Marshfield, Wis. OALR
Corboy, Phillip M., '36, 565 Young Bldg., Honolulu 9, T. H. OP
Cordes, Frederick C., '33..384 Post, San Francisco 8, Calif. OP
Cordray, David P., '41
Carle Hospital Clinic, 602 W. Univ. av., Urbana, Ill. ALR

Cornelius, C. E., '40....119 Summit av., Jenkintown, Pa. ALR
ⓉCorser, John B., '11...415 Mulberry st., Scranton 3, Pa. OP
Cortopassi, Andre J., '30,
324 S. Washington av., Saginaw, Mich. ALR
Cortopassi, Vital E., '41,
324 S. Washington ave., Saginaw, Mich. ALR
Corya, H. W., '30.....30 E. 40th, New York 16, N. Y. ALR
Cosgrove, Kingsley White, '40
115 E. Capitol, Little Rock, Ark. OALR
Costen, J. B., '25.Beaumont Medical bldg., St. Louis 8, Mo. ALR
Costenbader, Frank Duncan, '40
1150 Connecticut av., Washington 6, D. C. OP
Coston, Tullos O., '40,
Medical Arts bldg., Oklahoma City 2, Okla. OP
ⓉCott, C. C., '15....333 Linwood av., Buffalo 9, N. Y. ALR
Cotten, Willis Frank, '43.....202 State, McComb, Miss. ALR
Cottle, Maurice H., '36.30 N. Michigan av., Chicago 2, Ill. ALR
Courtney, Robert H., '35
Professional bldg., Richmond 19, Va. OP
Covell, Kermit Wendell, '46..610 Main St., Racine, Wisc. ALR
Cowan, Alfred, '28.....1930 Chestnut, Philadelphia 3, Pa. OP
Cowan, Clyde R., '33
69 Bay State road, Back Bay, Boston 15, Mass. ALR
Cowan, Thomas H., '41
1930 Chestnut St., Philadelphia 3, Pa. OP
Cowell, Edward Huntington, '46
427 Biltmore Way, Coral Gables, Florida OP
Cowen, Jack P., '37
1421-122 So. Michigan av., Chicago, Ill. OP
ⓉCowper, H. W., '14.....543 Franklin, Buffalo 2, N. Y. OP
Cox, Charles L., '36
Pere Marquette bldg., New Orleans 12, La. ALR
Cox, John R., '44, Capt. (MC)
202 S. 34th St., Camden, N.J. OP
Cox, Kelly, '39.....Medical Arts bldg., Dallas 1, Texas OP
Cox, Ronald A., '37
1779 Mass. av., N.W., Washington 6, D. C. OP
Cracovaner, Arthur J., '35
103 E. 78th st., New York 21, N. Y. ALR
Craddock, Wm. H., '38..703 Carew Tower, Cincinnati 2, Ohio ALR
ⓉCraft, Kenneth L., '20
1002 Hume-Mansur bldg., Indianapolis 4, Ind. ALR
Crage, Francis M., '35..25 E. Washington, Chicago 2, Ill. OP
Craig, Paul C., '33.....232 N. 5th, Reading, Pa. OP
Craig, Stuart Lessley, '45
135 E. 65th St., New York 21, N.Y. ALR
Cramton, Edward A., '37....33 Main, St. Johnsbury, Vt. OALR
Crandall, Alan Slade, '45
141 East 2nd South, Salt Lake City 1, Utah OP
Crane, Cyril Vincent, '38
6810 S. Loomis blvd., Chicago 36, Ill. OP
Crane, Donald VanDenburgh, '46
Medical Arts Bldg., Tulsa 3, Okla. OP
Crane, W. R., '29
Room 1027, 727 W. 7th, Los Angeles 14, Calif. ALR
Cranston, William J., Jr., '43
175 Clinton Ave., Kingston, N.Y. OP
Crawford, H. E., '43..139 Kinoole St., Hilo, Hawaii, T. H. OP
Crawford, James H., '44
#7 Way-Penny bldg., Watertown, S. Dak. OP
Creasy, Raymond C., '37..67 E. 59th, New York 22, N. Y. ALR
Crebbin, Alexander R., '38
Maison Blanche bldg., New Orleans 16, La. OP
Cregar, John S., '39....440 S. Harrison, East Orange, N.J. OP
ⓉCreighton, Wm. J., '10..37 S. 20th, Philadelphia 3, Pa. OP
Crespo, Jose E., '41.....28 W. 95th, New York 25, N. Y. ALR
Cress, William J., '34.....318 W. Market, Pottsville, Pa. ALR
Creveling, Earle LaMont, '42...P. O. Box 2446, Reno, Nev. ALR
ⓉCrisp, William H., '13, Metropolitan bldg., Denver 2, Colo. OP
Criswell, Robert H., '37....Phoenix bldg., Bay City, Mich. OP
Croissant, Charles A., '31..390 Main, Worcester 8, Mass. ALR
Croll, Leo J., '39.....5057 Woodward ave., Detroit, Mich. OALR
Croll, Maurice, '45....5057 Woodward Ave., Detroit, Mich. OALR
Croman, Joseph M., Jr., '24
115 Cass av., Mt. Clemens, Mich. OALR
ⓉCross, A. E., '14.....390 Main, Worcester 8, Mass. OP
ⓉCross, G. H., '19.....525 Welsh, Chester, Pa. OP
Crossen, Henry F., '39
David Whitney bldg., Detroit 26, Mich. ALR
ⓉCrossley, E. R., '11, 4144 Sheridan Rd., Chicago 13, Ill. OALR
Croushore, James E., '35
573 Fisher bldg., Detroit 2, Mich. ALR
Crowe, Samuel James, '45
Johns Hopkins Hospital, Baltimore, Md. ALR
Crume, John J., '31.....724 Polk, Amarillo, Texas ALR
Crumrine, Norman R., '28.....595 Third st., Beaver, Pa. OALR
Cruthirds, Archie Edward, '40
Professional bldg., Phoenix, Ariz. ALR
ⓉCulbertson, L. R., '98..Masonic Temple, Zanesville, Ohio OALR
Culler, Arthur M., '32
150 E. Broad st., Columbus 15, Ohio OP
ⓉCullom, M. M., '10
Bennie-Dillon bldg., Nashville 3, Tenn. OALR
Culpepper, Wm. S., '32, Col. (MC)
6601 E. 18th Ave., Denver, Colo. ALR
Cummings, Edward J., '37
1835 Eye St. N.W., Washington 6, D. C. ALR
Cummings, George O., '30...47 Deering, Portland 4, Maine ALR
Cunning, Daniel S., '28..121 E. 60th, New York 22, N. Y. OALR
Cunningham, Bernard P., '43
106 Center St., Bridgeport 4, Conn. ALR
Cunningham, Henry Kelso, '42
Medical Arts bldg., Knoxville 1, Tenn. ALR
Cunningham, John C., '43, 49 S. Winooski av., Burlington, Vt. OP
ⓉCurdy, R. J., '18....1102 Grand av., Kansas City 6, Mo. OP
Curran, Desmond, '37
1805 Federal Reserve bldg., Kansas City, Mo. OP
ⓉCurran, E. J., '19
Federal Res. Bank bldg., Kansas City 6, Mo. OP
Curran, Kevin, '39
Federal Res. Bank bldg., Kansas City 6, Mo. OP
Currier, Wilber D., '44
65 N. Madison Ave., Pasadena, Calif. ALR
ⓉCurry, Glendon E., '12
1204 Pitt Bank bldg., Pittsburgh 22, Pa. OP
Curtin, Eugene A., '23.....506 Spruce, Scranton 3, Pa. OALR
Curts, James H., '46
330 South Washington St., Saginaw, Mich. OP
Cushman, Beulah, '21....25 E. Washington, Chicago 2, Ill. OP
Cusick, Paul L., '43, 1108 Stroh Bldg., Detroit 26, Mich. OP
Cuthbert, Marvin Peare, '45
5611 No. Delaware st., Indianapolis, Ind. OP
Cutino, R.M., '30.....79 Pierrepont, Brooklyn 2, N. Y. OP
Cutler, Abraham Maurice, '41
750 Ocean av., Brooklyn 26, N. Y. ALR
ⓉCutler, Franklin E., '12.....Wecoma, Ore. ALR
Cutler, Harold M., '42
640 S. Kingshighway, St. Louis 10, Mo. ALR
Cutler, Max Helix, '45
1010, 307 N. Michigan, Chicago, Ill. ALR

Cutler, Morton, '46
122½ Main, South, Twin Falls, Idaho OP
Cutler, Norman L., '38
1300 Harrison st., Wilmington, Del. OP

## D

Dahl, Elmer O., '33
Medical Arts bldg., Minneapolis 2, Minn. ALR
Dailey, Gilbert L., '27.....618 N. 3rd, Harrisburg, Pa. OALR
Daily, Louis, '26....Medical Arts bldg., Houston 2, Texas OALR
Daily, Ray K., '26...Medical Arts bldg., Houston 2, Texas OALR
Daisey, Willard O., '37
30 Park av., Rehobath Beach, Del. ALR
Daley, Jacob, '41.....104 E. 40th, New York 16, N. Y. ALR
Dalton, S. Eugene, '38, 101 S. Surrey Ave.,
Ventnor, Atlantic City, N. J. ALR
Daly, John F., '44.....140 E. 54th st., New York, N.Y. ALR
Dame, L. R., '42.....78 Federal st., Greenfield, Mass. OP
Damitz, John Cyril, '43
2nd National Bank Bldg., Akron, Ohio OALR
Dan, Julius Martin, '46
1362 President st., Brooklyn 13, N.Y. OP
Daniel, Ruby K., '43...Medical Arts bldg., Dallas 1, Texas OP
Danielson, Ralph W., '34
Metropolitan bldg., Denver 2, Colo. OP
②Darmer, George A., '21....Terminal bldg., Aurora, Ill. OALR
Darrough, L. E., '32.....4105 Live Oak, Dallas, Texas ALR
Dart, M. O., '40....2701 S. Marion st., Englewood, Colo. ALR
Dattner, Herman B., '44
1000 Miners Nat'l Bank bldg., Wilkes-Barre, Pa. ALR
Davidson, H. P., '24...30 N. Michigan av., Chicago 2, Ill. OP
Davidson, Morris, '29....17 Park av., New York 16, N. Y. OP
Davies, Charles Edgar, '46
925 West Georgia St., Vancouver, B.C., Canada OP
Davies, H. F., '30.1835 Eye st. N.W., Washington 6, D. C. ALR
Davies, Morgan C., '32...269 E. State, Columbus 15, Ohio OP
①Davies, T. Alexander, '13.578 Sherbourne, Toronto, Can. OALR
Davies, Windsor S., '36
1302 Stroh Bldg., 28 W. Adams Ave, Detroit, Mich. OP
Davis, Arthur Henry, '41.Medical Arts bldg., Tulsa 3, Okla. ALR
Davis, David, '34.1835 Eye st. N.W., Washington 6, D. C. ALR
Davis, Frederick Allison, '27
224 W. Washington av., Madison 3, Wis. OP
①Davis, George E., '15..14 E. 90th, New York 28, N. Y. OALR
Davis, Hugh P., '37, 135 E. 65th St., New York 21, N. Y. ALR
Davis, James Edward, '40.179 Allyn st., Hartford 3, Conn. ALR
Davis, John Calvin, Jr., '28..Aquila Court, Omaha 2, Nebr. OALR
Davis, Leo L., '42, 1509 Farrell Lane, Richland, Wash. OP
Davis, Oscar, '36, Capt. (M.C.) USN
U. S. Naval Hospital, Pensacola, Fla. OALR
Davis, Ralph Allison, '45.....4753 Broadway, Chicago, Ill. OP
②Davis, Ralph F., '20.Medical Arts bldg., Portland 5, Ore. OALR
Davis, T. Carroll, '31..3128 N. Broad, Philadelphia 32, Pa. ALR
Davis, Thomas W., '23
O'Hanlon bldg., Winston-Salem 3, N. C. OALR
Davis, Warren B., '34
135 S. 18th, Philadelphia 3, Pa. ALR-PI S
Davis, Wm. C., '30.....150 E. Broad, Columbus 15, Ohio OP
Davison, Francis W., '36....Geisinger Hosp., Danville, Pa. ALR
Day, Kenneth M., '30.121 University pl., Pittsburgh 13, Pa. ALR
③Dayton, G. O., '18.1401 S. Hope, Los Angeles 15, Calif. OALR
Deal, John F., '28.....522 E. Capitol av., Springfield, Ill. OALR
Dean, Alfred, '23
Medical Arts bldg., Grand Rapids 2, Mich. OP
①Dean, F. W., '03.....22 S. 6th, Council Bluffs, Iowa OALR
Dean, L. W., Jr., '38
3720 Washington blvd., St. Louis 8, Mo. ALR
①Dean, Walter, '17..300 Francis bldg., Louisville 2, Ky. OALR
Dearmin, Robert M., '31,
3440 No. Meridian St., Indianapolis, Ind. ALR
De Blois, Elizabeth, '39
247 Commonwealth av., Boston 15, Mass. ALR
②DeBoe, M. P., '20....Huntington bldg., Miami 32, Fla. OALR
DeBord, Bert, Jr., '44....Scott-White Clinic, Temple, Tex. ALR
②Decker, J. C., '18.....Frances bldg., Sioux City 8, Iowa OALR
Decker, Phillip Harold, '42
25 W. Third st., Williamsport 5, Pa. ALR
Decker, Russell M., '38.65 N. Madison, Pasadena 1, Calif. ALR
Deems, Myers B., '43, Lt. Col. (M.C.),
1601 Leyden st., Denver, Colo. ALR
Deeton, William L., '38
13125 Shaker Square, Cleveland 20, Ohio ALR
③Defnet, William A., '20
28 Adams av. W., Detroit 26, Mich. ALR
②Deichler, L. Waller, '20
1930 Chestnut, Philadelphia 3, Pa. OALR
Delaney, Adrian J., '41, Capt. (MC), USN,
Nat'l. Naval Medical Center, Bethesda, Md. ALR
Delaney, James H., '31.....138 W. 9th, Erie, Pa. OP
Della Rocca, Anthony, J., '46
1400 Benson St., New York 61, N.Y. ALR
Delman, A. H., '39
U.S. Veterans Hosp., W. Roxbury, Boston 32, Mass. ALR
DeLozier, Leonard Cecil, '41
311 Central National Bank bldg., Peoria, Ill. ALR
Delph, John F., '28..700 N. Michigan av., Chicago 11, Ill. ALR
Denison, Ward C., '31.....316 Broadway, Paterson, N. J. ALR
Denman, Francis E., '38
541 Lincoln road, Miami Beach, Fla. ALR
①Dennis, F. L., '06..2745 Barnson pl., San Diego 3, Calif. ALR
Denyes, Gerald Foster, '40...316 Michigan, Toledo 2, Ohio ALR
Depping, C. W., '30.....17 W. 54th, New York 19, N. Y. ALR
deRoetth, Andrew F., '44
508 Old Nat'l Bank Bldg., Spokane, Wash. OP
Dessoff, Joseph, '38
1726 Eye st. N.W., Washington 6, D. C. OP
De Stio, Daniel S., '39..Highland bldg., Pittsburgh 6, Pa. ALR
Deutsch, William, Jr., '42
309 Johnson bldg., Muncie, Ind. ALR
DeVoe, Arthur Gerard, '41, Institute of Ophthalmology
635 W. 165th St., New York 32, N.Y. OP
De Vries, Harold G., '38.....36 E. 8th, Holland, Mich. ALR
Dewar, Murray M., '32
Medical Arts bldg., Grand Rapids 2, Mich. OP
②DeWeese, Clarence, '20
201 W. Second st., Lexington 15, Ky. OALR
①Dewey, C. H., '12..760 S. Oak Knoll av., Pasadena, Calif. OALR
DeWitt, Edward N., '28.881 Lafayette, Bridgeport 4, Conn. OP
DeWolf, Charles W., '38.....8 Porter st., Melrose, Mass. ALR
Dexter, Milton A., '30
1618 Westwood blvd., Los Angeles 24, Calif. ALR
Dezer, Charles N., Jr., '41....210 Main, Hackensack, N. J. ALR
Dickerson, Wm. E., '35, 802 Masonic Temple, Danville, Va. ALR
Dickey Clifford A., '44
450 Sutter St., San Francisco 8, Calif. OP
Dickinson, Wm. G., '36....State Tower, Syracuse 2, N. Y. OP
Dickson, J. Charles, '31
Medical Arts bldg., Houston 2, Texas ALR

Dickson, Robert L., '38...4803 Penn, Philadelphia 24, Pa. ALR
Diehl, James E., '44, 813 Wainwright Bldg., Norfolk, Va. ALR
Diehl, William Harold, '46..701 Chestnut St., Lebanon, Pa. ALR
Diess, Robert G., '42.....307 B.C.N.B. bldg, Butler, Pa. OP
Dietl, Ernest L., '40..527 Sherland bldg., South Bend, Ind. ALR
Dill, J. Lewis, '38..Henry Ford Hospital, Detroit 2, Mich. ALR
Dillemuth, Frederick Joseph, '41
404 E. 154th, New York 55, N. Y. ALR
Dillon, Elmo E., '38.....1952 Ridge rd., Homewood, Ill. OALR
Dimmitt, Frank W., '34.78 Waterman, Providence 6, R. I. OALR
Dimond, Waldo B., '33..701-710 Gay bldg. Madison, Wis. ALR
Dinan, Thomas Edward, '41
132 Union av., Framingham, Mass. ALR
Dineen, John B., '44...1537 Main st., Springfield 3, Mass. ALR
Ⓒ Dintenfass, Henry, '21..1305 Spruce, Philadelphia 7, Pa. ALR
Diven, Wilbur Lester, '41...408½ Main, Bismarck, N. D. ALR
Dix, Harold C., '42..206 Paddington Rd, Baltimore 12, Md. OP
Dixon, Fred W., '29.....Rose bldg., Cleveland 15, Ohio ALR
Dixon, O. Jason, '28.Professional bldg., Kansas City 6, Mo. ALR
Ⓣ Dixon, W. E., '18.....1617 E. 4th, Santa Ana, Calif. OALR
Dlugasch, Louis, '29....110 E. 36th, New York 16, N. Y. ALR
Dobbs, O. R., '30..9927 Riverview Drive, St. Louis 15, Mo. ALR
Ⓣ Dodd, Oscar, '03.....636 Church, Evanston, Ill. OP
Dodge, W. M. Jr., '34
1207 Central National Bank, Battle Creek, Mich. OP
Doherty, Wm. B., '27....150 W. 55th, New York 19, N. Y. OP
Dolowitz, David A., '43
1152 Gilmer Drive, Salt Lake City 5, Utah ALR
Dolph, Chancey H., '44.....317 Main, Baytown, Texas ALR
Donahue, Hugh C., '39
520 Commonwealth av., Boston 15, Mass. OP
Donlon, John J., '43, 141 Hempstead Ave.,
Rockville Center, N. Y. ALR
Donnell, Herbert, '38....507 W. Main, Waxahachie, Texas ALR
Donnelly, Edward J., '38..1321 Spruce, Philadelphia 7, Pa. OP
Donoghue, William Francis, Jr., '46
69 Chestnut St., Springfield, Mass. ALR
Ⓣ Donoher, William D., '17
635 S. Westlake av., Los Angeles 5, Calif. OALR
Donohoe, Edward C., '31..102 N. Brand blvd., Glendale, Cal. ALR
Donovan, John E., '38.....265 W. 10th, Erie, Pa. ALR
Donovan, Richard J., '43...422 N. Main, Fall River, Mass. OALR
Doran, Frank, '40.....Metz bldg., Grand Rapids 2, Mich. ALR
Dorge, Richard I., '23
1111 Nicollet av., Minneapolis 2, Minn. OALR
Dougherty, Clifford L., '31.55 E. Washington, Chicago 2, Ill. ALR
Douglas, Edward M., '36
87-85—153rd, Jamaica 2, L. I., N. Y. OP
Ⓒ Douglas, Frederick A., '22
State Bank bldg., La Crosse, Wis. OALR
Doust, Alfred F., '44....965 Lancaster av., Syracuse, N.Y. ALR
Ⓓ Dow, Frank E., '14.....4 West st., Northampton, Mass. OP
Dow, Julian Neal, '31,
9730 Wilshire blvd., Beverly Hills, Calif. OP
Dowling, Harvey E., '32
315 Southam bldg., Calgary, Alberta, Canada OP
Ⓓ Dowling, J. Ivimey, '14
116 Washington av., Albany 6, N. Y. OALR
Dowling, James Reginald, '40
508 Ohio Merchants bldg., Massillon, Ohio ALR
Downey, Harold R., '38
1740 M st., N.W., Washington 6, D. C. OP
Downing, Arthur H., '43.....406-6th av., Des Moines, Ia. OP
Downing, James A., '36
Bankers Trust bldg., Des Moines 9, Iowa ALR
Dreil, Maurice Joseph, '46
630 Medical Arts bldg., Seattle 1, Wash. OP
Drews, Leslie C., '43
516 Metropolitan bldg., St. Louis 3, Mo. OP
Driscoll, Frederick J., '33,
P.O. Box 1653, Ft. Lauderdale, Fla., ALR
Droegemueller, Wm. H., '39, 1845 11th Ave., Greeley, Colo. OP
Drooker, Joshua C., '40, 20 Charlesgate W., Boston, Mass. ALR
Druss, Joseph G., '36..1040 Park av., New York 28, N. Y. ALR
Dubins, Henry B., '43.....248 State, Albany 6, N. Y. OP
Dublin, George Joseph, '41
1930 Chestnut, Philadelphia 3, Pa. OP
Duboe, Sidney H., '42..4160 W. Madison, Chicago 24, Ill. ALR
Duclos, Gaston Normand, '45
#3 DuMoulin St., Drummondville, Quebec, Canada ALR
Duehr, Peter A., '39
224 W. Washington av., Madison 3, Wis. OP
Duke, Roncie R., '31.....706 Franklin, Tampa 2, Fla. ALR
Ⓒ Dulaney, Octavus, '17....Brown bldg., Louisville 2, Ky. ALR
Dumas, Edward DeVan, '36
Medical Arts bldg., San Antonio 5, Texas ALR
Dunaway, Carl E. '35, 711 DuPont Bldg. Miami, Fla. ALR
Dunbar, John Charles, '40.Empire bldg., Pittsburgh 22, Pa. OP
Duncan, Frank Benbow, '16....301 Polk St., Amarillo, Tex. OP
Duncan, Perry E., '37..405 E. Capitol Ave., Springfield, Ill. OP
Duncan, R. A., '29.....Amarillo bldg., Amarillo, Texas ALR
Ⓓ Dundas-Grant, Sir James (Retired)....Address Unknown
Dunlap, Henry A., '37
7815 E. Jefferson av., Detroit 14, Mich. OP
Ⓒ Dunlap, Lawrence G., '20..101 Main St., Anaconda, Mont. OALR
Dunn, Austin G., '37
111½ Strongs av., Stevens Point, Wis. ALR
Dunnavan, Floyd Leighton, '42
Arts bldg., Vancouver, Wash. OP
Dunnington, John H., '24, Inst. of Ophthalmology,
Pres. Hos., 635 W. 165th, New York 32, N.Y. OP-PLS
Dunphy, Edwin B., '31.....243 Charles St., Boston, Mass. OP
Ⓣ Duntley, G. S., '13.....604 Beverly av., Macomb, Ill. OALR
Durr, S. A., '27....511 San Fernando, San Diego 6, Calif. OP
Ⓣ Dutrow, H. V., '14.....426 Kramer Road, Dayton, Ohio OALR
Dvorak, Joseph E., '37.Davidson bldg., Sioux City 13, Iowa OP
Dwyer, James G., '36...375 Park av., New York 22, N. Y. ALR
Dyar, Edwin William '40,
5407 Kenwood av., Indianapolis 8, Ind. OP
Ⓔ Dyer, C. P., '20...508 N. Grand blvd., St. Louis 3, Mo. OP
Dykman, Augustus B., '37
833 S.W. 11th av., Portland 5, Ore. OP
Dysart, Ben Robnett, '41
65 N. Madison av., Pasadena 1, Calif. ALR

## E

Eagle, Watt W., '37.....Duke Hospital, Durham, N. C. ALR
Earl, Harry D., '36
1501 LeGrande Terrace, San Pedro, Calif. ALR
Earnest, Clarence E., '31.....Thatcher bldg., Pueblo, Colo. ALR
Ⓔ Easton, J. C., '04
First Nat'l Bank bldg., Springfield, Ohio OALR
Easton, Mahlon T., '39.....264 Beacon, Boston 16, Mass. OP
Ebeling, Wm. B., '30.....54 Greene av., Brooklyn 5, N. Y. OP
Eber, Carl Theo., '28, Carleton bldg., St. Louis 1, Mo. OP
Ebert, Reinhold O., '37.....64 Mt. Vernon, Oshkosh, Wis. OP
Edelman, Maurice I., '42
605 Lincoln rd., Miami Beach 39, Fla. ALR
Edgerton, Ambrose E., '31
450 Sutter, San Francisco 8, Calif. OP

Edmunds, Stanley L., '38..360 Glen St., Glen Falls, N.Y. ALR
①Edwards, C. J., '13
First Nat'l Bank bldg., Vicksburg, Miss. OALR
Edwards, David Lloyd, '46
203 Stanolind Bldg., Tulsa 3, Okla. OP
Edwards, Gwilym Austin, '42
Home Guards bldg., Van Wert, Ohio ALR
Edwards, S. A., '35...Home Guards bldg., Van Wert, Ohio ALR
②Edwards, Slocomb R., '18.107 S. Fifth, El Centro, Calif. OALR
Edwards, Thomas J., '38
1237 Lowry bldg., St. Paul 2, Minn. OP
Edwards, Thomas Marvin, '46
322 Citizens Building, Tampa 2, Florida ALR
Ehrenfeld, Edward, '44...185 Lexington av., Passaic, N. J. OP
Ehrlich, Leon Herbert, '45
211 Central Park W., New York 24, N.Y. OP
Ehrlich, Max, '40, 721 N. Broad st., Elizabeth 3, N. J. ALR
Ehrmann, Evelina W., '42
327 S. La Grange Road, La Grange, Ill. OP
Eilert, John Harold, '45
391 E. 149th st., New York 55, N.Y. OP
Eimer, C. E., '28.....462 N. Taylor av., St. Louis 8, Mo. ALR
Elfman, Louis Kenneth, '42
6330 Lebanon av., Philadelphia 31, Pa. ALR
Ellenberger, Carl, '40.14805 Detroit av., Lakewood 7, Ohio OP
②Elles, Norma B., '19
Niels Esperson bldg., Houston 2, Texas OALR
②Ellett, E. C., '98.....Exchange bldg., Memphis 3, Tenn. OP
Elliff, Edgar Alonzo, '41.....216 N. 3rd, Sterling, Colo. ALR
Elliot, Alfred J., '43
170 St. George st., Toronto, Ont., Canada OP
Elliott, J. Norman, '38..Griesheim bldg., Bloomington, Ill. ALR
Elliott, Raymond E., '41,
2150 St. Paul st., Rochester 5, N.Y. ALR
Ellis, Bert E., '31
303 Hume-Mansur bldg., Indianapolis 4, Ind. ALR
Ellis, C. Ward, '28.....M.O. Box 637, Lansing, Mich. ALR
Ellis, Orwyn H., '40..727 W. 7th st., Los Angeles 14, Calif. OP
Ellis, Van M., '44....1528 Spruce St., Philadelphia 2, Pa. OP
Elwyn, Herman, '45, 239 Central Park W., New York 24, N.Y. OP
Elz, Jule T., '37....3606 Gravois blvd., St. Louis 16, Mo. OP
Emenhiser, Lee K., '44
Medical Arts bldg., Oklahoma City, Okla. ALR
①Emerson, Linn, '06.....303 Park av., Orange, N. J. OALR
Emery, John R., '38...Medical Centre, White Plains, N. Y. ALR
Emrich, Paul Stanley, '46..64 Mt. Vernon, Oshkosh, Wisc. ALR
Emswiler, Herbert D., '37, 370 E. Town, Columbus 15, Ohio ALR
Engler, Clarence Wm., '33
2323 Prospect av., Cleveland 15, Ohio ALR
Enlows, Ella M. A., '34
150 N. Victoria Park rd., Ft. Lauderdale, Fla. ALR
Epstein, Sidney S., '41
2021 Grand Concourse, New York 53, N. Y. OP
Equen, Murdock S., '31
144 Ponce de Leon av., N.E., Atlanta, Ga. ALR
Erich, John B., '44.....Mayo Clinic, Rochester, Minn. PLS
Ernsting, Harry Carl, '42..8 W. Main st., Springfield, Ohio OP
Ersay, Emil Francis, '46
1030 Euclid Av., Cleveland 14, Ohio ALR
②Ersner, Matthew S., '20, 1915 Spruce, Philadelphia 3, Pa. ALR
Eschenbrenner, John W., Jr., '37
306 W. Broadway, Fort Worth 4, Texas OALR
Esterly, Daniel Beard, '46
104 Madison, Pasadena 4, Calif. OP
Esterman, Benjamin, '37.983 Park av., New York 28, N. Y. OP
Estlick, Richard E., '45
629 Wayne Pharmacal bldg., Ft. Wayne 2, Ind. ALR
Eubank, Ambrose E., '28.Bryant bldg., Kansas City 6, Mo. OALR
Eubank-Jones, Miriam D., '39, 944 Maple av., Evanston, Ill. OP
Evans, John Dorritte, '41
899 Madison Ave., Memphis 3, Tenn. ALR
Evans, J. N., '25.....23 Schermerhorn, Brooklyn 2, N. Y. OP
Evans, Maurice G., '32, 416 Marlborough, Boston 15, Mass. ALR
Evans, Samuel D., '44
1502 Park bldg., 355 Fifth av., Pittsburgh 22, Pa. OP
Evans, S. S., '32
Physicians & Surgeons bldg., Memphis 3, Tenn. ALR
Evans, Wm. H., '34, 510 Dollar Bank bldg., Youngstown, Ohio ALR
Evatt, Clay W., '40....91 Rutledge av., Charleston 16, S.C. ALR
Everett, A. Reginald, '39, 11 Wall st., New York 5, N. Y. ALR
Evers, Lorance Bixler, '41.....1036 Wall St., Bend, Ore. ALR
①Ewing, Fayette C., '99, (Retired)
"Nosegay," Pineville, La

## F

Fabricant, Noah D., '36.185 N. Wabash av., Chicago 1, Ill. ALR
Fackler, C. L., '30.....Country Club rd., York, Pa. OALR
Faggart, George H., '43.18 W. Oglethorpe av., Savannah, Ga. ALR
Faier, H. I., '43
1930 Wilshire blvd., Suite 203, Los Angeles, Cal. OP
Faier, Samuel Z., '35..1125 No. 11th st., Ft. Smith, Ark. OALR
①Fairbairn, John F., '12.925 Delaware av., Buffalo 9, N. Y. ALR
②Fairchild, Nora M., '22
939 S. Figueroa st., Los Angeles 15, Calif. OP
Fairfax, Kenneth T., '39.....423 Main st., Geneva, N. Y. OP
Fairing, John W., '24.....122 N. Main, Greensburg, Pa. OALR
Falkson, Kurt F., '42.225 W. 86th st., New York 24, N. Y. ALR
①Fallows, Howard D., '16
Foresters bldg., Mason City, Iowa OALR
Falls, Harold Francis, '43...Uni. Hosp., Ann Arbor, Mich. OP
Farmer, William Dempsey, '45
119 North Elm St., Greensboro, N.C. ALR
①Farnsworth, M. A., '17
Opera House Apt. bldg., La Junta, Colo. OALR
Farrell, James J., '35..... 250 Genesee, Utica 2, N. Y. OP
①Farrington, P. M., '04....Colburn Hotel, Denver, Colo. ALR
Farrior, J. Brown, '42....Ochsner Clinic, New Orleans, La. ALR
Fast, Ralph B., '36..136 E. Michigan, Kalamazoo 4, Mich. ALR
①Fawcett, Ivan, '13....75 Twelfth st., Wheeling, W. Va. OALR
Fawcett, Keith R., '46.....205 W. 2nd St., Duluth, Minn. OP
Fein, Harry, '40.....483 Beacon, Boston 15, Mass. ALR
Fein, Norman Nathaniel, '46
Trinity Hospital, Little Rock, Ark. ALR
Feldman, Irvin, '39
1722 Eye St., N.W., Washington, D.C. ALR
Feldman, Joseph, '40
668 Eastern Parkway, Brooklyn 13, N. Y. ALR
Fellows, M. Fording, '35
1209 Medical Arts bldg., Duluth, Minn. OP
①Fenton, R. A., '21, 1020 S. W. Taylor, Portland 5, Ore. OALR
Ferguson, E. R., '35.....404 Inman st., Cleveland, Tenn. ALR
Ferguson, George Burton, '40
1110 West Main st., Durham, N. C. ALR
Fernandez, Luis J., '40, Box 2206, San Juan 10, Puerto Rico OP
Fernandez, Ricardo F., '39, 15 Ponce de Leon av.,
Box 2206, San Juan 10, Puerto Rico OP
Fernlund, G. B., '28
8626—115th, Richmond Hill, New York 18, N. Y. ALR

**Ferris, Ralph G., '34**.55 W. Maple av., Birmingham, Mich. **ALR**
**Fewell, A. G., '31**.....1924 Pine, Philadelphia 3, Pa. **OP**
**Field, Homer Baker, '42**
12757 S. Western av., Blue Island, Ill. **OP**
Fields, Jack, '46.....30 North Michigan av., Chicago 1, Ill. **OP**
Figi, Frederick A., '27.....Mayo Clinic, Rochester, Minn. **LR-PIS**
**Filmer, George A., '41**
324 Metropolitan bldg., Denver 2, Colo. **OP**
**Findlay, E. K., '30**.....30 N. Michigan av., Chicago 2, Ill. **OP**
**Fine, Abraham, '32**....925 Ocean av., Brooklyn 26, N. Y. **OALR**
**Fineberg, Maxwell, '30**
607 N. Grand blvd., St. Louis 3, Mo. **ALR**
**Fineberg, Nathan L., '38**
475 Commonwealth av., Boston 15, Mass. **ALR**
**Finegan, James F., '41**
415 Medical Arts Bldg., Omaha 2, Nebr. **OP**
**Fink, Leo W., '39**.Medical Arts bldg., Minneapolis 2, Minn. **ALR**
**Fink, Otto E., '38**.....9 W. Madison, Danville, Ill. **OP**
**Fink, Walter H., '27**
Medical Arts bldg., Minneapolis 2, Minn. **OP**
**Finney, Nancy E., '37**..2453 Gilbert av., Cincinnati 6, Ohio **OP**
**Firestone, Charles, '40,**
326 Medical Dental Bldg., Seattle, Wash. **ALR**
**Fischbach, V. W., '31**
Union Central bldg., Cincinnati 2, Ohio **ALR**
**Fischer, Abraham, '39**
922 Sibley Tower bldg., Rochester 4, N.Y. **OP**
Fischer, Aubrey, '34
1835 Eye St., N.W., Washington 6, D. C. **ALR**
**Fischer, Louise, '44**, Medical Towers,
31 Lincoln Park, Newark 2, N. J. **ALR**
**Fischer, N. Arthur, '25**
Union Trust bldg., Pittsburgh 19, Pa. **ALR**
**Fisher, George Garthwaite, '41**
271 New York av., Brooklyn 16, N. Y. **ALR**
**Fisher, Gilbert Eugene, '42**
Medical Arts bldg., Birmingham 5, Ala. **ALR**
Fisher, Guy Rothwell, '42..1 E. Beverley st., Staunton, Va. **ALR**
**Fisher, H. Noland, '37**....106 N. Silver st., Olney, Ill. **OP**
**Fisher, Harry Richard, '46**
465 West End Ave., New York, N.Y. **ALR**
**Fisher, James A., '25**...501 Grand av., Asbury Park, N. J. **OALR**
**Fisher, Lawrence Christian, '41**..469 W. Market, York, Pa. **OP**
Fishman, Louis Z., '35, Green's Eye Hospital,
1801 Bush, San Francisco 9, Calif. **ALR**
**Fishof, Frank E., '41** Capt. (MC).....Address Unknown **ALR**
Fitzgerald, Fred J. C., '34
118 E. 65th St., New York 21, N.Y. **OP**
**Fitzgerald, James Robert, '43**
3215 W. North av., Chicago 47, Ill. **OP**
**Fitzgerald, William W., '37**.... ..27 Ludlow, Yonkers, N.Y.
also, 425 Madison av., New York, N. Y. **OP**
**Fitz-Hugh, Glassell Slaughter, '39**
104 E. Market st., Charlottesville, Va. **ALR**
**Fitzhugh, William McP., Jr., '38,**
384 Post, San Francisco 8, Calif. **ALR**
**Flack, Clarence Elbert, '45**....715 Lake St., Oak Park, Ill. **OP**
Flack, J. Vincent, '37, Institute of Ophthalmology
635 W. 165th St., New York 32, N.Y. **OP**
**Flaherty, Norman W., '39**
10573 W. Jefferson ave., River Rouge 18, Mich. **OP**
**Flanders, Merion, '42**..Professional bldg., Waterville, Maine **ALR**
①**Flatley, T. J., '14**.....1518 Fifth av., Moline, Ill. **OALR**
**Flickinger, Roger R., '45**
Park Hospital Clinic, Mason City, Ia. **ALR**
**Flora, William Kenneth, '45**..406 Main st., Johnstown, Pa. **ALR**
**Flynn, Charles T., '27**..41 Trumbull, New Haven 10, Conn. **ALR**
**Flynn, James A., '24**
1511 Rhode Island av. N.W., Washington 5, D. C. **ALR**
**Flynn, J. Donald, '41**
240 Kendall Professional bldg., Grand Rapids, Mich. **ALR**
②**Fogarty, Charles W., '20**, 372 St. Peter, St. Paul 2, Minn. **OALR**
**Folken, Frank G., '28**..201 S. Broadway, Albert Lea, Minn. **OALR**
**Follweiler, Frank L., '43**....423 York rd., Jenkintown, Pa. **ALR**
**Folsom, W. H., '27**.....11 N. Main, Fond du Lac, Wis. **OALR**
**Font, Juan H., '33**, P.O. Box 5363, Puerta de Tierra
Station, Medical Arts bldg., San Juan, Puerto Rico **ALR**
**Foote, Charles M., '44**
412 N. Hastings Ave., Hastings, Nebr. **OP**
**Foote, Wm. D., '43**..1800 Eye St. N.W., Washington, D.C. **OP**
③**Forbes, Sherman B., '22**....706 Franklin, Tampa 2, Fla. **OP**
**Ford, Hanby L., '28**.....Robeson bldg., Champaign, Ill. **OALR**
**Ford, Harry C., '44**
1014 Medical Arts bldg., Oklahoma City 2, Okla. **ALR**
**Ford, Leroy Sargent, '42**.....30 School St., Keene, N. H. **ALR**
**Ford, William W., '38**....109 E. Walnut, Green Bay, Wis. **ALR**
**Forrester, Alex M., '43**..922 Heyburn bldg., Louisville, Ky. **ALR**
②**Forsythe, S. T., '19**.....Rose bldg., Cleveland 15, Ohio **OALR**
**Foster, Clarence Batchelder, '42**
520 Professional bldg., Charlotte, N. C. **OP**
**Foster, George C., '36**.....Fargo Clinic, Fargo, N. D. **OALR**
**Foster, Harold, '35**
92-11 35th av., Jackson Hts., New York, N. Y. **ALR**
①**Foster, J. H., '13**....1304 Walker av., Houston 2, Texas **ALR**
**Foster, Warren H., '35**....217 Fifth av. S., Clinton, Iowa **ALR**
**Foster, Wayne J., '35**....Higley bldg., Cedar Rapids, Iowa **ALR**
**Fowler, Earle B., '25**....55 E. Washington, Chicago 2, Ill. **OP**
**Fowler, Edmund Prince, Jr., '40**
180 Ft. Washington Ave., New York 32, N. Y. **ALR**
**Fowler, Floyd J., '43**, Akron Clinic,
513 W. Market, Akron 3, Ohio **ALR**
**Fowler, James G., '37**...412 Linwood av., Buffalo 9, N. Y. **OP**
**Fowler, Robert H., '29**..610 Park av., New York 21, N. Y. **ALR**
**Fowlkes, John W., '29**....12 E. 63rd, New York 21, N. Y. **ALR**
**Fox, C. Calvin, '37**.....1806 Spruce, Philadelphia 3, Pa. **ALR**
**Fox, Meyer S., '40**
208 E. Wisconsin av., Milwaukee 2, Wis. **ALR**
**Fox, Nathan H., '45**.....4010 W. Madison, Chicago, Ill. **OP**
**Fox, Noah, '26**.....841 E. 63rd, Chicago 37, Ill. **ALR**
Fox, Samuel L., '43, 1205 St. Paul st., Baltimore 2, Md. **ALR**
**Fox, Sidney A., '37**...63 E. 75th St., New York, 21, N. Y. **OP**
**Frackelton, Ralph J., '37**
15701 Detroit av., Lakewood 7, Ohio **ALR**
**Fralick, F. Bruce, '33**
First National bldg., Ann Arbor, Mich. **OP**
**Francis, Charles C., '39**
610 West End av., New York 24, N. Y. **ALR**
**Francis, Norton L., '41**..4325 E. Lewis, Wichita 8, Kans. **ALR**
Frank, David I., '37, 60 Gramercy Park, New York 10, N. Y. **ALR**
**Frank, Herman W., '43**.....Medical bldg., Gadsden, Ala. **OP**
**Fraser, R. H., '25**
25 W. Michigan av., Battle Creek, Mich. **ALR**
**Fraser, Robert C., '29,**
6777 Hollywood blvd., Los Angeles 28, Calif. **ALR**
**Fraser, W. G., '27**.....Medical Arts bldg., Ottawa, Can. **OALR**
**Frazee, John Richard, '36**..311 Beacon st., Boston, Mass. **ALR**
**Frazer, John Paul, '45**
224 Aberdeen St., Rochester 11, N.Y. **ALR**
**Fread, Bernard, '37**.....45 E. 85th, New York 28, N. Y. **OP**

**Fred, Gustave B., '36**...520 Beacon st., Boston 15, Mass. **ALR**
**Freedman, A. O., '32**
1396 St. Catherine st. W., Montreal, Can. **ALR**
①**Freedman, L. M., '13**
475 Commonwealth av., Boston 15, Mass. **ALR**
**Freeman, Marvin S., '44**
409 Osborn bldg., Cleveland 15, Ohio **ALR**
**Freeman, Sheldon B., '40**
196 Linwood av., Buffalo 9, N. Y. **OP**
Freimuth, L. E., '27
University Club bldg., St. Louis 3, Mo. **ALR**
①**French, R. F., '11**..Masonic Temple, Marshalltown, Iowa **OALR**
**French, Robert W., '37**.....181 Pleasant, Malden, Mass. **OP**
Freund, E. M., '41.....762 Madison av., Albany 3, N. Y. **ALR**
**Frey, Walter D., '43**.....200 N. Upper, Lexington 15, Ky. **OP**
**Frey, W. G., Jr., '34**....121 E. 60th, New York 22, N. Y. **OP**
Fried, Joseph John, '45.5 West 86th St., New York 24, N.Y. **OP**
**Friedberg, Stanton A., '39**
122 So. Michigan Ave., Chicago 3, Ill. **ALR**
①**Friedenwald, Harry, '04**
1212 Eutaw pl., Baltimore 17, Md. **OPA**
**Friedenwald, J. S., '25**..1212 Eutaw pl., Baltimore 17, Md. **OP**
Friedman, Benjamin, '33
485 Central Park W., New York, N. Y. **OP**
**Friedman, Elmer A., '41**
6733 Newgard Ave., Chicago 26, Ill. **ALR**
**Friedman, Joseph Jay, '46**
57 Midwood St., Brooklyn 25, N.Y. **ALR**
**Friedman, Louis Leonard, '40**
6078 Jenkins Arcade, Pittsburgh 22, Pa. **ALR**
**Fringer, Robert Chatterton, '46**
216 News Tower, Rockford, Ill. **OP**
①**Fringer, W. R., '04**.....109 Sheridan st., Rockford, Ill. **OP**
**Fritsche, Theodore R., '37**.Fritsche bldg., New Ulm, Minn. **OALR**
**Fritz, Milo H.**, '46.....927 Park av., New York 28, N.Y. **OALR**
**Frost, Carl Grover, '41**..333 Linwood av., Buffalo 9, N. Y. **ALR**
**Frothingham, Edward, '28**
8626 94th, Woodhaven 21, L. I., N. Y. **OALR**
**Frothingham, Fiorie D., '41**
133 E. 58th, New York 22, N. Y. **ALR**
Fry, W. E., '31.....1930 Chestnut, Philadelphia 3, Pa. **OP**
**Fuchs, Valentine H., '28**
American Bank bldg., New Orleans 12, La. **ALR**
①**Fulkerson, C. B., '15**
107 W. Michigan av., Kalamazoo 9, Mich. **OALR**
**Fuller, John A., '28**....First Nat'l Bank bldg., Reno, Nev. **OALR**
①**Fuller, T. E., '15**.....209 Pine, Texarkana, Texas **OALR**
**Fulton, Harry C., '37**.....339 N. Duke, Lancaster, Pa. **OP**
**Furlong, Frank C., '44**, 713 Union st., Schenectady, N. Y. **ALR**
**Furlong, Thomas F., Jr., '35**
Times Medical bldg., Ardmore, Pa. **ALR**
**Furman, Martin A., '36**,
1082 Park Av., New York 28, N.Y. **ALR**
**Furstenberg, A. C., '25**
First Nat'l bldg., Ann Arbor, Mich. **ALR**
②**Fust, John H.**, '22.....231 W. 7th, Erie, Pa. **OALR**

## G

**Gabriel, C. K., '30**..Illinois State Bank bldg., Quincy, Ill. **OALR**
**Gadomski, Casimir Francis, '42**
331 S. Broad st., Elizabeth, N.J. **OP**
Gagion, Thomas Reed, '40.....23 Broad st., Pittston, Pa. **OP**
**Gaida, Joseph B., '37**....St. Mary's bldg., St. Cloud, Minn. **OP**
②**Gailey, Watson W.**, '22..1008 N. Main, Bloomington, Ill. **OP**
**Gaillard, Milton T.**, '44.....5 Park av., Baldwin, N. Y. **OP**
**Gaines, Shelley Rice, '46**
American Bank bldg., New Orleans 12, La. **OP**
**Gaines, Thomas R., '40**..Professional bldg., Anderson, S. C. **OP**
③**Gale, M. Jean, '18**.....Republic bldg., Denver 2, Colo. **OP**
**Gallizzi, Vincent D.**, '42,
307 Dime Bank bldg., Pittston, Pa. **ALR**
**Galloway, T. C., '27**.....636 Church, Evanston, Ill. **ALR**
**Gambill, Perry Judson, '42**
New Jersey State Hospital, Greystone Park, N. J. **ALR**
**Gamble, R. C.**, '29.....30 N. Michigan av., Chicago 2, Ill. **OP**
**Gans, Jerome A.**, '43....922 Keith bldg., Cleveland, Ohio **OP**
①**Garber, J. M., '11**.....14 N. Mulberry, Mansfield, Ohio **OALR**
Gardiner, Charles Ernest, '46, 701 Chestnut st., Lebanon, Pa. **ALR**
**Garland, Frederick E., '34**...483 Beacon, Boston 15, Mass. **LR**
**Garlichs, Richard Walton, '46**
216 North Manoa Road, Havertown, Pa. **ALR**
**Garlick, George B., '36**.144 Golden Hill, Bridgeport 3, Conn. **ALR**
**Garlock, Arthur Varney, '41**....217½ 3rd, Bemidji, Minn. **OP**
**Garnier, William Hampton, '41**
Doctors bldg., Madisonville, Ky. **ALR**
**Garofalo, Charles J., '37**.439—77th st., Brooklyn 9, N. Y. **ALR**
**Garrett, Ernest W., '35**...Hanna bldg., Cleveland 15, Ohio **ALR**
**Garrett, John D., '32**, Bar Ass'n bldg., Indianapolis 4, Ind. **ALR**
**Garrick, Thomas J., '36**...40 E. 61st, New York 21, N. Y. **ALR**
**Garten, Joseph L., '40**,
502 Medical Arts bldg., Minneapolis, Minn. **OALR**
**Gartner, Samuel**, '39....521 Park av., New York 21, N. Y. **OP**
Garvey, Thomas Quincy, '46, 115 E. 61st st., New York, N.Y. **ALR**
**Gaston, Ira E., '38**
833 Medical-Dental bldg., Portland 5, Ore. **OALR**
**Gatewood, E. T., '25**.Professional bldg., Richmond 19, Va. **ALR**
①**Gatewood, W. L., '15**
111 E. 61st st., New York 21, N. Y. **ALR**
**Gaudet, Lucien S., '28**.....405 Franklin, Natchez, Miss. **OALR**
**Gaudreau, Honore E., '34**..293 Bridge, Springfield 3, Mass. **OP**
**Geary, Russell Diener, '42**
337 Bridgeboro Road, Riverside, N. J. **ALR**
**Geeseman, George R., '42**
634 Washington rd., Pittsburgh 16, Pa. **OP**
**Geiger, Dillon**, '41....300 E. Kirkwood, Bloomington, Ind. **ALR**
**Gemeroy, Joseph C., '35**
664 Fisher Bldg., Detroit 2, Mich. **OP**
**Gemmill, Walter D., '39**.....130 Seventh, Monessen, Pa. **ALR**
**George, Arthur R.**, '41
291 "E" St., San Bernardino, Calif. **ALR**
**George, Forney P., '37**.....118 S. Hanover, Carlisle, Pa. **ALR**
**Gernon, William**, '41....542 S. Schuyler Av., Kankakee, Ill. **OP**
**Gershon, Nathan Isaac, '45**
26 Linden Ave. N.E., Atlanta, Ga. **ALR**
**Gerstley, Manfred J., '33**
83 E. Genesee St., Skaneateles, N.Y. **ALR**
**Gerstner, Martin Llewellyn, '40**
27 Admiral rd., Buffalo 16, N. Y. **ALR**
**Gerzog, Bennett G., '37**
891 St. Marks av., Brooklyn 13, N. Y. **ALR**
**Gessler, Wm. F., '33**
2902 Fairfield av., Ft. Wayne 6, Ind. **OALR**
**Gettes, Bernard Charles, '46**
2028 Pine St., Philadelphia, Pa. **OP**
**Gettes, Charles N., '44**, 481 Beacon st., Boston 15, Mass. **ALR**
**Gewanter, Robert, '37**
41-44 44th st., Long Island City 4, N. Y. **ALR**
**Gibb, William B., '44**, 1515 State st., Santa Barbara, Calif. **OP**

**Gibby, Harold J., '31**.....27 Elm, Worcester 8, Mass. ALR
**Gibson, Glen G., '35**.....255 S. 17th, Philadelphia 3, Pa. OP
**Gibson, William J., '43**....650 Main, New Rochelle, N. Y. ALR
**Gidoll, Sidney H., '42**
516 Sutter st., San Francisco 2, Calif. ALR
**Gifford, Harold, '37**....Medical Arts bldg., Omaha 2, Nebr. OP
Gilbert, Joseph G., '36
285 New York ave., Brooklyn 16, N.Y. ALR
**Gilbert, Ralph H., '41**..Metz bldg., Grand Rapids 2, Mich. OP
**Gilfillan, Homer J.**, '41.....Gilfillan Clinic, Bloomfield, Ia. ALR
②**Gill, E. G., '19**.....711 S. Jefferson, Roanoke 10, Va. ALR
**Gill, Earl King**, '36
416 Chaparral st., Corpus Christi, Texas ALR
**Gill, G. Bache, '23**
1726 Eye st. N.W., Washington 6, D. C. ALR
**Gill, Wm. Davis, '29**
705 E. Houston, San Antonio 5, Texas OALR
Gillespie, Elmer H., '38.....6 State St., Reno, Nev. ALR
①**Gillett, Wilbur G.**, '20,..201½ N. Main, Wichita 2, Kan. OP
**Gillette, D. F., '25**.....State Tower, Syracuse 2, N. Y. OP
**Gilligan, Alexander, '39**....1154 Dean, Brooklyn 16, N. Y. ALR
Gilligan, John Henry, '46..1831 Wilson Blvd. Arlington, Va. ALR
**Gillilland, O. S., '30**.Professional bldg., Kansas City 6, Mo. ALR
**Gillis, Marion Howard, Jr., '43**
204 Walnut, Salisbury, Md. OP
②**Gillum, J. R., '20**.....221 S. 6th, Terre Haute, Ind. OALR
Gilmore, Goodlatte Browne, '41
2940 Grand Concourse, New York 58, N. Y. ALR
**Gilmore, Milton A., '36**.....Box 172, Parkersburg, W. Va. OP
**Gilmore, Robert O., '39**.....1159 Hancock, Quincy, Mass. ALR
**Gipner, J. F., '26**...Medical Arts bldg., Rochester 7, N. Y. OP
Girard, Paul H., '44, 258 Genesee St., Utica, N. Y. OP
**Gissy, C. J., '43**..717 Metropolitan bldg., St. Louis 3, Mo. OP
②**Gittins, T. R., '20**....Davidson bldg., Sioux City 13, Ia. OALR
**Givner, Isadore, '36**.....108 E. 66th, New York 21, N. Y. OP
**Glasford, S. T., '24**.....416 Elizabeth, Pekin, Ill. OALR
Glasgow, S. Condit, '44, 8 E. Alisal St., Salinas, Calif. ALR
**Glatt, Morris A., '31**....55 E. Washington, Chicago 2, Ill. ALR
Glauber, Jerome J., '44....878 Lafayette, Buffalo 9, N.Y. ALR
Gleason, John E., '40
David Whitney bldg., Detroit 26, Mich. ALR
Glick, Harry N., '28 1504 S. Grand blvd., St. Louis 4, Mo. ALR
①**Glosser, Herbert H.**, '12.....47 N. Broad, Wellsville, N.Y. OP
Glover, Arthur C., '33.....311 W. Church, Elmira, N. Y. OALR
Glover, Lewis P., '38....1200 Fourteenth av., Altoona, Pa. OP
Glowacki, Ben F., '28....Maccabees bldg., Detroit 2, Mich. OALR
Glushak, Leopold, '38..1 West 85th, New York 24, N. Y. ALR
Goad, R. R., '31.....110 W. 2nd St., Muscatine, Iowa OP
Goar, Everett L., '26...1300 Walker av., Houston 2, Texas OP
Godwin, Dean E., '44, 117 E. 8th st., Long Beach 2, Calif. ALR
Goehring, Donald Everett, '41
Butler S. & T. bldg., Butler, Pa. ALR
Goetzman, Arthur C., '42,
308 United Office bldg., Niagara Falls, N. Y. OP
①**Goldbach, L. J., '11**.....6 East Eager, Baltimore 2, Md. OP
Goldberg, Herman K., '42
719 N. Charles st., Baltimore, Md. OP
Goldberg, Sol, '39, 8082 Jenkins Arcade, Pittsburgh 22, Pa. OP
Goldberg, Sylvan S., '35
Suite B, 121 N. San Vicente blvd., Beverly Hills, Cal. ALR
Goldcamp, Edward C., '32
Dollar Bank bldg., Youngstown 3, Ohio ALR
Goldcamp, John S., '41
810 Dollar Sav. & Tr. Co. bldg., Youngstown 3, Ohio OP
Goldenburg, Michael, '31
104 S. Michigan av., Chicago 3, Ill. OP
Goldhammer, Samuel, '45..Republic Bldg., Denver 2, Colo. OP
Golding, Joseph E., '25.....1 Nevins, Brooklyn 17, N. Y. OP
Goldmacher, H. B., '43.113 Elmora av., Elizabeth 2, N. J. ALR
Goldman, A. Milton, '28
1 N. Village av., Rockville Centre, L. I., N. Y. OP
Goldman, H. G., '23..57 W. 58th st., New York 19, N. Y. ALR
Goldman, Irving Brice, '40
121 E. 60th, New York, 22, N. Y. ALR
Goldman, Jerome, '46.....1 Johnson Av., Newark 8, N.J. OP
Goldsmith, Jacob, '41..30 E. 60th St., New York 22, N.Y. OP
①Goldsmith, Perry '12, Med. Arts bldg., Toronto 5, Can. ALR
Goldstein, Henry Z., '36....31 Lincoln Park, Newark, N.J. ALR
**Goldthwaite, Ralph H., '35**, Brig. Gen. (MC) (Retired)
4515 Mead av., Sacramento, Calif. OALR
**Gollom, Joseph, '38**.....425 Bloor st. W., Toronto, Can. ALR
**Gomon, Louis D., '37**.....Eddy bldg., Saginaw, Mich. OP
**Gonne, Wm. S., '36**
David Whitney bldg., Detroit 26, Mich. ALR
**Gooch, John B., '35**
Canal Bank bldg., New Orleans 12, La. ALR
Good, Palmer W., '37.....715 Lake St., Oak Park, Ill. OALR
Good, Richard P., '41
308 Armstrong-Landon bldg., Kokomo, Ind. ALR
**Good, Wm. M., '29**.....63 Center, Waterbury 18, Conn. ALR
Goodale, Robert L., '36..330 Dartmouth, Boston 16, Mass. ALR
**Goodall, Edwin B., '34**..71 Bay State Rd., Boston 15, Mass. OP
**Goode, Edward A., '29**
143—28 Sanford av., Flushing, N. Y. ALR
Goodell, William, '34....1066 S. Citrus ave., Sarasota, Fla. ALR
**Goodfellow, Thomas J., '39**
Arcade bldg., Saratoga Springs, N. Y. OALR
**Goodhill, Victor**, '42, 676 S. Westlake, Los Angeles 5, Calif. ALR
Goodman, E. Leonard, '36
1801 Eye st. N.W., Washington 6, D. C. OP
**Goodman, Thomas L., '31**
1601 Clover Lane, Fort Worth 7, Texas ALR
**Goodspeed, Harry B., '34**
36 Pleasant, Worcester 8, Mass. ALR
**Goodwin, Thomas M., '38**....Golden Clinic, Elkins, W. Va. ALR
Goodwin, Vernon L., '43
509 Central Tower, Youngstown 3, Ohio ALR
**Goodyear, H. M., '24**....Doctors bldg., Cincinnati 2, Ohio ALR
Gordon, Benjamin L., '44
Prof. Arts bldg., Atlantic City, N. J. OP
**Gordon, Charles H., '26**....46 Deering, Portland 4, Maine ALR
Gordon, Dan Morris, '45
525 E. 68th St., New York 21, N.Y. OP
Gordon, George Raoul, '46
913 S. 19th St., Birmingham, Ala. ALR
**Gordon, Glenn G., '29**.....Alexander bldg., Baker, Ore. ALR
Gordon, John Simpson, '46, 950 E. 59th st., Chicago 19, Ill. ALR
**Gordon, William, '33**.....1721 Pine, Philadelphia 3, Pa. ALR
**Gorin, Meyer, '42**.....35 Chestnut, Rochester 4, N. Y. ALR
**Gorman, James R., '36**...Allied Arts bldg., Lynchburg, Va. ALR
Gorney, Arthur J., '39, 14 Charlesgate West, Boston, Mass. ALR
①**Gorrell, T. J. H., '09**
16th and Vincennes, Chicago Heights, Ill. OALR
Gorrilla, Laurence Vincent, '42
511 S. Bonnie Brae st., Los Angeles 5, Calif. OP
②**Gosney, C. W.**, 20,
5217 Hollywood blvd., Los Angeles 27, Calif. OP
**Goss, Harold L., '37**....1305 Fourth av., Seattle 1, Wash. OP
**Gottlieb, Bernard N., '39**
387 Eastern Parkway, Brooklyn 16, N. Y. ALR
**Gough, Roy H., '31**....600 W. 10th, Fort Worth 2, Texas OALR

**Gould, Nathaniel Jay, '37**
940 Grand Concourse, New York 56, N. Y. OP
Gouterman, Joseph L., '30, 1930 Chestnut, Philadelphia 3, Pa. OP
②Goux, Raymond S., '22
David Whitney bldg., Detroit 26, Mich. OALR
Gowen, Thomas F., '39, Comdr. (MC) USNR
N.A.S. Dispensary, Norfolk, Va. ALR
Gowens, Henry L., Jr., '42
2217 Delancey, Philadelphia 3, Pa. OP
Grace, Charles C., '33.....145 King, St. Augustine, Fla. ALR
Gracey, George F., '24.....209 State, Harrisburg, Pa. OALR
①Gradle, Harry S., '11
14060 Valley Vista blvd., Sherman Oaks, Calif. OP
Graebner, Herbert, '34, American Airlines, NYMA,
Jackson Heights, New York, N. Y. ALR
Graef, Fred W., '36....140 E. 54th, New York 22, N. Y. ALR
Graham, George Albert, '45
842 Carroll St., Brooklyn 15, N.Y. OP
Granberry, Carl, '34
Maison Blanche bldg., New Orleans 16, La. ALR
②Granger, W. B., '20.....Gazette bldg., Emporia, Kans. OALR
①Grant, Fred E., '12
Kalamazoo Nat'l Bank bldg., Kalamazoo 9, Mich. OALR
Grant, H. W., '30.Lowry Med. Arts bldg., St. Paul 2, Minn. OP
Grant, Maurice David, '41
1860 Morris av., New York 53, N. Y. ALR
③Graves, Basil, '26.....Address Unknown
Greear, James M., Jr., '29
1740 M st. N.W., Washington 6, D. C. OP
Green, Hyman S., '35..25 E. Washington st., Chicago, Ill. OALR
①Green, John, '05
3720 Washington blvd., St. Louis 8, Mo. OP
Green, Stanley L., '44, 129 W. Lexington, Independence, Mo. OP
Greenburg, Maynard M., '39
516 Medical Arts bldg., Omaha 2, Nebr. OP
Greene, James S., '36....61 Irving pl., New York 3, N. Y. ALR
Greene, Laurence W., '28..Republic bldg., Denver 2, Colo. ALR
Greene, Maurice L., '30
501 Missouri Theater bldg., St. Louis 3, Mo. OP
Greene, Philip Burkland, '46
517 Old National Bank bldg., Spokane, Washington OP
Greenfield, Samuel D., '27
169 New York av., Brooklyn 16, N. Y. ALR
Greenfield, William J., '28, 50 Anderson, Hackensack, N. J.,
and 30 E. 40th, New York 16, N. Y. ALR
Greenlee, Max R., '38.Mahaska Theatre bldg., Oskaloosa, Ia. ALR
Greenwood, Glenn J., '30
154 Medio dr., Los Angeles 24, Calif. ALR
Greetham, James S., '46, Major (MC)
McCornack General Hosp., Pasadena 2, Calif. OP
②Greever, B. L., '20.....100 N. Main, Hutchinson, Kan. OALR
②Gregg, John B., '20
Security Nat'l Bank bldg., Sioux Falls, S. D. OALR
Gresser, Edward B., '29...39 E. 75th, New York 21, N. Y. OP
Gressette, James H., '44.....Orangeburg, S. C. ALR
Griesemer, Z. Lawrence, '43,
1145 E. Jersey, Elizabeth 4, N. J. OP
Griesman, Bruno L., '39...3 E. 66 st., New York 21, N.Y. ALR
Grieve, Harry Gordon, '44
315 Scollard bldg., Victoria, B.C., Canada OALR
Griffey, Edward W., '29
Medical Arts bldg., Houston 2, Texas OALR
Griffey, Walter P., '39
U. S. Marine Hospital, Staten Island 4, N. Y. OP
Griffin, Edwin A., '34
18 Prospect Park West, Brooklyn 15, N. Y. ALR
Griffith, Harold M., '30
Johnstown Bank & Trust bldg., Johnstown, Pa. OP
Grigsby, Roll O., '40....100 E. Second st., Ashland, Wis. ALR
Grimaldi, Americo John, '40
100 West 59th, New York 19, N. Y. OP
Grimmett, Roger Skelton, '40
5938 Newton Wynd., Vancouver, B.C., Canada ALR
Gromer, Terry John, '46
110 Metropolitan bldg., Denver, Colo. ALR
Gromet, Maurice T., '45
105 Lincoln Rd., Brooklyn 25, N.Y. ALR
②Gross, Julius E., '20
Veterans Administration, Bay Pines, Fla. ALR
Grossman, Herman P., '38..210 Angell, Providence 6, R. I. OP
Grossman, Erwin E., '44
238 W. Wisconsin av., Milwaukee, Wis. OP
Grove, Bruce Alexander, '42....426 W. Market, York, Pa. OP
Grove, Russell C., '37....60 E. 58th, New York 22, N. Y. ALR
①Grove, W. E., '14
324 E. Wisconsin av., Milwaukee 2, Wis. OALR
Gruppe, Karl W., '38.....258 Genesee, Utica 2, N. Y. ALR
Gsell, George F., '38..911 Beacon Bldg., Wichita 2, Kans. OP
②Gsell, J. F., '20....911 Beacon bldg., Wichita 2, Kans. OALR
Gubner, Julius, '37.....25 Lefferts av., Brooklyn 25, N. Y. ALR
Guerry, DuPont III, '45
503 Professional bldg., Richmond, Va. OP
②Guggenheim, L. K., '18
321 Glenroy av., Los Angeles 24, Calif. ALR
Guibor, George P., '33, 303 Central Life bldg., Ottawa, Ill. OP
Gulick, John Duncan, '41.704 Union, Schenectady 5, N. Y. ALR
Gump, Millard E., '39....411 30th St., Oakland 9, Calif. OP
①Gundelach, C. A., '12
University Club bldg., St. Louis 3, Mo. ALR
Gundersen, Trygve, '35..101 Bay State rd., Boston, Mass. OP
Gunderson, Ernest O., '44..2511 Hearst Av., Berkeley, Cal. OP
Gunderson, Harvey Clifford, '45
2001 Collingwood Ave., Toledo 2, Ohio ALR
Gundrum, Lawrence Kramer, '42
1930 Wilshire blvd., Los Angeles 5, Calif. ALR
Gunn, Milus L., '29..... 107 E. Mound, Harlan, Ky. ALR
Gurau, Henry H., '46
215 Bankers Trust Bldg., Des Moines, Iowa OP
Gurlov, John J., '37
1235 Grand Concourse, New York 52, N. Y. ALR
Gurwin, Bernard J., '45
900 17th St. N.W., Washington 6, D.C. OP
Gustafson, Olga Frideberg, '38
185 N. Wabash av., Chicago 1, Ill. OP
Gutkin, Morton Lewis, '45..6734-6th Av., Brooklyn, N.Y. ALR
Guttman, M. Reese, '29.30 N. Michigan av., Chicago 2, Ill. ALR
Guy, Loren P., '34.....40 E. 62nd, New York 21, N. Y. OP
②Guyton, B. S., '18....512 Van Buren av., Oxford, Miss. OALR
Guyton, Jack Smallwood, '41
Johns Hopkins Hospital, Baltimore 5, Md. OP
Guzek, Joseph T., '43....616 Connell bldg., Scranton 3, Pa. OP

## H

Haas, Louis Richard, '42....Smith Clinic, Pittsburg, Kan. OALR
①Haden, Henry C., '13.....1914 Travis, Houston 3, Texas OP
Haessler, F. Herbert, '29...Wells bldg., Milwaukee 2, Wis. OP
Hagaman, Van Dyke, '34
Lamar Life bldg., Jackson 2, Miss. ALR

Hagan, Cornelius E., Jr., '42
1918 S. Oneida, Spokane, Wash. ALR
Hageman, George Ricker, '42
Longmont Hospital, Longmont, Colo. ALR
Hagens, Elmer William, '41.30 N. Michigan, Chicago 2, Ill. ALR
Haggart, Harry H., '37,
920 Carew Tower, Cincinnati 2, Ohio ALR
Hagood, Daniel S., '43
822 First Nat'l Bank bldg., Montgomery 4, Ala. OP
Hague, Elliott B., '39.....454 Franklin, Buffalo 2, N. Y. OP
Hahn, Wm. H., '30.....198 Clinton Av., Newark, N.J. OP
Hahn, William N., '38
City Nat'l Bank bldg., Omaha 2, Nebr. OP
Haik, George M., '43, 812 Maison Blanche bldg.,
New Orleans, La. OP
Haines, Henry L., '43....309 State st., New London, Conn. ALR
Haire, Paul G., '35.....6 Pleasant, Malden 48, Mass. OP
Hale, Arthur S., '28.....Eaton Tower, Detroit 26, Mich. OALR
Haley, Paul J. D., '37.....So. Harwick, Mass. ALR
Haley, Wm. T., '33.....531 Beacon, Boston 15, Mass. ALR
Hall, Alice K., '24.....25 E. Washington, Chicago 2, Ill. ALR
Hall, Colby, '42.....1136 W. 6th St., Los Angeles, Calif. ALR
Hall, Donald S., '44
Street Clinic Library, Crawford & Monroe sts.,
Vicksburg, Miss. OALR
Hall, Edwin P., '28.....Box 212, Oneonta, N.Y. ALR
Hall, Lawrence H., '39....1520 Oak, So. Pasadena, Calif. OALR
Hall, Raymond A., '39
514 Home Sav. & Loan bldg., Youngstown, Ohio ALR
Hall, Sobisca S., '34, Empire Bank Bldg., Clarksburg, W. Va. ALR
Hallaron, John J., '44
3405 St. Charles av., New Orleans, La. ALR
Hallberg, Olav Erik, '42.....Mayo Clinic, Rochester, Minn. ALR
Hallum, Alton V., '34.....478 Peachtree, Atlanta 3, Ga. OP
②Halsted, Fred S., '18
Metropolitan bldg., Denver 2, Colo. OALR
Halton, Edward P., '37
1456 Northampton, Holyoke, Mass. ALR
Hamilton, Howard Eugene, '42
10515 Carnegie av., Cleveland 6, Ohio ALR
Hamilton, Richard G., '40
Medical Arts bldg., Pittsburgh 13, Pa. ALR
Hamlin, Fred E., '26.....P.O. Box 2276, Roanoke, Va. ALR
Hammond, Arthur, '35
David Whitney bldg., Detroit 26, Mich. ALR
Hampsey, Joseph W., '37....May bldg., Pittsburgh 22, Pa. ALR
Hamrick, DeWitt, '40.....601 Jackson, Corinth, Miss. ALR
Hanckel, Richard White, Jr., '42
96-A Bull, Charleston 16, S. C. ALR
Hancox, Cecil W., '39..Bahr Arcade bldg., Pottstown, Pa. ALR
Hand, Frank, '35.....450 Sutter, San Francisco 8, Calif. ALR
Hands, Sidney G., '26.....201 W. 2nd, Davenport, Iowa ALR
②Haney, Wm. P., '20, Medical Arts bldg., Omaha 2, Nebr. OALR
Hankins, George Geddy, '41
Medical Arts bldg., Newport News, Va. ALR
Hanley, Swift, '37....40 W. 55th, New York 19, N. Y. ALR
Hanley, Jeanne Roeling, '39
Maison Blanche bldg., New Orleans 16, La. ALR
Hannon, R. Emmet, '42.364 Hanna bldg., Cleveland 15, Ohio OP
②Hans, C. L., '19..Provident Bank bldg., Cincinnati 2, Ohio OP
Hansel, French K., '26.634 N. Grand av., St. Louis 3, Mo. ALR
②Hansen, Erling W., '20
Medical Arts bldg., Minneapolis 2, Minn. OP
Hanser, Samuel Albert, '43
3424 Longfellow blvd., St. Louis 4, Mo. OP
Hanson, A. George, '45
1528 Medical Dental Bldg., Seattle 1, Wash. OP
Hanson, Frederick Charles, '46
162 Angell St., Providence, R.I. OP
Hanson,, Wm. L., '29, Spivey bldg., East St. Louis, Ill. ALR
Hantman, Irvin, '39, Capt. (M.C.),
c/o Greenberg, 3647 Cottage av., Baltimore, Md. ALR
Happ, Linley C., '41, 170 Waterman st., Providence 6, R.I. ALR
Hare, H. James, '33..436 S. Boyd Av., Los Angeles 33, Cal. ALR
Haralson, Charles H., '29.Medical Arts bldg., Tulsa 3, Okla. OP
②Harbert, John P., '19, 135 N. Main, Bellefontaine, Ohio OALR
②Hardesty, J. F., '22
Missouri Theatre bldg., St. Louis 3, Mo. OP
Hardie, G. C., '30....290 W. Michigan av., Jackson, Mich. OALR
Harding, Myron S., '38
308 Hume-Mansur bldg., Indianapolis 4, Ind. OP
Hardy, Guerdan, '38..539 N. Grand blvd., St. Louis 3, Mo. OALR
Hardy, LeGrand H., '28...23 E. 79th, New York 21, N. Y. OP
①Hardy, W. F., '14.....Humboldt bldg., St. Louis 3, Mo. OP
Hardy, Walter B., '36.2121 Highland, Birmingham 5, Ala. ALR
Hargitt, Charles A., '26.17 Schermerhorn, Brooklyn 2, N. Y. OP
Harkins, Herbert P., '42
1930 Chestnut st., Philadelphia 3, Pa. ALR
Harkins, William Blake, '41
3720 Washington ave., St. Louis 8, Mo. ALR
①Harkness, Gordon F., '11
Putnam bldg., Davenport, Iowa OALR
Harley, Robison D., '46
101 So. Indiana Av., Atlantic City, N.J. OP
Harlowe, Harold Douglas, '40
Garberson Clinic, Colony bldg., Miles City, Mont. ALR
Harned, John W., '39..902½ S. Main st., Hopkinsville, Ky. ALR
Harner, Clyde E., '39..491 W. Gilman st., Banning, Calif. OALR
Harnick, Israel, '39..1132 Delaware av., Buffalo 9, N. Y. ALR
Harper, DeWitt Lawrence, '45
111 East North st., Greenville, S.C. ALR
Harrell, Voss, '26.....Hofman bldg., Detroit 1, Mich. ALR
Harrill, James A., '41
Bowman Gray School of Medicine, Winston Salem, N. C. ALR
Harrington, John Thomas, '40, Comdr. (M.C.), USNR,
Address Unknown ALR
Harrington, Silas Frederick, '42
4227 Herschell, Dallas 4, Texas ALR
①Harris, C. M., '10.Johnstown Trust bldg., Johnstown, Pa. OALR
Harris, Carl B., '41
319 Hume Mansur bldg., Indianapolis 4, Ind. OP
Harris, Edward W., '30
240 East State st., Columbus 15, Ohio ALR
①Harris, H. B., '10.Fidelity Medical bldg., Dayton 2, Ohio OALR
Harris, Harold Earl, '42.Cleveland Clinic, Cleveland 6, Ohio ALR
Harris, Herbert H., '39
1039 Mellie Esperson bldg., Houston 2, Tex. ALR
Harris, Herman Lee, '46
Reynolds Arcade bldg., Bristol, Va. ALR
Harris, J. Harley, '31
Methodist Hosp., Doctors bldg., Memphis 4, Tenn. ALR
Harris, Louis D., '44.....242 Trumbull, Hartford, Conn. OP
Harris, Morris, '44.....102 Broad st., Bloomfield, N.J. OP
Harris, Robin, '23.....964 N. State, Jackson 6, Miss. ALR
Harrison, Fred H., '28
Second Nat'l Bank bldg., Connellsville, Pa. OALR
Harrison, Glen H., '44..307 Washington st., Waukegan, Ill. OP

Hart, Noah Leon, '44, Capt. (M.C.), 0-460831,
Station Hospital, A.A.F., Kearney, Nebr. ALR
Hart, Verling Kersey, '46.108 West 7th St., Charlotte, N.C. ALR
Hartenbower, George E., '38, 1212 Broadway, Normal, Ill. ALR
Hartgraves, Hallie, '38
St. Mary's Convent, Kenosha, Wis. OP
Hartman, Deane C., '44
727 W. Seventh st., Los Angeles 14, Calif. OP
Hartman, Eugene C., '33
610½ Market, Parkersburg, W. Va. OALR
Hartshorne, Isaac, '26..30 W. 59th, New York 19, N. Y. OP
①Hartzell, Sol. M., '11
137 So. Peck dr., Beverly Hills, Calif. OALR
Harvey, Elmer Frank, '46
1422 Euclid Av., Cleveland 15, Ohio OP
①Harvey, N. Darrell, '04.112 Waterman, Providence 6, R.I. OALR
②Haskell, Alfred W., '18.....142 High, Portland 3, Maine OP
①Haskin, W. H., '13...375 Park av., New York 22, N. Y. ALR
Hasty, F. E., '25.....1203 Asturia, Coral Gables, Fla. ALR
Hatcher, William F., '38
804 Medical Arts bldg., Roanoke 11, W. Va. OP
①Hauer, A. M., '16.....350 E. State, Columbus 15, Ohio ALR
①Haughey, Wilfrid H., '11.Post bldg., Battle Creek, Mich. OALR
Haughton, Thomas J., '44
313 McCallum-Hill bldg., Regina, Sask., Canada ALR
Hauser, I. Jerome, '38,
606 David Broderick Tower, Detroit 26, Mich. ALR
Hauser, Maurice J., '43
2311 Broderick Tower, Detroit 26, Mich. OP
Hausmann, Gertrude S., '44.629 16th st., Denver 2, Colo. OP
Haven, Walter K., '44
1009 Nicollett av., Minneapolis 2, Minn. OALR
Havens, Fred Z., '36....Mayo Clinic, Rochester, Minn. ALR-PI S
②Hawman, Erle G., '20
4051 N. Broad, Philadelphia 40, Pa. OP
Hawn, Hugh W., '44
807 Broadway, Fargo Clinic, Fargo, N. D. OP
Hawthorne, Allen T., '38
110 N. Braddock, Winchester, Va. ALR
Hayden, Jerome William, '42
4458 Madison st., Chicago 24, Ill. ALR
Hayes, Merrill B., '40.....710 Madison st., Chester, Pa. ALR
Hayes, Robert T., '36....42 Coburg, St. John, N. B., Can. ALR
Hays, Arthur V., '46.....1300 27th ave., Gulfport, Miss. ALR
Hays, Charles E., '39....U. S. Bank bldg., Johnstown, Pa. ALR
Hazen, S. Frank, '39.....231 Chestnut, Meadville, Pa. ALR
①Heard, Mary K., '10
804 4th Ave. N. St. Petersburg 4, Fla. (Retired) OP
Heare, Louis C., '39.....Box 312, Port Arthur, Texas OP
Hearn, Paul P., '43..384 Post St., San Francisco 8, Calif. ALR
Heath, Parker, '24..David Whitney bldg., Detroit 26, Mich. OP
Hebble, Howard Miller, '40
320 Chester av., Moorestown, N. J. ALR
Hebert, Arthur W., '27.450 Sutter, San Francisco 8, Calif. OALR
Heckert, Frank B., '44
1105 Bank of Lansing bldg., Lansing, Mich. OP
Hedemark, Norman G., '43
218 Eastman Bldg., Boise, Idaho OALR
Hedlund, G. O., '36.....616 Mentor av., Painesville, Ohio ALR
③Heeb, Harry J., '18
212 W. Wisconsin av., Milwaukee 3, Wis. OALR
①Heed, C. R., '10.....1205 Spruce, Philadelphia 7, Pa. OP
Heetderks, D. R., '30
Medical Arts bldg., Grand Rapids 2, Mich. ALR
Heidelman, George, '39
Union Central bldg., Cincinnati 2, Ohio OP
Heinberg, Charles J., '31...Medical Center, Pensacola, Fla. ALR
Heine, Lyman H., '38.....635 N. Main, Fremont, Nebr. ALR
Heine, William Howard, '41
Odd Fellow bldg., Fremont, Nebr. ALR
①Heitger, J. D., '05.....Heyburn bldg., Louisville 2, Ky. OALR
Helbling, Franklin Kirkland, '45
34 No. Ash St., Ventura, Calif. ALR
Held, Albert E., '39..732 Eastern Pkwy., Brooklyn 13, N.Y. ALR
②Hempstead, Bert E., '20..Mayo Clinic, Rochester, Minn. ALR
Henderson, Charles Henry, '45..800 Park Ave., Norton, Va. OP
Henderson, James Alexander, '46
The Quincy Clinic, 1416 Maine St., Quincy, Ill. ALR
Henderson, L. E., '30.....129 Clinton, Watertown, N. Y. OP
Hendricks, Louis J., '31...Carew Tower, Cincinnati 2, Ohio OP
Henner, Robert, '37, Capt. (M. C.),
2315 Sheridan rd., Highland Park, Ill. ALR
②Hennessey, Wm. W., '20.....333 Essex, Salem, Mass. OALR
①Henning, Carl, '08
1752 Massachusetts av. N.W., Washington 6, D. C. OP
①Henninger, L. L., '13
65 N. Madison av., Pasadena 1, Calif. OALR
Henrich, Mary Ingeborg, '46
6214 Winthrop av., Chicago, Ill. OP
Henrickson, John William, '46
2001 Fourth Ave., San Diego, Cal. ALR
Henry, George Arnold, '40,
Medical Arts Bldg., Toronto 5, Ont. Canada ALR
Henry, L. L., '43.....595 E. Colorado, Pasadena 1, Calif. OP
Henry, Robert T., '42.....Grady bldg., Colfax, Wash. OP
Hensle, Otto S., '40....250 Prospect av., Hackensack, N.J. ALR
②Henton, George E., '18
812 S.W. Washington, Portland 5, Ore. OP
Henton, George H., '37
812 S. W. Washington st., Portland 5, Ore. OP
Herr, Albert H., '37....129 N. Elizabeth st., Lima, Ohio ALR
Hersh, Joseph H., '44..57 W. 57th st., New York 19, N.Y. ALR
Hert, Cecil B., '40....29 N. Goodman st., Rochester, N.Y. ALR
Hertz, William J., '36 .....125 N. 8th, Allentown, Pa. ALR
Herzig, Arthur J., '41.667 Madison av., New York 21, N. Y. ALR
Herzog, Max, '41.....2815 8½ av., Rock Island, Ill. OP
①Hess, William L., '04
2018 Fairburn, West Los Angeles 25, Calif. OALR
Hester, Marion William, '46
317 Marble Arcade Bldg., Lakeland, Florida OP
Hetrick, J. A. Werner, '38
667 Madison av., New York 21, N. Y. ALR
Hetrick, L. E., '23.....40 E. 61st, New York 21, N. Y. ALR
and 260 North av., New Rochelle, N. Y.
①Hetzel, C. C., '06.....Eccles bldg., Ogden, Utah OALR
Hetzel, Clarence Charles, Jr., '46
721 Eccles Bldg., Ogden, Utah ALR
Hewitt, Wright P., '44.51 Brattle st., Cambridge 38, Mass. ALR
Hewson, Wm., '31....2031 Locust st., Philadelphia 3, Pa. ALR
Hickey, Cornelius S., '46
39 Bay State Road, Boston 15, Mass. ALR
Hickey, Harold L., '30.....Republic bldg., Denver 2, Colo. ALR
Hickman, Warren R., '34...211 Third st., Logansport, Ind. ALR
Hicks, Vonnie M., '26....Professional bldg., Raleigh, N. C. OP
Hicks, William J., '30.....15 King, Middletown, N. Y. ALR
Hicks, William Wynne, '42
202 E. 6th st., Ellensburg, Wash. ALR
Higbee, D. R., '27....3245 Fourth av., San Diego 3, Calif. ALR

**Higgins, R. P., '24**.....20 Court, Cortland, N. Y. OALR
①**Higgins, S. G., '11**....411 E. Mason, Milwaukee 2, Wis. OALR
**High, Clifton E., '35**....Combs-Worley bldg., Pampa, Texas OP
**High, Howard Charles, '46**
324 E. Wisconsin av., Milwaukee 2, Wis. ALR
**High, Isaac B., '31**.....326 N. 5th, Reading, Pa. ALR
**Hilding, Anderson C., '31**
Medical Arts bldg., Duluth 2, Minn. OALR
**Hildreth, H. Rommel, '34**
Metropolitan bldg., St. Louis 3, Mo. OP
**Hilgartner, Henry L., Jr., '35**, 202 W. 13th St., Austin, Texas OP
**Hilger, Jerome Andrew, '46**
444 Lowry Medical Arts Bldg., St. Paul, Minn. ALR
②**Hill, C. E., '19**.....160 Bloor st. W., Toronto 5, Can. OP
**Hill, Emerson S., '37**.....51 Main, Torrington, Conn. ALR
**Hill, Frederick T., '28**.Professional bldg., Waterville, Maine ALR
**Hill, Howard E., '39**
402 W. Jackson st., POB 774, Muncie, Ind. ALR
**Hill, Howard F., '28**.....177 Main, Waterville, Maine OP
**Hilleman, George A., '40**
267 Alexander st., Rochester 7, N. Y. ALR
**Hillyer, Ernest C., '43**
1033 Gayley av., Los Angeles 24, Calif. OP
**Himelfarb, H. M., '41**..180 E. Queen st., Chambersburg, Pa. ALR
**Hinderer, Kenneth H., '39**
420 Medical Arts bldg., Pittsburgh 13, Pa. ALR
**Hipp, Walter, '45**.....140 E. 54th St., New York, N.Y. OP
**Hipskind, Myron Maxwell, '40**
6060 Drexel av., Chicago 37, Ill. ALR
**Hirschfelder, Max Kurt, '41**....408 W. 2nd, Centralia, Ill. OP
**Hirschmann, Hans A., '39**
1502 Mott av., Far Rockaway, N. Y. ALR
**Hirst, Otto C., '32**.....255 S. 17th, Philadelphia 3, Pa. ALR
**Hitschler, William J., '39**
5 E. Chestnut av., Philadelphia 18, Pa. ALR
**Hitz, John B., '34**....411 E. Mason st., Milwaukee 2, Wis. OP
**Ho, Wayne Y. H., '37**
Gotham Hotel, 975 Ingraham st., Los Angeles, 14, Calif. ALR
**Hobart, Carl, '29**..3601-A Gravois Ave., St. Louis 16, Mo. OP
**Hochfilzer, John J., '35**
Lowry Med. Arts bldg., St. Paul 2, Minn. ALR
**Hodge, George E., '30**....1414 Drummond, Montreal, Can. ALR
**Hodge, James V., '39**.....105 Market, Kingsport, Tenn. ALR
②**Hodsdon, B. F., '18**.....P.O. Box 923, Miami 6, Fla. OALR
**Hoeltgen, Maurice M., '43**...1607 W. 51st, Chicago 9, Ill. OP
**Hoffman, L. G., '27**....30 N. Michigan av., Chicago 2, Ill. OALR
**Hoffman, N. Fulmer, Jr., '43**
2126 Spruce st., Philadelphia 3, Pa. ALR
②**Hoffman, Walter F., '20**..1317 Marion, Seattle 4, Wash. OP
**Hoffman, Walter Lees, '39**
Medical Arts bldg., Minneapolis 2, Minn. OP
**Hoffman, William A., '32**
30 N. Michigan av., Chicago 2, Ill. OP
**Hoffmann, John N., '27**....405 Brant bldg., Canton 2, Ohio ALR
**Hofling, Charles A., '29**....19 W. 8th St., Cincinnati 2, Ohio OP
**Hofmann, Albert P., '36**..3149 Vine, Cincinnati 19, Ohio ALR
**Hofmann, W. P., '25**.....Kahl bldg., Davenport, Iowa OALR
**Hogan, Chester F., '41**.....62 Main st., Houlton, Maine ALR
**Hogan, Michael John, '46**
450 Sutter St., San Francisco 8, Calif. OP
**Hogan, W. L., '24**.....750 Main, Hartford 3, Conn. OP
**Hogsett, Smith Fuller, '42**
1581 Paulsen Medical Bldg., Spokane, Wash. OP
①**Hogshead, J. M., '12**
602 Georgia av., Chattanooga 3, Tenn. OALR
①**Hogue, D. W., '08**.....34 W. High, Springfield, Ohio OALR
①**Hohf, Julius A., '15**..... Clinic bldg., Yankton, S. D. OALR
**Hoitash, Frederick J., '44**
P. O. Box 1066, Huntington 13, W. Va. OP
**Holcombe, Virgil Eugene, '40**
Medical Arts bldg., Charleston 1, W. Va. ALR
**Holden, Harold M., '36**
3875 Wilshire blvd., Los Angeles 5, Calif. ALR
**Holden, Wm. H., '39**.....755 Park av., New York, N.Y. ALR
**Holding, Bruce F., '36**.....Bell bldg., Montgomery 4, Ala. ALR
**Holinger, Paul H., '37**..700 N. Michigan, Chicago 11, Ill. L
**Hollabaugh, Charles F., '38**,
414 Doctors bldg., Nashville 3, Tenn. OP
**Holland, Emory E., '33**..1907 E. Main st., Richmond, Ind. OP
**Holland, John Gill, '41**.....725 Church, Lynchburg, Va. ALR
**Hollender, Abraham R., '31**,
605 Lincoln rd., Miami Beach, Fla. ALR
**Holloway, J. Minor, '32**
1200 Prince Edward, Fredericksburg, Va. ALR
**Hollowell, Frederick Dixon, '40**
705 Lamar Life bldg., Jackson, Miss. ALR
**Holmen, Robert W., '43**
Lowry Medical Arts bldg., St. Paul 2, Minn. ALR
**Holmes, Dorothy Behner, '46**
1816 R. Street N.W., Washington 9, D.C. OP
**Holmes, Edgar M., '36**..330 Dartmouth, Boston 16, Mass. ALR
**Holmes, Will W., '35**.....R. D. No. 4, Logansport, Ind. ALR
**Holohan, Joseph L., '37**.....330 State, Albany 6, N.Y. OALR
**Holstein, Theodore, '42**
1800 Sutterville rd., Sacramento, Calif. OP
**Holt, Helen, '43**.....720 N. Michigan av., Chicago 11, Ill. OP
**Holzberger, John C., '32**
7005 Freshpond, Brooklyn 27, N. Y. ALR
**Holzer, Wm. F., '31**.....36 Pleasant, Worcester 8, Mass. OP
**Hombach, Frank Joseph, '38**
1421 State, Santa Barbara, Calif. OP
①**Hompes, J. J., '15**.....Sharp bldg., Lincoln 8, Nebr. OALR
**Honiss, John R., '37**....277 Alexander, Rochester 7, N. Y. ALR
**Hooker, James F., '39** Capt. (MC) USN
U. S. Naval Hospital, Memphis 15, Tenn. ALR
**Hooker, Lyle, '45**....416 Esperson bldg., Houston 2, Tex. OP
**Hoople, Gordon D., '29**..Medical Arts bldg., Syracuse, N.Y. ALR
**Hooss, C. H. A., '30**
2333 S. Grand blvd., St. Louis 4, Mo. OP
**Hooval, J. H., '41**.....213 Fallis bldg., Ontario, Calif. ALR
**Hoover, Walter B., '34**
605 Commonwealth, Boston 15, Mass. ALR
**Hope, Robert M., '37**.108 Rutledge av., Charleston 16, S.C. ALR
**Hopkins, G. H., '30**.....702 N. Main, Pueblo, Colo. OP
**Hopp, Eugene S., '44**
2000 Van Ness Ave., San Francisco 9, Calif. ALR
**Hornback, George A., '35**....500 Broadway, Hannibal, Mo. ALR
**Hornbogen, Daniel Powell, '41**
401 Savings Bank bldg., Marquette, Mich. OP
**Horner, Warren D., '35**, 490 Post st., San Francisco 2, Calif. OP
**Horsfield, Russell Thomas, '45**
715 Medical and Dental bldg., Seattle 1, Wash. OP
**Hory, Joseph S., '39**
2488 Grand Concourse, New York 58, N. Y. OP
**Hotchkiss, Walter T., '31**
541 Lincoln rd., Miami Beach 39, Fla. ALR
**Houlahan, James M., '36**, 117 S. 2nd av., Mt. Vernon, N. Y.
and 653 Park av., New York 21, N. Y. OP
**House, Howard Payne, '41**,
1136 W. 6th st., Los Angeles 14, Calif. ALR

House, Hugh O., '38
1835 Eye st. N.W., Washington 6, D. C. ALR
House, Leland Richmond, '40
1727 New Jersey st., Los Angeles 33, Calif. ALR
Houser, Ben P., '42.....458 E. Broad St., Tamaqua, Pa. ALR
Houser, Karl M., '34...2035 Delancey, Philadelphia 3, Pa. ALR
Howard, Harvey J., '28. Park Plaza Hotel, St. Louis 8, Mo. OP
Howard, Henry Sargent, '43
940 Jefferson bldg., Peoria 2, Ill. ALR
①Howard, J. W., '12, (Retired),
6026 McGee, Kansas City 2, Mo. OALR
Howard, John C., Jr., '42,
1408 Professional bldg., Kansas City, Mo. ALR
Howard, Lloyd G., '23....Park bldg., Council Bluffs, Iowa OALR
Howard, Robert E., '31
Union Central bldg., Cincinnati 2, Ohio ALR
Howard, W. H., '23.....Oelwein, Ia. OALR
Howard, William H., '37
9152 Commercial av., Chicago 17, Ill. OP
Howard, William M., '38. 389 Linwood av., Buffalo 9, N. Y. OP
Howarth, Ervin McIntyre, '40
705 Main st., Santa Paula, Calif. ALR
②Howe, A. W., '20..Medical Arts bldg., Tacoma 2, Wash. OALR
Howell, Donald M., '31, Wiechmann bldg., Saginaw, Mich. ALR
Howell, Homer P., '42.....2709 Alida st., Oakland, Calif. OP
Howell, Stacy C., '39
478 Peachtree st. N.E., Atlanta 3, Ga. OP
Howland, Alvin W., '39
255 So. 17th st., Philadelphia 3, Pa. OP
Howley, Bartley Mawell, '45
67 Livingston Ave., New Brunswick 2, N.J. ALR
Hoxie, Derrick, '43, Comdr. (MC) USN
U. S. Naval Hospital, Portsmouth, Va. ALR
Hubbard, Wm. B., '31..302 Paterson bldg., Flint 3, Mich. OALR
Hubble, William Fields, '42. Citizens bldg., Decatur 30, Ill. ALR
Huber, Walter A., '32...Medical Arts bldg., Tulsa 3, Okla. OP
Hudock, John P., '41....208 Jefferson Ave., Endicott, N.Y. ALR
Huffman, William Clyde, '46
University Hospitals, Iowa City, Ia. ALR
Huggins, Harrison D., '44
12 Whipps bldg., Kalispell, Mont. ALR
Hughes, L. W., '25.....965 Broad, Newark 7, N. J. OP
Hughes, Ray W., '28
303 N. Lakeside Drive, Lake Worth, Fla. OALR
②Hughes, T. E., '21....1000 W. Grace, Richmond 20, Va. ALR
①Hughes, W. F., '11
401 Hume-Mansur bldg., Indianapolis 4, Ind. OP
Hughes, Wendell L., '29
Professional bldg., Hempstead, N. Y. OP-PI S
Hughes, William Franklin, '43
401 Hume-Mansur Bldg., Indianapolis 4, Ind. OP
Hughes, William Lauch, '40..Lamar bldg., Jackson 2, Miss. ALR
Hulka, J. H., '34, 30-28 36 st., Long Island City 3, N. Y. OALR
Hull, Allen, '35.....401 76th, Brooklyn 9, N. Y. OP
Hunnicutt, Leland G., '38
275 S. Holliston av., Pasadena 5, Calif. ALR
Hunt, Guy A., '36.....R.D. 5, Butler, Pa. OP
Hunt, Thomas Ewell, '39
First Nat'l Bank bldg., Paris, Texas ALR
Hunt, Westley M., '29..907 Fifth av., New York 21, N. Y. ALR
Hunt, Wm. Remfry, '38
Southern Permanente Hospital, Fontana, Calif. ALR
Hunt, Wm. T., Jr., '34..367 Brookway, Merion Station, Pa. OALR
Hunter, J. F., '30.....503 Penn av., Turtle Creek, Pa. ALR
Hunter, Robert John, '30,
2035 Delancey st., Philadelphia 3, Pa. ALR
Hurd, Allan C., '40.....62 Church St., Gardiner, Maine ALR
Hurd, Fritz D., '39. Medical Arts bldg., Great Falls, Mont. ALR
②Hurff, J. Wallace, '19.....671 Broad, Newark 2, N. J. OALR
②Hurst, V. Reeves, '22.....Hurst Clinic, Longview, Texas OALR
Hurwitz, Paul, '43, Suite 1532 Field Annex bldg.,
25 E. Washington st., Chicago 2, Ill. OP
②Husik, D. N., '21.....1820 Spruce, Philadelphia 3, Pa. ALR
Huston, Marshall D., '44..302½ Main st., Cedar Falls, Ia. ALR
Hutcherson, John Kenneth, '41
Francis bldg., Louisville 2, Ky. OP
Hutchinson, Abbott T., '28
4086 Reynolds av., Coconut Grove, Fla. ALR
Hutchinson, Ben. B., '38
Lubbock Memorial Hospital, Lubbock, Texas OP
Hutchison, K. O., '30
1509 Sherbrooke st. W., Montreal, Can. ALR
Hutchison, Lloyd S., '42..45 E. Orange st., Lancaster, Pa. ALR
Hyman, Morris, '36..305 Doctors ldg., Cincinnati 2, Ohio ALR
Hymes, Charles, '39
Medical Arts bldg., Minneapolis 2, Minn. OP
Hynes, Frank Joseph, '41. 121 E. 60th, New York 22, N. Y. ALR

## I

Ide, David, '39. 1749 Grand Concourse, New York 53, N. Y. ALR
①Imperatori, C. J., '11. 108 E. 38th, New York 16, N. Y. ALR
Inciardi, James A., '46..149 Midwood st., Brooklyn 25, N.Y. OP
Ingalls, Raymond G., '41, Institute of Oph., Presbyterian Hosp.,
635 W. 165th st., New York, N.Y. OP
①Inglis, Harry J., '14. 43 Bay State rd., Boston 15, Mass. ALR
Ingram, Hollis Carlyle, '40, 303 Exchange bldg., Orlando, Fla. OP
Insel, Herman Herbert, '42
770 Fidelity Medical Bldg., Dayton 2, Ohio OALR
Ireland, Percy E., '36
330 Medical Arts bldg., Toronto, Canada ALR
Irgens, Edwin R., '38
Wills Hospital, 1601 Spring Garden St., Philadelphia 30, Pa. ALR
②Irvine, A. Ray, '18, 9730 Wilshire blvd., Beverly Hills, Cal. OP
Irvine, Robert S., '29...490 Post, San Francisco 2, Calif. OALR
Irvine, S. Rodman, '38
9730 Wilshire blvd., Beverly Hills, Calif. OP
Irwin, John Joseph, '42
Baronne bldg., New Orleans 12, La. ALR
Irwin, Thomas M., '42
Medical Arts bldg., Park st., Jacksonville, Fla. ALR
②Irwin, Vincent J. Jr., '20
1634 Main, Springfield 3, Mass. OALR
Irwin, Willard J., '42
Medical Arts Bldg., Knoxville, Tenn. ALR
Isbell, D., '40.....552 McCallie av., Chattanooga 3, Tenn. ALR
Isbell, Euclid A., '43.....948 Forrest av., Gadsden, Ala. ALR
Ivins, William Clifford, '40. 455 W. State, Trenton 8, N. J. ALR
Ivy, Robert H., '34. 1930 Chestnut, Philadelphia 3, Pa. ALR-PI S

## J

Jackson, Atras E., '28
Medical Arts bldg., Ft. Worth 2, Texas OP
①Jackson, Chevalier, '04..255 S. 17th, Philadelphia 3, Pa. L
Jackson, Chevalier L., '34. 255 S. 17th, Philadelphia 3, Pa. L
Jackson, Clarence C. R., '37
2065 Adelbert Road, Cleveland 6, Ohio ALR
②Jackson, J. D., '19.....232 W. 8th, Erie, Pa. OALR
Jacobs, Clyde Hillock, '41
Geisinger Memorial Hospital, Danville, Pa. OP

Jacobs, Milton, '33....122 S. Michigan av., Chicago 3, Ill. OP
①Jacobs, S. M., '13.....15 W. 81st, New York 24, N. Y. OALR
Jacoby, Mark W., '33.....Hanna bldg., Cleveland 15, Ohio OP
Jaeckle, Charles Eisele, '45
136 Evergreen Place, East Orange, N.J. OP
Jaffe, Benjamin, '39....566 Bergen av., Jersey City 4, N.J. OP
Jaffe, Morris, '42
940 Grand Concourse, New York 56, N. Y. OP
James, George R., '37
Wyoming Nat'l Bank bldg., Casper, Wyo. OP
James, James E., '44
128 W. Fourth st., Bethlehem, Pa. ALR
James, Voyle, '40
6253 Hollywood blvd., Hollywood 28,Calif. ALR
James, Wm. M., '31...508 N. Grand av., St. Louis 3, Mo. OP
Jamison, Edgar L., '41.....19 Louisa av., Greenville, Pa. ALR
Janse, Hector M., '37.....3717 Main, Houston 4, Texas ALR
Jarrott, James T., '43...131 Fulton av., Hempstead, N. Y. OP
Jarvis, DeForest C., '29....Quarry Bank bldg., Barre, Vt. ALR
①Jean, George W., '22 1415 Laguna st., Santa Barbara, Cal. OP
Jellis, Walter, '41...71 College av., W. Somerville 44, Mass. ALR
Jenkins, Albert H., '39..2749 W. Arthur av., Chicago, Ill. ALR
Jennings, Charles W., '31
121 University pl., Pittsburgh 13, Pa. OP
Jensen, August Frithiof, '40
1119 Cottonwood st., Grand Forks, N. D. OP
Jensen, Carl Dana, '39,
1315 Medical & Dental Bldg., Seattle 1, Wash. OP
Jerome, Bourne, '43
1750 Medical Arts bldg., Minneapolis, Minn. OP
Jervey, James W., Jr., '45..101 Church St., Greenville, S.C. OALR
Jesberg, Simon, '32
500 S. Lucas av., Los Angeles 13, Calif. ALR
Jessel, Erwin, '42.....1160 Fifth Av., New York 29, N.Y. OP
Johns, Juanita Pearl, '33
313 Commonwealth av., Boston 15, Mass. OP
Johnson, Carl Cordes, '45
5 Bay State Rd., Boston 15, Mass. OP
Johnson, Charles I., '39
403 Commonwealth av., Boston, Mass. ALR
Johnson, David M., '32, 327 E. State St., Columbus, Ohio ALR
Johnson, Don, '39.1835 Eye st. N.W., Washington 6, D. C. ALR
Johnson, Ernest D., '38....317 W. Main, Alhambra, Calif. OALR
Johnson, Fordyce A. H., '44
98 No. Madison, Pasadena 4, Calif. ALR
Johnson, Gayle T., '39, Major (M.C.) 0-482051
Address Unknown ALR
①Johnson, H. R., '13.....320 Jefferson, Fairmont, W. Va. OALR
Johnson, Henrietta M., '32
595 E. Colorado, Pasadena 1, Calif. OP
Johnson, Henry P., '35.....32 Deering, Portland 3, Maine ALR
Johnson, Hobart Clemens, '45
839 Belgrade Av., Mankato, Minn. OP
Johnson, Leighton F., '37
29 Bay State road, Boston 15, Mass. ALR
Johnson, Lorand V., '38
10515 Carnegie, Cleveland 6, Ohio OP
Johnson, Malcolm R., '41
645 Medical Arts bldg., Minneapolis 2, Minn. ALR
Johnson, Roger Harry, '45..522 Cobb Bldg., Seattle, Wash. OP
Johnson, Thomas H., '23..30 W. 59th, New York 19, N. Y. OP
Johnson, Thomas W., '46
529, 39 East Ohio, Indianapolis 4, Indiana ALR
Johnston, Hewitt, '36...303 Exchange bldg., Orlando, Fla. ALR
Johnston, James Gilliam, '39
1520 E. Fourth, Charlotte 4, N. C. OP
Johnston, Kenneth B., '32
1509 Sherbrooke st. W., Montreal, Can. OP
①Johnston, W. H., '12..1515 State, Santa Barbara, Calif. ALR
Johnston, William Raby, '42
1515 State st., Santa Barbara, Calif. ALR
Joinville, Euclide V., '38...Stroh bldg., Detroit 26, Mich. ALR
Jones, Arthur C., '44....518 Eastman bldg., Boise, Idaho ALR
Jones, Cecil Cain, '28.Equitable bldg., Des Moines 9, Iowa OALR
Jones, David E., '38
Chamber of Commerce, Indianapolis 4, Ind. ALR
Jones, David H., '35.....140 E. 54th, New York 22, N. Y. LR
Jones, Edley H., '28...1301 Washington, Vicksburg, Miss. OALR
Jones, Edmund L., '26..Wheeling Clinic, Wheeling, W. Va. OALR
Jones, Edward Earl, '40, San Diego Co. Gen. Hosp.
North End of Front St., San Diego 3, Calif. ALR
①Jones, Elgin W., '13..29 Lafayette St., Wakefield, Mass. OP A
Jones, Emmett L., Jr., '44
15 S. Liberty st., Cumberland, Md. ALR
②Jones, F. W., '19.....101 E. Broadway, Alton, Ill. OALR
Jones, Florentine B., '39.....416 S. 17th, Mattoon, Ill. ALR
Jones, Fred G., '36.....258 Genesee, Utica 2, N. Y. ALR
Jones, John Allen, '46
401 S. Court St., Montgomery, Ala. ALR
Jones, Leonard W., '25.16 N. Goodman, Rochester 7, N. Y. OALR
Jones, Lester T., '37...1020 S. W. Taylor, Portland 5, Ore. OALR
Jones, M. F., '30.....121 E. 60th, New York 22, N. Y. ALR
Jones, U. G., '27....Market & Boone, Johnson City, Tenn. ALR
Jones, Vincent L., '28
Missouri Theatre bldg., St. Louis 3, Mo. OALR
Jones, W. Yerby, '43.....436 Sycamore, Buffalo 4, N. Y. OP
Jones William M., '44....5254 Michigan av., Chicago, Ill. OP
Jones, William S., '27
521 Sheridan road, Menominee, Mich. OALR
②Jordan, George T., '21..215 Court st., Vermillion, S. D. OALR
Jordan, Harvey Ernest, Jr., '46
1912 Spruce St., Philadelphia 3, Pa. ALR
Jordan, James Shannon, '40
Medical Arts bldg., Scranton 3, Pa. OP
Jordan, Lewis W., '41.Medical Arts bldg., Portland 5, Ore. ALR
Jordan, Philip J., '43
910 Medico-Dental bldg., San Jose 20, Calif. ALR
Jordan, Raymond Ellsworth, '46
121 University Place, Pittsburgh, Pa. ALR
Joseph, Nime K., '44.....40-14th st., Wheeling, W. Va. ALR
Joy, Harold H., '26..State Tower bldg., Syracuse 2, N. Y. OP
Joyce, Wm. M., '36.....121 Main st., Middletown, Conn. ALR
Joynt, A. J., '24.....525 Sycamore, Waterloo, Iowa OALR
Joynt, M. J., '30.....Royal bldg., Le Mars, Iowa ALR
Judd, Delbert K., '35....420 Arcade bldg., Kankakee, Ill. ALR
①Judd, H. B., '17.....40 E. 61st, New York 21, N.Y. ALR
Judd, J. H., '30.....Medical Arts bldg., Omaha 2, Nebr. OP
②Judge, Harry V., '20.....46 Willett, Albany 6, N.Y. OP
Judge, Thomas A., '37...735 N. Water, Milwaukee 2, Wis. ALR
Juers, Arthur L., '37..55 E. Washington st., Chicago 2, Ill. ALR

## K

Kafka, M. Martyn, '40
109-05 72nd av., Forest Hills, L. I., N. Y. ALR
Kahn, Kenneth Myer, '40
204 Pape bldg., Staten Island, N.Y. ALR
Kaiser, Robert F., '44..1705 Knox Ave., Bellingham, Wash. ALR
Kallen, Leo A., '46..667 Madison Av., New York 21, N.Y. ALR
Kammerling, Theo. S., '32.616 N. Central, Chicago 44, Ill. OALR

**Kane, J. T., '37.88-49—163rd st., Jamaica 3, L. I., N. Y. ALR**
**Kaplan, Carl, '37....69-17 Forest av., Brooklyn 27, N. Y. ALR**
**Kaplan, Leon, '42..881 Lafayette st., Bridgeport 4, Conn. OP**
Kaplan, Samuel, '35, Maj. (MC).....Address Unknown ALR
**Kapp, Carl G., '35.440 Westminster av., Elizabeth 3, N. J. ALR**
**Kapp, Henry, '36.....50 Merrimack, Haverhill, Mass. ALR**
**Karkeet, Robert B., '28.833 S.W. 11th av., Portland 5, Ore. ALR**
Kasnetz, Joseph P., '39
678 Eastern Parkway, Brooklyn 13, N. Y. ALR
Kasper, Kelvin Anthony, '38
135 S. 18th, Philadelphia 3, Pa. ALR
**Katz, Dewey, '37.....99 Pratt, Hartford 3, Conn. OP**
Katz, Harry Kolman, '42
1206 Cottman st., Philadelphia 11, Pa. OP
Katz, I. H., '41.....713 E. Genesee, Syracuse, N. Y. OP
Katzin, Herbert Maurice, '45
1148 Fifth Ave., New York 28, N.Y. OP
②Kauffman, A. B., '22.....Chimes bldg., Syracuse, N.Y. ALR
Kauffman, M. Luther, '38
Medical Arts bldg., Jenkintown, Pa. OP
Kaufman, Aaron, '46.....483 Beacon St., Boston 7, Mass. ALR
①Kaufman, A. Spencer, '17
255 So. 17th St., Philadelphia 3, Pa. ALR
**Kaufman, Maxwell, '40.....85 Maujer, Brooklyn 6, N. Y. ALR**
**Kaufman, Samuel I., '41.185 N. Wabash av., Chicago 1, Ill. OP**
**Kayser, Reuben, '39.**
442 Bay Ridge Parkway, 75th st., Brooklyn 9, N. Y. ALR
Kazanjian, V. H., '33
475 Commonwealth av., Boston 15, Mass. LR-PI S
**Kearney, Harold L., '37**
1403 Delachaise, New Orleans 15, La. ALR
Kearny, R. A., '32
2311 Connecticut av. N.W., Washington 8, D. C. ALR
**Keating, T. F., '27.....Maccabee bldg., Detroit 2, Mich. OALR**
**Keefe, William J., '29**
205 E. Sidney av., Mt. Vernon, N. Y. OALR
**Keeler, Lansing H., '34..89-04—148th, Jamaica 9, N. Y. OALR**
Keeler, Nelson S., '39....411 Thirtieth, Oakland 9, Calif. ALR
Keil, Frank C., '39..660 Madison av., New York 21, N. Y. OALR
Keil, Peter A., '36.118 Pennsylvania av., Brooklyn 7, N. Y. ALR
Keim, William Franklin, Jr., '46
15 Washington St., Newark 2, N.J. ALR
Keiper, G. F., Jr., '30.....113 N. Church, Visalia, Calif. ALR
①Keith, W. E., '16.Professional bldg., Kansas City 6, Mo. OALR
②Keller, S. A., '20,
303 S. Minnesota av., Sioux Falls, S. D. OALR
Kelley, Isaac D., Jr., '28..3903 Park Ave., St. Louis 10, Mo. ALR
Kelley, Samuel F., '44....47 E. 61st st., New York, N.Y. ALR
Kelley, Vincent J., '33
520 Commonwealth av., Boston 15, Mass. ALR
Kelly, Francis B., '45
521 Medical Professional bldg., Corpus Christi, Tex. OP
**Kelly, Geo. F. J., '34.....37 S. 20th, Philadelphia 3, Pa. OP**
Kelly, Joseph D., '28
140 E. 54th, New York 22, N. Y. OALR-PI S
Kelly, Vincent J., '44....310 Arcade bldg., Kankakee, Ill. OALR
②Kemler, Joseph I., '22, 1908 Eutaw pl., Baltimore 17, Md. OALR
**Kemp, Roy C., '38.....343 State, Albany 6, N. Y. ALR**
**Kend, Lee, '32.....133 E. 58th, New York 22, N. Y. ALR**
**Kennedy, Eldred W., '28**
Gas & Electric bldg., Rochester 4, N. Y. OP
**Kennedy, Francis L., '43**
Missouri Theatre bldg., St. Louis 3, Mo. ALR
**Kennedy, Patrick James, '40**
6910 Market st., Upper Darby, Pa. OP
Kennedy, Roscoe J., '39
2020 E. 93d, c/o Cleveland Clinic, Cleveland 6, Ohio OP
**Kennedy, Wm. A., '36....372 St. Peter, St. Paul 2, Minn. OP**
**Kennon, Beverley Randolph III '45**
908 Medical Arts Bldg., Norfolk 10, Va. OP
Kenyon, Byron B. '42...44 Pondfield rd., Bronxville, N. Y. ALR
Kerns, Thomas C., '41....1110 West Main, Durham, N. C. ALR
Kershner, C. M., '43....Watson Clinic, Brookings, S. Dak. OP
Kershner, W. E., '34.....117 Front St., Bath, Maine OALR
Kerst, John Arthur, '42..1444 Lake Shore Dr., Chicago, Ill. OP
Kerwin, Raymond W., '38
310 S. Michigan av., Chicago 4, Ill. OALR
Keskey, George I., '44.....Rose bldg., Marquette, Mich. ALR
**Kessler, Mana, '38.....311 Center av., Bay City, Mich. ALR**
Kettelkamp, Fred O., '26
Ferguson bldg., Colorado Springs, Colo. ALR
Key, Samuel N., '26
505 Capital Nat'l Bank bldg., Austin 16, Tex. OALR
Key, Samuel Newton, Jr., '46
120 W. 7th St., Austin 16,Tex. OP
Keyes, John E. L., '33
22199 McCauley Rd., Shaker Heights 22, Cleveland, Ohio OP
**Keyton, John A., '34.....304 N. Oates, Dothan, Ala. ALR**
①Kiehle, Frederick A., '15
1020 Southwest Taylor, Portland 5, Ore. OP
**Kieler, Charles B., '44..M. I. B. Hosp., Cooperstown, N.Y. ALR**
**Kilbourne, P. H., '24**
Fidelity Medical bldg., Dayton 2, Ohio OALR
Kilgore, George L., '35
411 Thorn St., San Diego 3, Calif. OP
Kilmann, Martin H., '45
118 East 54th St., New York 22,N.Y. OP
Kimata, Harold T., '43..165 Elk Place, New Orleans 13, La. ALR
Kimbrig, Max, '40.....20 High st., Huntington, N. Y. ALR
①Kincaid, J. H., '03
421 West Church av., Knoxville 42, Tenn. OALR
Kincannon, LeRoy T., '28,
2300 Highland Ave., Birmingham 5, Ala. OALR
King, Charles M., Jr., '44
899 Madison Ave., Suite 116, Memphis, Tenn. OP
②King, Charles R., '20.....Spitzer bldg., Toledo 4, Ohio OALR
King, Claire B., '37....409 3rd St. N.W., Canton 2, Ohio OP
②King, Edward, '21, 6777 Hollywood Blvd.,
Hollywood 28, Calif. ALR
King, George L., '29.....537 E. Market St., Alliance, Ohio OP
King, James Thomas, '46
803 Medical Arts bldg., Atlanta 3, Ga. ALR
**King, Lowell W., '33.....470 E. State, Salem, Ohio ALR**
①King, O. H., '16, Medical Arts Bldg., Hot Springs, Ark. OALR
King, Raymond H., '44....3690 Hershel, Jacksonville, Fla. ALR
**King, Walter F., '38...519 Delaware av., Buffalo 2, N. Y. OP**
Kingston, Louis C., '44.....91 Tremont St., Barre, Vt. ALR
Kinkade, Joseph M., '38..45 E. Jackson st., Tucson, Ariz. ALR
**Kinney, Charles Ed., '34**
10515 Carnegie av., Cleveland 6, Ohio ALR
**Kirby, D. B., '24....780 Park av., New York 21, N. Y. OP-PI S**
Kirch, Walter, '43
1023 Bankers Trust bldg., Des Moines 9, Ia. ALR
Kirkbright, James C., '35..40 E. 61st, New York 21, N. Y. ALR
Kirkpatrick, Hazen L., '37....715 Mills Bldg., Topeka, Kan. ALR
③Kirkpatrick, Henry, '24, Lt. Col.....Address Unknown
②Kistner, F. B., '18..1216 S.W. Yamhill St., Portland, Ore. ALR
Klaff, Daniel David, '46
3720 Washington blvd., St. Louis 8, Mo. ALR
Klawans, Harold L., '43..20 E. Jackson blvd., Chicago 4, Ill. ALR

Kleiman, Bernard S., '44, Capt. (M.C.)
2517 Linden Av., Baltimore 17, Md. ALR
Kleinert, Margaret N., '29
23 Bay State Road, Boston 15, Mass. ALR
Kleinfeld, Louis, '36..100 E. 74th st., New York 21, N.Y. ALR
Kler, Joseph H., '37
151 Livingston av., New Brunswick, N. J. ALR
Klien, Bertha A., '38
58 E. Washington, Sec. 2, Chicago 2, Ill. OP
①Kline, H. G., '12...State Tower bldg., Syracuse 2, N. Y. ALR
Kline, O. R., '30.....514 Cooper, Camden, N. J. ALR
②Klingner, Thomas O., '20
951 So. Pickwick, Springfield, Mo. OALR
Klotz, Wm. F., '38.....1879 Poplar, Memphis, Tenn. ALR
Kluever, Herman C., '36....221 No. 18th St., Ft. Dodge, Ia. OP
①Knapp, Arnold, '08.....10 E. 54th, New York 22, N. Y.OP
Knapp, Arthur Alexander, '33
112 E. 73rd St., New York, N. Y. OP
Knapp, F. N., '25....Medical Arts bldg., Duluth 2, Minn. OP
Knauer, Wm. J., '30...Buckman bldg., Jacksonville 2, Fla. OALR
Knight, Granville F., '37
Medical Centre bldg., White Plains, N. Y. ALR
Knight, John S., '32..5800 High Drive, Kansas City, Mo. ALR
Knighton, Willis S., '31..121 E. 61st, New York 21, N. Y. OP
Knisely, Alan DeG., '38..722 Nat'l Bank bldg., Lima, Ohio ALR
Knopf, Saul, '37.....30 E. 40th, New York 16, N. Y. ALR
Knowles, W. Mood, '39....4227 Herschell, Dallas 4, Tex. ALR
Knowlton, Don S., '39
408 Columbia Medical bldg., Washington 6, D.C. ALR
Knudsen, Arnold F., '44
419 Bay Ridge Parkway, Brooklyn 9, N.Y. ALR
Kobrak, Heinrich, '38
University of Chicago, 950 E. 59th St., Chicago 37, Ill. ALR
Koch, Ferdinand L. P., '40,
60 East 75th st., New York 21, N.Y. OP
Koch, Harold J., '43..6235 S. Kedzie av., Chicago 29, Ill. OP
Koebbe, E. E., '29.....Columbus, Nebr. ALR
Koenig, Ivan J., '28.....40 W. North st., Buffalo 2, N. Y. OP
Koff, Raphael Joseph, '46
1917 Wilshire blvd., Los Angeles 5, Calif. OP
Kogut, Lewis V., '41
5466 Broadway Ave., Cleveland 4, Ohio OP
Koke, Martin P., '44.....233 A. st., San Diego, Calif. OP
Koller, H. M., '36.Medical Arts bldg., Minneapolis 2, Minn. OP
Koller, Louis R., '32
Medical Arts bldg., Minneapolis 2, Minn. ALR
①Kopetzky, Samuel J., '08, Col. (M.C.),
71 East 80th, New York 21, N. Y. ALR
Kopp, Mortimer M., '42.79 Argyle rd., Brooklyn 18, N. Y. ALR
Kornberg, Maurice, '38
235 E. 22nd st., New York 10, N.Y. ALR
Kornblut, Alfred, '46
1539 Fairfield Ave., Bridgeport 5, Conn. ALR
Kornzweig, Abraham L., '37
1200 Fifth av., New York 29, N. Y. OP
Kos, Clair M., '43.....534 29th, Des Moines, Ia. ALR
①Kraft, Oscar H., '12
1518 N. Dearborn parkway, Chicago 10, Ill. OP
Krall, J. Thomas, '42..1927 Spruce St., Philadelphia 3, Pa. OP
Kramer, Julius C., '34...409 Columbus av., Sandusky, Ohio ALR
Krause, Arlington C., '37...950 E. 59th, Chicago 37, Ill. OP
Kravitz, Daniel, '40...861 Park place, Brooklyn 16, N. Y. OP
Kreag, Karl A., '39....167 Joralemon, Brooklyn 2, N. Y. ALR
①Krebs, Adolph, '13.....500 Penn av., Pittsburgh 22, Pa. OP
Kremen, Abraham, '41
2355 Eutaw Place, Baltimore 17, Md. OP
②Kress, George H., '18
131 S. Rampart blvd., Los Angeles 4, Calif. OALR
Krewson, William Egbert, 3rd, '40
1930 Chestnut st., Philadelphia 3, Pa. OP
Krieger, Alexander A., '43
4101 Jenkins Arcade, Pittsburgh 22, Pa. OP
①Krieger, Geo. M., '10,
701 Washington, Michigan City, Ind. OALR
Krimsky, Emanuel, '32...745 Eastern Pkwy., Brooklyn, N.Y. OP
Krimsky, Joseph, '28
2924 Winters Rd., Huntington, W. Va. OALR
Krone, William Frank, '42
31 Lincoln Park, Newark 2, N. J. ALR
Kronenberg, Bernard, '40
737 Park av., New York 21, N.Y. OP
Kronfeld, Peter C., '31.....904 W. Adams, Chicago 7, Ill. OP
①Krug, Ernest F., '08..988 Fifth av., New York 21, N. Y. OP
Kruskal, I. D., '25...101 Lafayette av., Brooklyn 17, N. Y. OP
Krystaf, Joseph G., '37
1004 Fillmore av., Buffalo 11, N. Y. ALR
Kuhn, Clifford W., '45
9545 S.W. 62nd Dr., Portland, Ore. ALR
Kuhn, H. S., '30.....112 Rimbach, Hammond, Ind. OP
Kuhn, Hugh A., '28.....112 Rimbach, Hammond, Ind OALR
Kulesher, Henry T., '46
3825 Pershing Drive, Arlington, Virginia ALR
②Kully, B. M., '22
3875 Wilshire blvd., Los Angeles 5, Calif. ALR
Kully, Herman E., '35.Medical Arts bldg., Omaha 2, Nebr. ALR
Kulvin, Max M., '31, 5110 Kenwood av., Chicago 15, Ill. OALR
Kundert, Fred W., '38...The Monroe Clinic, Monroe, Wis. ALR
Kuntz, Herman William, '45
501 Hume Mansur bldg., Indianapolis 4, Ind. ALR
Kupp, John H., '44.....582 Delaware av., Palmerton, Pa. ALR
Kuritzky, Isadore, '40
1480 Nostrand av., Brooklyn 26, N. Y. ALR
Kutscher, Charles F., '44
520 Medical Arts bldg., Pittsburgh 13, Pa. OP

## L

Lachman, George S., '37
450 Sutter St., San Francisco, Calif. OP
LaClair, Charles Huston, Jr., '40
1021 W. Main St., Norristown, Pa. ALR
Laff, H. I., '30.....Metropolitan bldg., Denver 2, Colo. ALR
①La Force, E. F., '08
Iowa State Bank bldg., Burlington, Iowa OALR
LaGattuta, Frank, '40
2488 Grand Concourse, Bronx 58, N. Y. OP
Laird, Archibald, '39.....12 Main, Wellsboro, Pa. OP
Laird, Robert G., '32.114 E. Fulton, Grand Rapids 2, Mich. OP
Lamb, Andrew J., '46..10006 Carnegie Ave., Cleveland, Ohio ALR
Lamb, Harry H., '29.....220 Main, Davenport, Iowa ALR
①Lamb, R. S., '06, Stoneleigh court, Washington 6, D. C. OP
Lamberson, Frank Abrom, '42
19600 Grand River, Detroit 23, Mich. ALR
Lamberson, Wm. H., '37
1st Nat'l Bank bldg., Colorado Springs, Colo. ALR
LaMotte, Wm. O., '35
Medical Arts bldg., Wilmington 9, Del. OALR
Lampert, Max T., '31.....First Nat'l bldg., Minot, N.D. ALR
①Lancaster, W. B., '13
520 Commonwealth av., Boston 15, Mass. OP
Lance, Verne L., '44....1113 Quarrier, Charleston, W. Va. ALR

**Landegger, George P., '40,**
860 Roosevelt Bldg., 727 W. 7th St., Los Angeles 14, Calif. OP
**Landis, J. E., '30.....232 N. Sixth, Reading, Pa. ALR**
Lane, Franklin F., '33, Capt. (MC) USN
7430 Boyer St., Mt. Airy, Philadelphia, Pa. OALR
**Lang, George H., '32.....202 E. Liberty, Savannah, Ga. ALR**
**Lange, Adolph C., '37.....3903 Olive, St. Louis 8, Mo. OP**
Langworthy, Henry G., '44..394 W. 10th st., Dubuque, Ia. ALR
Lanson, F. Arthur, '44
646 Washington Rd., Pittsburgh 16, Pa. ALR
Lapointe, Jean, '39....300 Carre St. Louis, Montreal, Can. ALR
**LaReau, Hector G., '35..4803 Lincoln av., Chicago 25, Ill. ALR**
②Large, Secord H., '04....170 Eddy rd., Cleveland, Ohio ALR
**Larimore, Fred Campbell, '40**
500 Penn av., Pittsburgh 22, Pa. OP
①Larkin, Bernard J., '16
305 Hume Mansur bldg., Indianapolis 4, Ind. OP
Larmore, Joseph L., '44
712 Anderson Bank bldg., Anderson, Ind. ALR
Larose, Henry, '46.1218 Richards bldg., New Orleans 12, La. OP
③Larsell, Olaf, '43, Ph.D., Sc.D.,
University of Oregon, Portland, Ore.
①Larsen, Carl L., '16..Lowry Med. bldg., St. Paul 2, Minn. OALR
**Larson, Bertil T., '38.....Riker bldg., Pontiac 15, Mich. ALR**
Larson, G. Arthur, '33....608 Black bldg., Fargo, N. D. OALR
②LaRue, C. L., '22...Highland Clinic, Shreveport 50, La. OALR
**Lasky, Mortimer A., '39**
Fox Medical bldg., 1 Nevins, Brooklyn, N. Y. OP
①Lassen, Fritz, '15.....Pueblo Clinic bldg., Pueblo, Colo. OALR
**Lassiter, Chester L., '38**
Hamilton Nat'l Bk., Chattanooga 2, Tenn. ALR
**Last, Murray A., '36...1095 Park av., New York 28, N. Y. OP**
Lathbury, Edwin F., '42..333 Linwood av., Buffalo 9, N.Y. ALR
**Lathrop, Frank D., '40**
Lahey Clinic, 605 Commonwealth Av., Boston, Mass. ALR
**Latimer, Marvin L., '38**
129 Whitney av., New Haven 10, Conn. ALR
②Lauber, Prof. Dr. Hans, '34
Grillparzerstrabe 7/15, Wien I/1, Austra OP
**Lauppe, Frederick A., '40**
1801 David Whitney bldg., Detroit 26, Mich. OP
**Laval, Joseph, '31.....136 E. 64th, New York 21, N. Y. OP**
②Lavigne, A. A., '18..667 Madison Ave., New York 21, N.Y. OALR
**Lavine, Oscar, '46**
Suite 620, 1801 Eye St., N.W., Washington 6, D.C. OP
①Lawrence, Granville A., '13
Medical Arts bldg., Philadelphia 3, Pa. OP
**Lawson, Lawrence J., '31.....636 Church, Evanston, Ill. ALR**
**Lawwill, Stewart, '28**
Medical Arts bldg., Chattanooga 3, Tenn. OALR
Lazar, A. M., '38.....30 N. Michigan, Chicago 2, Ill. ALR
Lazar, Nathan K., '36
Rm 2025, 25 E. Washington st., Chicago 2, Ill OP
**Leach, Robert S., '41**
Medical Arts bldg., Knoxville 2, Tenn. OP
Leahey, Brendan D., '36.....9 Central, Lowell, Mass. OP
Leahey, George A., '37.....45 Merrimack, Lowell, Mass. OP
Leamer, Bruce V., '38, Capt. (MC) USN, School of Aviation
Medicine—U.S. Naval Air Station, Pensacola, Fla. ALR
**Leasure, J. Kent, '31**
611 Hume-Mansur bldg., Indianapolis 4, Ind. ALR
**Leath, Maclean B., '44..511 N. Main st., High Point, N. C. ALR**
**Leavenworth, R. O., '30**
Lowry Med. bldg., St. Paul 2, Minn. OP
①Leavy, C. A., '12....9054 Clayton Rd., St. Louis 17, Mo. ALR
**Lebensohn, James E., '27**
6200 N. Kenmore av., Apt. 1022, Chicago 40, Ill. OALR
Le Compte, E. D., '27
9 Exchange pl., Salt Lake City 1, Utah OALR
Lederer, Francis L., '25..307 N. Michigan Av., Chicago, Ill. ALR
**Lee, Otis S., Jr., '45.....112 Rimbach st., Hammond, Ind. OP**
**Lee, Wayne Robert, '43**
613 Medical Arts bldg., Burlington, Iowa ALR
**Leech, V. M., '30.....55 E. Washington, Chicago 2, Ill. OP**
Lees, Charles Ray, '46
806 Medical Arts bldg., Fort Worth, Tex. OP
**LeFevre, Willis H., '35.....234 N. Duke, Lancaster, Pa. ALR**
**Lefkon, Irving M., '39..37-14 73rd, Jackson Heights, N. Y. ALR**
②Lefler, Anna B., '18
2419 W. 18th St., Los Angeles 6, Calif. OALR
**Leggett, Albert E., '34**
614 Fincastle bldg., Louisville 2, Ky. OP
①Lehan, James W., '16....Park Place bldg., Greeley, Colo. OALR
**Lehrfeld, Louis, '39.....1321 Spruce, Philadelphia 7, Pa. OP**
**Leich, Charles F., '41, 124 S. E. First St., Evansville 8, Ind. ALR**
Leinfelder, Placidus J., '37
University Hosp., Iowa City, Iowa OP
**LeJeune, F. E., '27...Ochsner Clinic, New Orleans 15, La. ALR**
**Lemel, Samuel L., '35.1030 Euclid av., Cleveland 15, Ohio ALR**
①Lemere, H. B., '12,
9615 Brighton Way, Beverly Hills, Calif. OALR
②Lemoine, A. N., '20....Rialto bldg., Kansas City 6, Mo. OP
**Lempert, Julius, '43....119 E. 74th, New York 21, N. Y. ALR**
**Lenholt, Eric H., '44...101 Lenox av., Daytona Beach, Fla. ALR**
**Lennarson, Vincent A. E., '46**
4 So. Genesee st., Waukegan, Ill. ALR
**Lennon, Robert Watson, '41.....Morris bldg., Joliet, Ill. OP**
Lentine, Joseph, '38,
395 Commonwealth Ave., Boston 15, Mass. ALR
**Lenzon, Aloysius Francis, '45**
206 Marquette St., LaSalle, Ill. OP
Leonard, Jacob C. Jr., '41
3½ E. First av., Lexington, N. C ALR
**Leopold, Irving H., '43**
1930 Chestnut St., Philadelphia, 3, Pa. OP
Lepard, Cecil W., '37
David Whitney bldg., Detroit 26, Mich. OP
**Lerner, Macy L., '23.....332 Park av., Rochester 7, N. Y. OP**
**Leshin, Norman, '31,..55 E. Washington st., Chicago 2, Ill. ALR**
①Lester, Harry S., '09.....Murray bldg., Streator, Ill OALR
Le Tellier, Forrest S., '36 Doctors bldg., Knoxville 30, Tenn. OP
**Levbarg, John J., '37,**
176 W. 87 st., New York 24, N. Y. ALR
**Levin, Abram A., '37....Carew Tower, Cincinnati 2, Ohio OP**
**Levin, Bert G., '45....1210 Lowry bldg., St. Paul 5, Minn. ALR**
**Levin, Nathaniel M., '39....517 DuPont bldg., Miami, Fla. ALR**
**Levitt, Edward J., '46**
840-842 Maccabees bldg., Detroit 2, Mich. ALR
**Levitt, Jesse M., '35....991 Ocean av., Brooklyn 26, N.Y. OP**
**Levy, Abram, '34..1401 Plainfield av., So. Plainfield, N. J. OP**
**Levy, Harry H., '31.....119 Murray, Binghamton, N. Y. OP**
**Levy, Irvin, '39.....222 W. State, Trenton 8, N. J. OP**
①Levy, Louis, '16..Commerce Title bldg., Memphis 3, Tenn. OALR
**Lewin, Michael, '42..149 Columbus av., Tenafly, N.J. ALR PLS**
**Le Win, Thurber, '29....112 Linwood av., Buffalo 9, N. Y. OP**
**Lewis, Charles Kelley, '45**
2109 Sterick bldg., Memphis, Tenn. ALR

①Lewis, Fielding O., '21, (Retired).....Media, Pa. ALR
②Lewis, George A., '20.....213 Main, Roundup, Mont. OALR
②Lewis, J. D., '19
650 Hot Springs Dr., Santa Barbara, Calif. OALR
①Lewis, James Prince, '15...412 Beacon St., Boston, Mass. ALR
Lewis, Philip M., '28....Exchange bldg., Memphis 3, Tenn. OP
Lewis, Samuel D., '43.....49 Pearl, Hartford 3, Conn. OP
Lewis, Samuel J., '37.....S. F. C. bldg., Augusta, Ga. ALR
Lewy, Alfred, '42.....25 E. Washington st., Chicago 2, Ill. ALR
Lewy, Robert B., '39.....25 E. Washington, Chicago 2, Ill. ALR
Leyda, James H., '27...Metropolitan bldg., Denver 2, Colo. ALR
Lieberman, Alfred T., '44
29 E. Mt. Vernon Pl., Baltimore, Md. ALR
Lieberman, George Emanuel, '45
1721 Pine St., Philadelphia, Pa. ALR
Lieberman, Harold, '46....175 June St., Worcester, Mass. ALR
Lierle, Dean M., '28
University Hospital, Iowa City, Iowa ALR-PI S
Lifschutz, Jacob, '25...30 N. Michigan av., Chicago 2, Ill. OALR
Liggett, Harold, '36...44-05 Parson blvd., Flushing, N.Y. ALR
Light, George, '37.....709 N. 11th, Milwaukee 3, Wis. ALR
①Lillie, Harold I., '20.....Mayo Clinic, Rochester, Minn. ALR
Limbach, Earl Frederick, '40
30 W. 59th, Station G, New York 19, N. Y. ALR
Lindberg, Vernon L., '42
Physicians & Surgeons Bldg., Minneapolis, Minn. OP
Lindsay, John R., '36.....950 E. 59th, Chicago 37, Ill. ALR
Lingeman, B. N., '30...Ben Hur bldg., Crawfordsville, Ind. ALR
Lingeman, E. L., '29
411 Hume-Mansur bldg., Indianapolis 4, Ind. ALR
Linhart, William Oran, '46
521 Medical Arts Bldg., Pittsburgh, Pa. OP
②Linn, J. G., '19.....Jenkins Arcade, Pittsburgh 22, Pa. OP
Linton, Claire S., '37
c/o Cottonseed Delinting Corp., Kennett, Mo. ALR
Lippmann, Otto, '43, Capt. (MC)
Regional Hospital, Ft. Benning, Ga. OP
①Lischkoff, M. A., '13.....Blount bldg., Pensacola, Fla. OALR
Lisman, Jack Victor, '46...654 Madison Av., New York, N.Y. OP
③Lister, Sir William T., '23.....Address Unknown
②Litchfield, J. T., '19
Medical Arts bldg., Minneapolis 2, Minn. OALR
Littell, J. Jerome, '31
603 Hume-Mansur bldg., Indianapolis 4, Ind. ALR
Little, Milton F., '38.....49 Pearl, Hartford 3, Conn. OP
Littwin, Charles, '33...950 Queen Anne rd., Teaneck, N. J. OALR
Liva, Arcangelo, '40....5 Pangborn pl., Hackensack, N. J. OALR
Livingston, George S., '30
185 N. Wabash av., Chicago 1, Ill. ALR
Livshin, Norman, '46.....631 Univ. bldg., Syracuse, N.Y. ALR
①Lloyd, A. W., '15.....8150 Forest Ave., Hammond, Ind. OALR
Lloyd, Ralph I., '25....14 Eighth av., Brooklyn 17, N. Y. OP
Locke, William Walter, '46
507 Empire bldg., Birmingham, Ala. ALR
Lockwood, Chester C., '42..218 S. 5th St., Las Vegas, Nev. OP
Loder, L. L., '43, 506 Hackley
Union Nat'l Bank bldg., Muskegon 11, Mich. OP
Lodge, Edmund Anderson, '45
791/2 Prospect St., Gloucester, Mass. ALR
Loeb, Wm. J., '42..1125 Schofield bldg., Cleveland 15, Ohio ALR
Loewe, Walter R., '34..114 Madison av., New York, 28, N.Y. OP
②Loftus, John Edward, '22
1930 Chestnut, Philadelphia 3, Pa. ALR
Logue, Lyle J., '31.....1304 Walker, Houston 2, Texas ALR
①Lokey, H. M., '09....Medical Arts bldg., Atlanta 3, Ga. OALR
Lombardo, Melchiore, '23
142 Joralemon, Brooklyn 2, N. Y. OP
Long, Albert Emanuel, '46..169 Main St., Weston, W. Va. OP
Long, John C., '36.....Metropolitan bldg., Denver 2, Colo. OP
Long, Samuel H., '39
Medical Arts bldg., Chattanooga 3, Tenn. ALR
Longaker, Edwin P., '43
41 E. Montgomery av., Ardmore, Pa. ALR
②Loomis, E. A., '18
Medical Arts bldg., Minneapolis 2, Minn. OALR
Loomis, George L., '43...310 Choate bldg., Winona, Minn. ALR
Looper, Edward A., '23.104 W. Madison, Baltimore 1, Md. OALR
Lordan, John P., '39
1930 Wilshire blvd., Los Angeles 5, Calif. OP
Lore, John M., '36...325 W. End av., New York 23, N. Y. ALR
Loutfallah, Michel, '44,
1826 State st., Santa Barbara, Calif. OP
Lovesey, Burton E., '39
76 Bay State road, Boston 15, Mass. ALR
Lowenstein, Harold H., '44
Veterans Administration Hosp., Columbia, S.C. OP
Lowenthal, Gerson, '35
277 Doctor's bldg., Cincinnati 2, Ohio ALR
Lower, Raymond A., '38
1455 Paulson Medical Arts bldg., Spokane, Wash. ALR
Lowry, B. W., '25.....Citizens Bank bldg., Tampa 2, Fla. OALR
Lowy, Karl, '44.....187 S. Goodman St., Rochester, N.Y. ALR
Loy, A. W., '43, Capt. (MC) USN
Navy #100, c/o FPO, New York, N.Y. OP
Lubart, Joseph, '44...310 West 86th st., New York, N.Y. ALR
Luber, Samuel, '38
1801 Eye st., N. W., Washington 6, D. C. ALR
Lucas, Harold R., '28
Medical-Dental bldg., Portland 5, Ore. OALR
Ludwig, William Henry, '40
Medical Arts bldg., Tacoma 2, Wash. ALR
①Luedde, W. H., '16..Metropolitan bldg., St. Louis 3, Mo. OP
Luhr, Alfred F., '31.....40 W. North, Buffalo 2, N. Y. OP
Luke, William R. F., '38
Medical Arts bldg., Toronto 5, Can. OP
①Lukens, Charles, '06.....316 Michigan, Toledo 2, Ohio OALR
Lukens, J. A., '30.....316 Michigan, Toledo 2, Ohio ALR
②Lukens, Robert McD., '20, Suite 1001, Medical
Arts bldg., 16th & Walnut, Philadelphia 3, Pa. ALR
Lundon, A. E., '27.....1463 Bishop, Montreal, Can. ALR
Lundvick, Cyril V., '38
Medical Arts bldg., Tacoma 2, Wash. OP
Luongo, Romeo A., '31...2054 Locust, Philadelphia 3, Pa. ALR
Lupo, Carl W., '32...215 St. Johns Pl., Brooklyn 17, N.Y. ALR
Lupton, Irving M., '28.Medical Arts bldg., Portland 5, Ore. OALR
Lurie, M. H., '30.....483 Beacon, Boston 15, Mass. ALR
Lux, Paul, '25.....Bryant bldg., Kansas City 6, Mo. ALR
Lyle, Donald J., '27.....Carew Tower, Cincinnati 2, Ohio OP
Lyman, Edward H., '44....308 N. 6th St., St. Louis 1, Mo. ALR
Lyman, Harry W., '23.....308 N. 6th, St. Louis 1, Mo. ALR
Lynam, Patrick J., '41..1415 Kalakaua ave., Honolulu, T.H. ALR
Lynch, Joseph S., '42.....302 E. 9th St., Chester, Pa. OP
Lynch, Mercer G., '39....Ochsner Clinic, New Orleans, La. ALR
Lynch, Robert J., '31..Equitable bldg., Des Moines 9, Iowa ALR
③Lyons, H. R., '20....1301 N. State St., Chicago 10, Ill. ALR

## M

Mabee, Oscar Judson, '41
Medical Arts bldg., Mitchell, S. D. ALR
MacCardell, Frank Cameron, '40
193 Waterman st., Providence 6, R. I. ALR

MacCready, Paul B., '31.442 Temple, New Haven 11, Conn. ALR
Macdonald, James Orville, '41
67 E. 59th, New York 22, N. Y. ALR
Macdonald, J. C., '30
301 N.W. 12th, Oklahoma City 3, Okla. ALR
MacDonald, Philip G., '35.115 E. 61st, New York 21, N.Y. ALR
Macdonald, Roderick, '40.330 East Main, Rock Hill, S. C. ALR
MacFarlane, Howard W., '41
David Whitney bldg., Detroit 26, Mich. ALR
①Macfarlane, P. B., '16,
Medical Arts bldg., Hamilton, Ont., Can. OALR
Mackenzie, Roland C., '39....30 Grant, Waltham 54, Mass. OP
②MacLachlan, A. A., '18..May bldg., Pittsburgh 22, Pa. ALR
MacLean, Angus L., '35, 1201 N. Calvert, Baltimore 12, Md. OP
MacMillan, Andrew L., Jr., '37
46 Pleasant, Concord, N. H. ALR
MacMillan, J. A., '25.....1410 Stanley, Montreal 2, Can. OP
MacMillan, Karl D., '44
1206 Clark bldg., Pittsburgh 22, Pa. OP
Macneil, Frank A., '38.Medical Arts bldg., Winnipeg, Can. ALR
Macnie, John P., '35..2501 Palisade av., New York 63, N. Y. OP
②Macnie, J. S., '19
Medical Arts bldg., Minneapolis 2, Minn. OALR
Macpherson, Duncan, '36
920 Fifth av., New York 21, N. Y. ALR
Madeley, Howard Randall, '40
727 Sonoma st., Vallejo, Calif. OP
Magee, F. Earle, '37.....222 Seneca, Oil City, Pa. ALR
②Maggard, D. I., '20....Orpheum bldg., Wichita 2, Kan. OALR
Maghy, Charles A., '26
Medico Dental bldg., San Diego 1, Calif. OP
Mahoney, Charles L., '40..1438 S. Center, Terre Haute, Ind. ALR
Mahoney, Frank A., Jr., '43..213 Union st., Bennington, Vt. ALR
Mahoney, Paul L., '28...Donaghey bldg., Little Rock, Ark. ALR
②Maiden, S. D., '18
Kennedy V. A. Hospital, Box 24, Memphis 15, Tenn. OALR
Maier, Haskel Levitt, '45
936 Medical Arts bldg., Tacoma 2, Wash. ALR
Maier, Marcus M., '43.....513 Front, Hempstead, N. Y. ALR
Maliniac, Jacques W., '36,
106 E. 78th, New York 21, N. Y. ALR-PI S
Mallon, Frank E., '37, The Williamsburgh Bank bldg.,
1 Hanson Place, Brooklyn 17, N. Y. OP
Malone, F. B., '30...2609 Nineteenth st., Lubbock, Texas ALR
Malone, Phocian W., '36.....811 Main, Big Spring, Texas ALR
Maloy, Wayland H., '33
608½ W. Sheridan av., Shenandoah, Iowa OP
Mandeville, Clifford Bagley, '41
Hackley Union Bank, Muskegon, Mich. ALR
Maness, Guy Milford, '41
119 7th av. North, Nashville 3, Tenn. ALR
Manigan, Thomas P., '39,
431 Patterson, Memphis 11, Tenn. ALR
Manion, Marlow W., '36
601 Hume-Mansur bldg., Indianapolis 4, Ind. ALR
Mann, J. J., '34.....255 State, Perth Amboy, N. J. ALR
Mann, Mortimer, '43
323 Hume-Mansur bldg., Indianapolis 4, Ind. OP
Mann, William A., '38..30 N. Michigan av., Chicago 2, Ill. OP
②March, Clara A., '22....465 Ashland av., Buffalo 13, N.Y. OP
Marchman, Oscar M., '35
814 Medical Arts bldg., Dallas 1, Texas ALR
Marchman, Oscar M., Jr., '44,
816 Medical Arts bldg., Dallas 1, Tex. OP
Marcotte, Raymond H., '32.....196 Main, Nashua, N. H. ALR
Marcove, Maurice E., '39...1612 Tremont, Denver 2, Colo. OP
Marden, Philip Ayer, '46
1912 Spruce St., Philadelphia 3, Pa. ALR
①Markel, J. Clyde, '17
806 Pitt Bank bldg., Pittsburgh 22, Pa. OP
Marks, Julian B., '29
1093 Broxton av., Los Angeles 24, Calif. ALR
Marks, Moses I., '42
10515 Carnegie Ave., Cleveland 6, Ohio ALR
Marks, Paul L., '42....131 No. 8th St., Baton Rouge, La. OP
Marks, Roland F., '39
450 Sutter St., San Francisco 8, Calif. ALR
Marks, Samuel B., '28.....203 W. 2nd, Lexington 15, Ky. ALR
Marmelstein, Maurice, '43
1st Nat'l Bank bldg., Carbondale, Pa. OP
Marmor, William A., '46..607 N. Grand blvd., St. Louis, Mo. ALR
Marsden, W. C., '30....99 N. Franklin, Wilkes-Barre, Pa. OALR
Marshall, Don, '36,
1410 American Bank Bldg., Kalamazoo, Mich. OP
Marshall, George G., '23.....Gryphon bldg., Rutland, Vt. OALR
Marshall, Hal E., '35,
22 Colonial Court, Eastborough, Wichita, Kans. ALR
①Marshall, Watson, '14
121 University pl., Pittsburgh 13, Pa. ALR
Marshall, William J., '38..Montana block, Missoula, Mont. ALR
Martin, Ashby G., '36
895 Lexington av., New York 21, N. Y. ALR
Martin, Henry F., '40, 904 Government St., Mobile 20, Ala. ALR
Martin, Hilmar G., '39
231 W. Wisconsin av., Milwaukee 3, Wis. OP
Martin, N. A., '37.....Gallipolis Clinic, Gallipolis, Ohio ALR
Martin, Paul T., '38.235 E. Santa Clara, San Jose 20, Calif. ALR
Martin, Roy D., '29....Masonic bldg., Grand Island, Nebr. ALR
Martin, W. O., Jr., '30.....478 Peachtree, Atlanta 3, Ga. OP
Mason, Roy E., '28.....Frisco bldg., St. Louis 1, Mo. OP
Masor, Philip L., '41, 306 E. 15th St., New York 3, N.Y. OP
Master, John R., '43.....1058 Rose bldg., Cleveland, Ohio ALR
Masters, John M., '38
805 Hume Mansur Bldg., Indianapolis 4, Ind. OP
Masters, Robert J., '28
805 Hume-Mansur bldg., Indianapolis 4, Ind. OP
Mather, Homer Raymond, '42
1st Nat'l Bank bldg., Latrobe, Pa. ALR
Mather, Homer Raymond, Jr., '45
1st Nat'l Bank bldg., Latrobe, Pa. OP
Matheson, John Hollis, '39
802 Equitable bldg., Des Moines 9, Ia. OP
Mathews, Raymond H., '38
186 South st., Morristown, N. J. ALR
Mathias, Daniel Francis, '40
Central Tower bldg., Akron 8, Ohio ALR
Matthews, John L., '43, 414 Navarro St., San Antonio 5, Tex. OP
①Matthews, Justus, '10
Medical Arts bldg., Minneapolis 2, Minn. LR
Mattis, Robert Dean, '42,
375 North Taylor av., St. Louis 8, Mo. OP
Matusow, A. P., '35
1100 Grand Concourse, New York 56, N. Y. ALR
Matusow, Meyer, '44
6 Old Mamaroneck Rd., White Plains, N.Y. OP
Matzkin, Lloyd L., 43....1525 E. 53rd St., Chicago 15, Ill. ALR
Maumenee, Alfred E., '43
Johns Hopkins Hospital, Baltimore 5, Md. OP
Maw, Raymond B., '39
699 E. South Temple, Salt Lake City 2, Utah ALR
Maxwell, Hal W., '43
Professional bldg., Hempstead, L.I., N.Y. OP
Maxwell, Harvey C., '35..1718 N. Main, Santa Ana, Calif. OALR

Maxwell, James H., '34
1st Nat'l Bank bldg. Ann Arbor, Mich. ALR
Maxwell, Joseph S., '39. 323 Adams St., Fairmont, W. Va. OP
May, Frederick T., '41....76 W. Adams, Detroit 26, Mich. OP
①May, J. V.. '15.....1703 Main, Marinette, Wis. OALR
May, James W., '44..1016 Rialto bldg., Kansas City 6, Mo. OP
①Maybaum, J. L., '16..1070 Park av., New York 28, N. Y. ALR
Mayerberg, Emil R., '29
Medical Arts bldg., Wilmington 9, Del. ALR
McAdams, Wm. R., '33.....723 Congress, Portland 4, Me. OP
McAlester, Andrew W., Jr., '20
Bryant bldg., Kansas City 6, Mo. OP
McAlester, Andrew W., 3rd, '32
2003 Bryant bldg., Kansas City, Mo. OP
McAlpine, Paul Theodore, '42
Summit Medical Group, 129 Summit Av., Summit, N.J. OP
McAndrews, Leo F., '31...Mermont Plaza, Bryn Mawr, Pa. OP
McAskill, James E., '32
Woolworth bldg., Watertown, N. Y. ALR
McAuley, Albert G., '38
1301 Dorchester st. W., Montreal, Can. OP
McAuliffe, G. W., '32
630 Park av., New York 21, N. Y. ALR-PI S
②McBean, Geo. M., '20..25 E. Washington, Chicago 2, Ill. OALR
McBean, James Blish, '38
302 Seventh av., S. W., Rochester, Minn. ALR
McBride, Linn Frederick, '42, 1501 Asbury av., Evanston, Ill. ALR
McBride, Noel S., '40....12 S. 7th st., Terre Haute, Ind. OP
McCall, J. W., '43.....610 Rose bldg., Cleveland 15, Ohio ALR
②McCannel, Archie D., '20
Corner 1st st. S.W. and Central av., Minot, N. D. OALR
McCart, Howard W. D., '39
170 St. George st., Toronto 5, Ontario, Can. ALR
McCarthy, C. T., '40.....1810 Pine, Philadelphia 3, Pa. ALR
②McCaskey, Carl H., '11
Guaranty bldg., Indianapolis 4, Ind. ALR
McCaslin, Murray F., '40
Union Trust bldg., Pittsburgh 19, Pa. OP
McClanahan, Robert, '37..60 E. 75th, New York 21, N. Y. OP
McClintock, Walter L., '39, Capt. (M.C.),
120 Putnam st., Quincy, Mass. ALR
McCloskey, Paul, '40.....338 Locust st., Johnstown, Pa. ALR
McClure, George, '39.....411 Thirtieth, Oakland 9, Calif. ALR
McCollom, Robert L., '42..115 E. 61st st., New York, N. Y. ALR
McCollough, T. B., '32
121 University Place, Pittsburgh 13, Pa. ALR
McComiskey, Arthur James, '40
3420 Prytania st., New Orleans 15, La. ALR
McConkie, Willis L., '36.....Carroll, Iowa ALR
①McCool, J. L., '11...450 Sutter, San Francisco 8, Calif. OP
McCormick, T. F., '30.....Wells bldg., Milwaukee 2, Wis. ALR
McCoy, L. L., '30.....1317 Marion, Seattle 4, Wash. OP
①McCready, J. Homer, '12
121 University pl., Pittsburgh 13, Pa. ALR
McCrory, James Lynn, '46..607 N. Grand, St. Louis 3, Mo. ALR
McCuaig, Duncan Roderick, '40, 40 E. 61st, New York, N. Y. ALR
McCuaig, Victor C., '39...40 E. 61st, New York 21, N. Y. ALR
②McCulloch, Robert J. P., '19
Medical Arts bldg., Toronto 5, Can. OALR
McCullough, C. J., '29
Washington Trust bldg., Washington, Pa. OALR
McCullough, Lester E., '42
1553 Woodward av., Detroit 26, Mich. OP
McCullough, Malcolm K., '39
Medical Arts bldg., Dallas 1, Texas OP
McCurdy, Gordon Judson, '40..General Del., La Honda, Cal. ALR
McCutchan, Frank, '38....100 W. Innes, Salisbury, N. C. ALR
McDaniel, Reuben L., '14
401 St. James bldg., Jacksonville, Fla. OP
McDannald, C. E., '27...100 W. 59th, New York 19, N.Y. OP
McDavitt, Bertha S. S., '29..Main & Av. A, Temple, Texas ALR
McDermott, John J., '24..505 Pleasant, St. Joseph, Mich. OALR
②McDiarmid, H. Oliver, '18
35 Clement block, Brandon, Man., Can. OALR
②McDonald, C. L., '19.....Rose bldg., Cleveland 15, Ohio OALR
McDonald, P. Robb, '40
255 S. 17th St., Philadelphia, Pa. OP
McDougall, J. Calhoun, '32
Medical Arts bldg., Atlanta 3, Ga. ALR
McDougall, Wm. L., '31....Doctors bldg., Atlanta 3, Ga. ALR
①McDowell, N. D., '14.275 Alexander, Rochester 7, N. Y. OALR
McEachern, Margaret A., '39
Medical Arts bldg., Toronto, Can. ALR
McEwen, James Woods, '45
670 Cherry St., Terre Haute, Ind. OP
McFarland, Corley B., '46
122 N. Lafayette Blvd., South Bend, Ind. OP
McFarlane, Donald C., '37
380 Queens av., London, Ont., Can. ALR
McGannon, Francis Lambert, '41
14900 Detroit av., Lakewood 7, Ohio ALR
McGarry, H. Isabelle, '43.....636 Church, Evanston, Ill. OP
McGarvey, Wm. E., '29
Jackson City Bank bldg., Jackson, Mich. OALR
McGavic, John S., '40....132 Morris Ave., Bryn Mawr, Pa. OP
McGeary, George E., '37
Medical Arts bldg., Minneapolis 2, Minn. ALR
McGehee, Melvin W., '42, 425 11th, Huntington 1, W. Va. ALR
McGillicuddy, O. B., '30
1908 Olds Tower bldg., Lansing, Mich. ALR
McGovern, Francis H., '39
Masonic Temple bldg., Danville, Va. ALR
McGowan, Joseph Francis, '43
New Medical bldg., Asheville, N. C. ALR
McGrath, Harry, '41
12 Shore Lane, Bay Shore, L. I., N. Y. OP
McGrath, James B., '37
2615 Grand Concourse, New York 58, N. Y. ALR
McGrath, Philip R., '39, 842-845 Jefferson bldg., Peoria, Ill. ALR
McGraw, James Lorenz, '46
619 University bldg., Syracuse, N.Y. OP
McGregor, Gregor W., '31.170 St. George, Toronto 5, Can. ALR
①McGuire, H. H., '13..105 N. Braddock, Winchester, Va. OP
McGuire, John, '43.....Peery bldg., Bluefield, W. Va. ALR
McGuire, William P., '40..105 N. Braddock, Winchester, Va. OP
McHenry, L. Chester, '20
Medical Arts bldg., Oklahoma City 2, Okla. ALR
McIntire, Frederic J., '37....63 N. Common, Lynn, Mass. ALR
McIntyre, George F., '37.25 E. Washington, Chicago 2, Ill. ALR
McKay, Edward D, '43..Scott & White Clinic, Temple, Tex. OP
McKee, Joseph W., '28
Professional bldg., Kansas City 6, Mo. OP
McKee, Thomas L., '39,
436 E. Las Olas blvd., Ft. Lauderdale, Fla. OP
McKee, Thomas P., '39
207 E. Watauga av., Johnston City, Tenn. OALR
①McKennan, James W., '17
Washington Trust bldg., Washington, Pa. OP
McKenzie, George Edward, '45
602 DuPont bldg., Miami, Fla. ALR
McKenzie, William Raymond, '41
101 W. Read st., Baltimore 1, Md. ALR

②McKeown, Elmer E., '18..Republic bldg., Denver 2, Colo. OALR
McKinney, A. R., '23
330 S. Washington av., Saginaw, Mich. OALR
McKinney, James Wesley, '41
Exchange bldg., Memphis 3, Tenn. OALR
McLane, Jesse Newman, '42
1212 N. Palafax St., Pensacola, Fla. ALR
McLaughlin, John J., '41
Owen & Lacrosse aves. Lansdowne, Pa. ALR
McLaughlin, R. C., '43
3780 Wilshire bldg., Los Angeles 5, Calif. ALR
①McLaughlin, Ralph S., '44,
805 Atlas bldg., Charleston, W. Va. OP
McLaurin, James Walter, '41..251 3rd, Baton Rouge 6, La. ALR
McLaurin, John G., '26..4710 Munger av., Dallas 4, Texas ALR
McLean, John M., '40...525 E. 68th, New York 21, N. Y. OP
McLellan, Thomas G., '35
Second Nat'l Bank bldg., Connellsville, Pa. OP
McLemore, Carl Sloan, '40..1202 S. Kuhl ave., Orlando, Fla. ALR
McLeod, Angus M., '36, Capt. (M.C.),
Camp Borden Military Hospital, Camp Borden, Ont., Can. ALR
McLeod, John, '39, Major (MC)
c/o Dr. Hugh Miller, 40 E. 55 St., Kansas City, Mo. OP
McMahon, Bernard J., '26
Missouri Theatre bldg., St. Louis 3, Mo. ALR
①McMurray, J. B., '13
Washington Trust bldg., Washington, Pa. OALR
McMurray, John S., '43....6 S. Main St., Washington, Pa. ALR
McNair, Stirling S., '41..312 N. Congress St., Jackson, Miss. OP
McNally, John Joseph, '46..121 E. 60th St., New York, N.Y. ALR
McNally, Wm. James, '28....Linton Apts., Montreal, Can. ALR
McNay, Miller Ost, '41
472 Central av., St. Petersburg 5, Fla. ALR
McNichols, W. A., '25.....101 W. First st., Dixon 3, Ill. OALR
McPherson, Samuel D., '35
McPherson Hospital, Durham, N. C. ALR
McQuiston, Ralph J., '37
608 Guaranty bldg., Indianapolis, Ind. ALR
McQuoid, Robert Moffat, '40....39 Columbia, Bangor, Me. ALR
McRae, John H., '30
Medical Arts bldg., Grand Rapids 2, Mich. OP
McReynolds, George S., Jr., '40
Merimas Professional bldg. Galveston, Tex. ALR
McRoberts, Earl S., '38..55 E. Washington, Chicago 2, Ill. ALR
McWilliam, Charles S., '37....205 Liberty, Newburgh, N.Y. OP
McWilliams, Charles A., '27
1300 27th av., Gulfport, Miss. ALR
Mead, Ernest Charles, '41
106½ E. California, Gainesville, Texas ALR
Means, R. G., '30.....40 S. 3rd, Columbus 15, Ohio ALR
Meek, Raymond E., '31..729 Park Av., New York 21, N.Y. OP
Meer, Nadhim Edward, '45..400 Park ave., Paterson, N.J. ALR
Meinberg, William H., '39.....906 Olive, St. Louis 1, Mo. OP
Mellinger, Wm. J., '27
323 E. Sola st., Santa Barbara, Calif. ALR
Meloy, Wm. C., '33,
2019 R st. N.W., Washington 9, D.C. ALR-PI S
Melton, Bernard L., Jr., '31..15 E. Monroe, Phoenix, Ariz. OP
Meltzer, Philip E., '34.20 Charlesgate W., Boston 15, Mass. ALR
Mendel, James H., '32....Ingraham bldg., Miami 32, Fla. ALR
Mengel, Sterling F., '40..612 W. Market st., Pottsville, Pa. OP
Menger, Louis C., '29...202 Weirfield, Brooklyn 21, N. Y. ALR
Mensher, Ira Wadsworth, '42.50 Plaza, Brooklyn 17, N. Y. OP
Menzies, Clifford G., '39
733 Woodward av., Iron Mountain, Mich. ALR
②Mercer, Ray, '20.....811-13 W.C.U. bldg., Quincy, Ill. OALR
①Mercer, Richard E., '03...126 Monterey, Detroit 3, Mich. LR
Meredith, Lawrence Cornelius, '40
124 Middle av., Elyria, Ohio ALR
Merica, F. W., '30...14805 Detroit av., Lakewood 7, Ohio ALR
Merkel, Byron M., '38,
1112 Equitable bldg., Des Moines 9, Iowa ALR
Merkel, C. C., '28
85 Kercheval av., Grosse Pointe Farms 30, Mich. ALR
Merrick, Charles Gordon, '40...Leon bldg., Ft. Myers, Fla. ALR
①Merrill, H. G., '17..3245 Fourth av., San Diego 3, Calif. OP
Merrill, Rowland Hyde, '46
1010 First National Bank, Salt Lake City, Utah OP
Mertins, Paul, Jr., '37..32 Clayton St., Montgomery, Ala. ALR
Merz, Earl H., '43....4319 N. Greenview, Chicago 13, Ill. OP
Mesirow, Maurice E., '35.117 E. Cook, Santa Maria, Calif. OP
Messenger, Harry K., '39
313 Commonwealth av., Boston 15, Mass. OP
①Messinger, H. C., '14....210 Angell, Providence 6, R. I. OP
Messier, A. Eugene, '25.....390 Main, Worcester 8, Mass. OALR
①Metz, Roy B., '12...1422 Euclid av., Cleveland 15, Ohio OP
Meyer, George P., '37.....410 Haddon av., Camden, N. J. OP
Meyer, Monte F., '45....645 Canal bldg., New Orleans, La. ALR
Meyer, Samuel J., '28...58 E. Washington, Chicago 2, Ill. OP
Meyers, Paul T., '42
1109 U.S. Bank bldg., Johnstown, Pa. ALR
Mezz, David, '37...330 New York av., Brooklyn 13, N. Y. ALR
①Middleton, A. B., '11.....Clermont, Fla. OALR
③Middleton, Harry E., '20.....206A W. 3rd, Alton, Ill. OALR
Miears, Claude H., '45.....713 Scarbrough, Austin, Tex. ALR
Miksch, Henry Fred, '41.....209 S. Park, Rochester, Pa. ALR
Milder, Benjamin, '43.735 Leland ave., University City 5, Mo. OP
Miller, A. Boyd, '43
1006 Professional bldg., 429 Penn av., Pittsburgh 22, Pa. ALR
Miller, Alden H., '39..500 S. Lucas ave., Los Angeles, Calif. ALR
②Miller, Alice R., '19
505 Guaranty bldg., West Palm Beach, Fla. OALR
Miller, Alvin R., '39.....4th and Pike, Seattle 1, Wash. ALR
Miller, Claude K., '45
Widby-Hadley bldg., Wenatchee, Wash. OP
Miller, Daniel, '44..20 Charlesgate West, Boston 15, Mass. ALR
Miller, Edwin Ephron, '42
Medical Arts bldg., Knoxville 1, Tenn. ALR
Miller, G. Leslie, '35.14804 Detroit av., Lakewood 7, Ohio OP
Miller, H. Edward, '30.634 N. Grand blvd., St. Louis 3, Mo. ALR
Miller, Harold Rexford, '45
114 E. 54th St., New York 22, N.Y. OP
①Miller, Hugh, '20.....Argyle bldg., Kansas City 6, Mo. OALR
Miller, Isidore, '37
2021 Grand Concourse, New York 53, N. Y. ALR
Miller, John, '30.....63 North st., Greenwich, Conn. ALR
Miller, Joseph A., '32...364 Prospect, South Orange, N. J. ALR
Miller, J. W., '29.....242 E. 19th, New York 3, N. Y. ALR
Miller, M. Valentine, '34
114 W. Phil-Ellena, Mt. Airy, Philadelphia 19, Pa. ALR
Miller, Meyer S., '44
213 Fitzpatrick bldg., Calgary, Alberta, Can. ALR
Miller, Ralph H., '39
6257 Rogers Park pl., Pleasant Ridge, Cincinnati 13, Ohio OP
Miller, Saul, '34.....88-05 150th st., Jamaica 2, N. Y. OP
Miller, Sidney N., '43
113 Hooker av., Poughkeepsie, N. Y. ALR
Miller, Theo. E., '31..First Trust bldg., Pasadena 1, Calif. ALR
Millett, Frank A., '33.....40 High, Greenfield, Mass. OALR

Milligan, Roy H., '36
607 N. Grand Blvd., Room 205, St. Louis 3, Mo. ALR
Mills, Chester R., '34
520 Commonwealth av., Boston 15, Mass. ALR
Mings, Dwain Edward, '46
1684 N. Prospect Ave., Milwaukee, Wis. OP
Minns, John Edward, '41....316 Michigan, Toledo 2, Ohio ALR
Minor, H. F., '18
Commerce Title bldg., Memphis 3, Tenn. OALR
Minsky, Armen A., '45
626 LaSalle bldg., Minneapolis, Minn. ALR
Minsky, Arthur, '43..8601 Homelawn st., Jamaica 3, N. Y. OP
Minsky, Henry, '26.....2 E. 95th, New York 28, N. Y. OP
Minton, Wm. H., '20....Corby bldg., St. Joseph 9, Mo. OALR
Mirow, David, '38.....237 E. 20th, New York 3, N. Y. ALR
Mishler, Donald L., '36
604 S. Cincinnati av., Tulsa 3, Okla. ALR
Missal, Sylvester Clemens, '46
2323 Prospect Ave., Cleveland 15, Ohio ALR
Mitchell, Douglas Gatlin, '46
703 Medical Arts bldg., Fort Worth, Tex. OP
Mitchell, E. K., '18
710 W. Lehigh av., Philadelphia 33, Pa. ALR
Mitchell, H. E., '30..14805 Detroit av., Cleveland 7, Ohio ALR
Mithoefer, W., '11.....Carew Tower, Cincinnati 2, Ohio ALR
Mittendorf, A. D., '12.399 Park av., New York 22, N. Y. OP
Mobley, Marion R., '39....125 W. Cheves st., Florence, S.C. ALR
Moehle, Walter, '32...15 Schermerhorn, Brooklyn 2, N. Y. OP
Moffat, Harold Forster, '42
1415 Kalakaua Ave., Honolulu, Hawaii OP
Moffett, Daniel B., '38
1150 Connecticut av. N.W., Washington 6, D. C. ALR
Mogan, Richard F., '44
523 West 6th st., Los Angeles 14, Calif. ALR
Mohun, Meade, '43.....23 Second av., San Mateo, Calif. ALR
Moll, Arthur M., '32
Powers Theatre bldg., Grand Rapids 2, Mich. ALR
Molsberry, Jasper Merl, '41
425 E. Wisconsin av., Milwaukee 2, Wis. OP
Molt, Wm. F., '11
Bankers Trust bldg., Indianapolis 4, Ind. ALR
Monaco, Louis, '43.
1100 Roosevelt bldg., Los Angeles 14, Calif. OALR
Monahan, J. A., '13.....181 Chestnut, Clinton, Mass. OALR
Monahan, James J., '35.....31 S. Jordin, Shenandoah, Pa. ALR
Monahan, Richard O'Brien, '44
378 W. Church St., Elmira, N.Y. ALR
Moncreiff, William F., '38..58 E. Washington, Chicago 2, Ill. OP
Mond, Edward, '40.....Eaton Tower, Detroit 26, Mich. ALR
Monette, C. J., '37.....814 Elm, Manchester, N. H. ALR
Monosmith, O. B., '05.....O. P. S. bldg., Lorain, Ohio OALR
Monsees, Wayne Emerson, '45..233 A St., San Diego 1, Cal. OP
Monserrate, Manuel Maria, '41
73 Main, Binghamton, N. Y. ALR
Monson, LaFayette, '34.450 Sutter, San Francisco 8, Calif. ALR
Monson, S. H., '10.B. F. Keith bldg., Cleveland 15, Ohio OALR
Montgomery, D. Cameron, '28
Weinberg bldg., Greenville, Miss. ALR
Montgomery, John L., '40.Medical Arts bldg., Knoxville, Tenn. OP
Moon, Seymour B., '36.....R. D. #2, Coraopolis, Pa. OP
Moore, Donald E., '43..713 East Genesee, Syracuse 2, N. Y. OP
Moore, Ellis Columbus, '41
First National Bank bldg., Olean, N. Y. ALR
Moore, George H., '19.101 Long Lane, Upper Darby, Pa. OALR
Moore, J. Hallock, '35
1229 1st Huntington Nat'l Bank bldg., Huntington 1, W. Va. ALR
Moore, James A., '41.....530 E. 90th, New York 28, N. Y. ALR
Moore, P. G., '30....Republic bldg., Cleveland 15, Ohio OP
Moore, Paul M., Jr., '30.Cleveland Clinic, Cleveland 6, Ohio ALR
Moore, Roy S., '28...State Tower bldg., Syracuse 2, N. Y. ALR
Moore, T. W., '01..1050 Fifth av., Huntington 1, W. Va. OP
Moore, Thomas Scott, '39.....107 N. 2nd, Niles, Mich. OP
Moore, Walter V., '37....1 Nevins st., Brooklyn 17, N. Y. OP
Moorhead, Robert L., '33..125 Remsen, Brooklyn 2, N. Y. ALR
Moran, Charles T., '37
1014 Heyburn bldg., Louisville, Ky. OP
Morgan, Alonzo Lloyd, '40.170 St. George, Toronto 5, Can. OP
Morgan, George E., '44..65 No. Madison, Pasadena 1, Calif. OP
Morgan, Harold E., '42.....314 E. 3rd st., Newton, Kan. OP
Morgan, Irving J., '39
Jenkins Arcade bldg., Pittsburgh 22, Pa. OP
Morgan, William A., '39
1835 Eye st. N.W., Washington 6, D. C. ALR
Morgana, Dante James, '41.....13 Main, Lockport, N. Y. OP
Moriarty, Richard W., '28
3 Mamaroneck Road, White Plains, N. Y. OALR
Moriconi, Albert F., '39....438 Hamilton av., Trenton, N.J. ALR
Morledge, Roy V., '33....Hart-Albin bldg., Billings, Mont. ALR
Morley, George A., '20....P.O. Box 452, Crookston, Minn. OP
Morman, William Daniel, '41
3507 Franklin av., St. Louis 6, Mo. ALR
Morris, A. G., '18.Medical Arts bldg., Rochester 7, N. Y. ALR
Morris, Francis O'Neil, '46
820 S. Painter av., Whittier, Calif. ALR
Morris, S. A., '41
405 N. Bedford dr., Beverly Hills, Calif. OP
Morrison, Frederick M., '37,
717 Church St., Lynchburg, Va. OP
Morrison, Geo. S., '44..308 W. 2nd st., Roswell, N. Mex. ALR
Morrison, James D., '43
238 Hart-Albin bldg., Billings, Mont. OP
Morrison, Wm. Howard, '38
Medical Arts bldg., Omaha 2, Nebr. OP
Morrison, W. R., '38....Hart-Albin bldg., Billings, Mont. OALR
Morrison, W. Wallace, '26
140 E. 54th St., New York 22, N.Y. ALR
Morrissett, Leslie E., '37..261 Lake ave., Greenwich, Conn. ALR
Morrone, Francis X., '43.117 S. 2nd av., Mt. Vernon, N. Y. ALR
Morse, Leo M., '45
25 E. Washington St., Suite 1523, Chicago, Ill. ALR
Morsman, L. Wm., '20..Morsman Clinic, Hibbing, Minn. OP
Morton, J. B., '24....90 E. Warren av., Detroit 1, Mich. OALR
Morwitz, Samuel M., '36.55 E. Washington, Chicago 2, Ill. ALR
Moser, G. P., '43.....455 Market St., Bloomsburg, Pa. ALR
Mosher, Harris P., '18....127 Front, Marblehead, Mass. ALR
Mosher, Henry Adams, '42
11 Beatrice Circle, Belmont 78, Mass. OP
Mosher, Walter F., '44....34 N. Ash st., Ventura, Calif. ALR
Moshinsky, Alex. L., '36..237 E. 20th, New York 3, N. Y. ALR
Moss, L. Conner, '37
2223 Observatory Place, N.W., Washington, D.C. OP
Moss, Olin W., '31.....Weber bldg., Lake Charles, La. ALR
Motley, Fred Elliot, '38...106 W. 7th, Charlotte 2, N. C. ALR
Mott, Walter C., '40..... 75 Willett, Albany 6, N. Y. OP
Motto, M. Paul, '29.....Rose bldg., Cleveland 15, Ohio OP
Moulton, Everett C., '15
Merchants Nat'l Bk. bldg., Ft. Smith, Ark. OALR
Moulton, H., 1900
Merchants Nat'l Bk. bldg., Ft. Smith, Ark. OALR
Mountain, Edward R., '42..128 S. Union st., Olean, N.Y. ALR
Mountjoy, Philip S., '44.....Corsicana, Tex. ALR

Mowry, Ben, '40.....First Nat'l Bk. bldg., Findlay, Ohio ALR
Moyer, John Sanford, '34
901 Washington av., Brooklyn 25, N.Y. ALR
Mueller, Lawrence W., '39
3423 S. Washington rd., Fort Wayne, Ind. ALR
Mueller, Werner, '37.....412 Beacon, Boston 15, Mass. ALR
Muirhead, Robert Mowatt, '45
8 East Broadway, Salt Lake City 1, Utah ALR
Muldoon, W. E., '36
Nix Professional bldg., San Antonio 5, Texas OP
Mullen, Carroll R., '37...2025 Locust, Philadelphia 3, Pa. OP
Mullen, Clifford J., '35
424 Brotherhood bldg., Kansas City 11, Kans. OP
Mulligan, Harold R., '37,
1680 N. Vine St., Hollywood 28, Calif. ALR
②Mulsow, Jack E., '20..Shorecrest Hotel, Milwaukee, Wis. OALR
Mumma, Claude S., '32
U. S. Veterans' Facility, Los Angeles 25, Calif. ALR
①Muncy, Wm. M., '14....162 Angell, Providence 6, R. I. OALR
①Mundt, G. Henry, '12
30 N. Michigan av., Chicago 2, Ill. OALR
Munoz-MacCormick, Carlos Eduardo, '45
Monterrey bldg., Park Terrace, Santurce, Puerto Rico ALR
Munson, F. T., '30.2201 E. Jefferson av., Detroit 7, Mich. ALR
Murphy, A. B., '30
Medical & Dental bldg., Everett, Wash. ALR
Murphy, Edward S., '36, 309 Eddy av., Missoula, Mont. OP
Murphy, Rex L., '37...Metropolitan bldg., Denver 2, Colo. ALR
Murphy, Wm. E., '34.....390 Main, Worcester 8, Mass. ALR
Murray, Henry J., '31.....53 South st., Stamford, Conn. ALR
Murtagh, John Anthony, '41
Hitchcock Clinic, Hanover, N. H. ALR
Muskat, Irving, '33.....25 E. Washington, Chicago 2, Ill. OALR
Mussun, W. G., '24.....Rose bldg., Cleveland 15, Ohio ALR
Myers, David, '36.....1834 Pine St., Philadelphia, Pa. ALR
Myers, Elmer A., '20,
1708 Hammond av., Superior, Wisc. OALR
Myers, E. Lee, '26.....3903 Olive, St. Louis 8, Mo. ALR
②Myers, John L., '20..Shukert bldg., Kansas City 6, Mo. OALR
Myers, Stanley A., '39
810 Dollar Bank bldg., Youngstown, Ohio ALR
Myerson, Mervin C., '23
416 N. Bedford Dr., Beverly Hills, Calif. ALR

## N

Nacey, Lawrence J., '44..221 Alexander, Rochester, N. Y. ALR
①Nadeau, E. G., '15.....Belin bldg., Green Bay, Wis. OALR
①Naftzger, J. B., '17
6777 Hollywood blvd., Hollywood 28, Calif. OALR
③Nager, Prof. F., '25..Freiestrasse 30, Zurich, Switzerland OALR
Nail, James B., '36..1300 Eighth st., Wichita Falls, Texas ALR
Nairn, George Waverly, '41, Wyoming County
Community Hospital, Warsaw, N.Y. OALR
①Nance, Willis O., '03...6845 Euclid av., Chicago 49, Ill. OALR
Nantz, Frank A., '45...225 Bodmann ave., Cincinnati, Ohio OP
Nash, C. Stewart, '29...277 Alexander, Rochester 7, N. Y. ALR
Nash, Herbert T., '37.....31 N. State, Chicago 2, Ill. ALR
Naugle, Thomas Calvert, '45
1200 12th St., N., Columbus, Miss. ALR
Nebinger, Reid, '33.....19 W. Market, Danville, Pa. ALR
Neblett, Herbert Clarence, '40
401 North Tryon, Charlotte 2, N. C. OP
Neely, Paul T., '36...Medical Arts bldg., Portland 5, Ore. ALR
②Neeper, Edward R., '18
Exch. Nat'l Bk. bldg., Colorado Springs, Colo. OP
Neer, Edmonde DeW., '36.115 E. 61st, New York 21, N. Y. ALR
Neff, E. Eugene, '32.224 W. Washington, Madison 3, Wis. OP
Neidlinger, Wm. J., '42
576 Farmington ave., Hartford 5, Conn. ALR
Neivert, Harry, '46.....555 Park Ave., New York, N.Y. ALR
Neller, Walter I., '33....121 Wickham, Middletown, N. Y. OP
②Nelson, Charles F., '19
Schofield bldg., Cleveland 15, Ohio OALR
Nelson, Fritz, '44 1121 N. Tejon, Colorado Springs, Colo. OP
Nelson, John Russell, '46
Wyoming Nat'l Bank bldg., Casper, Wyo. ALR
Nelson, Leo A., '30.....Box 28, Dallas 1, Texas ALR
②Nelson, Louis A., '18
Lowry Med. Arts bldg., St. Paul 2, Minn. OP
Nelson, Nathaniel F., '40.4910 15th av., Brooklyn 19, N.Y. OP
Nelson, Orville N., '36,
1008 First Federal bldg., St. Petersburg, Fla. OALR
Nemours, Paul R., '29
1506 Hodiamont av., St. Louis 12, Mo. ALR
Nerbonne, Joseph J., '39.....231 Main, Brockton 1, Mass. OALR
Nesbit, Mark E., '39
616 1st Nat'l Bank bldg., Madison 3, Wis. OP
Nesbit, Wellwood, '36.....1 S. Pinckney, Madison 3, Wis. ALR
Nethercut, G. W., '35...25 E. Washington, Chicago 2, Ill. OP
Nethercut, Ruth A., '38.450 Sutter, San Francisco 8, Calif. ALR
Neuberger, Julius Frederick, '42,
325 E. 57th St., New York 22, N. Y. ALR
Neumann, Wm. H., '28
Sheboygan Clinic, Sheboygan, Wis. OALR
Neuschatz, Gerald, '37
327 Central Park West, New York 25, N. Y. ALR
New, Gordon B., '25.....Mayo Clinic, Rochester, Minn. L-PI S
Newby, Harley D., '44...619½ Main, Rapid City, S. D. ALR
Newell, Frank W., '43..2605 Pittsfield bldg., Chicago 2, Ill. OP
Newlands, W. A., '34....359 East Main, Mt. Kisco, N. Y. ALR
and 19 S. Broadway, Tarrytown, N. Y.
Newman, Erwin W., '38...1606 Capitol av., Cheyenne, Wyo. OP
Newman, Saul C., '44..1082 Park Av., New York 28, N.Y. ALR
Newton, F. H., '25....Medical Arts bldg., Dallas 1, Texas OALR
Newton, Morris H., '34...Burrell bldg., Little Falls, N. Y. OP
Newton, Ray E., '43..1010 Reynolds bldg., Jackson, Mich. OP
Nicholson, Charles T., '45..422 Tyler Pl., Alexandria, Va. ALR
Nicholson, John Bixby, '46
401 Savings Bank Bldg., Marquette, Mich. ALR
Nippe, Leonard, '32..... 316 Michigan, Toledo 2, Ohio OALR
②Nisselson, Max, '18
250 Riverside drive, New York 25, N. Y. ALR
Nodine, Edwin Rogers, '42
1110 Bell bldg., Montgomery 4, Ala. ALR
Noe, Carl A., '41,....601 Higley bldg., Cedar Rapids, Ia. OALR
Noonan, Paul Ecret, '45....238 No. Park Av., Warren, Ohio ALR
Noonan, W. J., '25.....4753 Broadway, Chicago 40, Ill. OALR
Nordstrom, Chester A., '37
Exchange Bank bldg., Franklin, Pa. ALR
Norris, Charles Morgan, '46
3401 N. Broad St., Philadelphia 40, Pa. ALR
Norris, Mary Alice, '43
404 Hume-Mansur bldg., Indianapolis 4, Ind. OP
Northcott, Thomas A., '26.40 E. 61st, New York 21, N. Y. OP
Northington, Page, '35, Capt. (MC)....Address Unknown ALR
Norton, Arthur H., '31....1162 Willamette, Eugene, Ore. OP
Nova, Jules M., '33.....164 Pine St., Freeport, N.Y. OP

②Novak, Frank J., Jr., '19
30 N. Michigan av., Chicago 2, Ill. ALR
Novick, Joel N., '38
1726 Eye st. N.W., Washington 6, D. C. ALR
Noyes, Leighton Helmuth, '46
67 Main St., Brattleboro, Vt. ALR
Nugent, Maurice Wilfred, '42
1930 Wilshire blvd., Los Angeles 5, Calif. OP
Nugent, Oscar B., '30..231 W. Washington, Chicago 6, Ill. OP
①Nutter, C. F., '14.....16 Amherst, Nashua, N. H. OP
Nutting, Raymond J., '39..411 Thirtieth, Oakland 9, Calif. OP

## O

Oakes, Milton C., '44..24 No. Mulberry St., Mansfield, Ohio OP
Oaks, Lewis Weston, '40..33 East 2nd South, Provo, Utah OALR
①Oberg, Carl M., '10,
300 Physicians & Surg. bldg., Minneapolis 2, Minn. OALR
O'Brien, C. S., '24.....University Hosp., Iowa City, Iowa OP
O'Brien, Elmer W., '41.5 N. Goodman, Rochester 7, N. Y. ALR
O'Brien, G. R., '30
307 Sterling place, Brooklyn 17, N. Y. ALR-PI S
O'Brien, James Miles, '46
Dartmouth Eye Institute, Hanover, N.H. OP
①O'Brien, Steve A., '15,
1st Nat'l Bank bldg., Mason City, Iowa OALR
①O'Connor, D. F., '18
27 S. Kingman rd., South Orange, N.J. OALR
O'Connor, Philip D., '34.30 N. Michigan av., Chicago 2, Ill. OP
②O'Connor, Roderic P., '18.....411 30th, Oakland, Cal. OP
O'Connor, Thomas Philip, '42
30 N. Michigan av., Chicago 2, Ill. ALR
O'Connor, Timothy Francis, '40
4634 Main av., Ashtabula, Ohio ALR
O'Dell, John C., Jr., '37
Medical Arts bldg., 1022 Park st., Jacksonville 4, Fla. ALR
Odeneal, Thomas H., '29...Coker bldg., Winter Haven, Fla. OALR
Odom, Robert E., '38..378 Lora av., Youngstown 4, Ohio ALR
Odom, Stanley G., '34, Col. (MC)
Staten Island Area Station Hosp., Staten Island 6, N.Y. OALR
O'Donnell, Merrill Charles, '46
725 Jenkins Bldg., Pittsburgh, Pa. ALR
Offenbach, Bertha, '46
270 Commonwealth Ave., Boston, Mass. OP
Odgen, Frederick W., '44
130 S. Duncan St., Fayetteville, Ark. ALR
Ogden, J. C., '36..Professional bldg., Long Beach 2, Calif. OP
Ogus, Alfred, '43, Capt. (MC),
Army Air Base, Salina, Kan. ALR
Ohly, John H., '32....22 Schermerhorn, Brooklyn 2, N. Y. OP
Ohmart, Walter A., '36....Republic bldg., Denver 2, Colo. OP
O'Hora, James T., '37
571 Fisher bldg., Detroit 2, Mich. ALR
O'Kane, George H., '38
55 E. 86th St., New York 28, N. Y. ALR
O'Keefe, John Joseph, '46
255 S. 17th St., Philadelphia 3, Pa. ALR
Olkon, Dora B., '37
8015 Beverly blvd., Los Angeles 36, Calif. OP
Olmsted, K. Elizabeth Pierce, '44,
568 Lafayette av., Buffalo 13, N. Y. OP
Olsen, Arthur Roy, '46
1421 State St., Santa Barbara, Calif. ALR
②Olsho, S. L., '12.....235 S. 15th, Philadelphia 2, Pa. OALR
Olson, Archie, '41.....Medical Arts bldg., Duluth 2, Minn. OP
Olson, George W., '39.....1759 Fulton, Fresno 1, Calif. ALR
Opin, Edward, '38
2065 Grand Concourse, New York 53, N. Y. ALR
Oppegaard, C. L., '30..220 S. Broadway, Crookston, Minn. ALR
①Orcutt, D. C., '17....55 E. Washington, Chicago 2, Ill. OALR
O'Reilley, Bernard E., '37
641 Lowry, Med Arts Bldg., St. Paul 2, Minn. OP
①O'Reilly, W. F., '13.....44 S. Common, Lynn, Mass. OALR
Ornston, Darius G., '44,
36 Carpenter Lane, Philadelphia 19, Pa. ALR
O'Rourke, Carroll, '32.....604 W. Berry, Ft. Wayne, Ind. ALR
O'Rourke, D. H., '27.....Republic bldg., Denver 2, Colo. OP
Orr, Eugene, '32.....Doctors bldg., Nashville 3, Tenn. ALR
①Orton, Henry B., '17....224 Delevan av., Newark 4, N.J. ALR
Osherwitz, Morris Stanford, '45
456 Doctors bldg., Cincinnati 2, Ohio OP
Ostrom, Louis, '45.....124 18th St., Rock Island, Ill. ALR
Ostrom, William C., '39,
1350 Union, Schenectady 8, N. Y. OP
Ostrow, Irving, '44, 41 Hooker Ave., Poughkeepsie, N. Y. ALR
O'Toole, Francis Austin, '40..181 Chestnut, Clinton, Mass. ALR
Otrich, Grover C., '32....Commercial bldg., Belleville, Ill. ALR
①Overman, Frederick V., '11,
222 W. Jefferson, Tipton, Ind. ALR
Overton, J. Woodhull, '43....P.O. Box 293, Newburgh, N.Y. OALR
Owen, Walter E., '43...1242 Jefferson bldg., Peoria 2, Ill. ALR
Owens, Ella M. Uhler, '41
Johns Hopkins Hospital, Baltimore 5, Md. OP
Owens, Harold, '44..1052 W. 6th, Los Angeles 14, Calif. ALR
Owens, William Councilman, '46
Johns Hopkins Hospital, Baltimore 5, Md. OP
Owsley, Guy A., '41
Col. (M.C.), 214 N. High, Hartford City, Ind. ALR
Owsley, Robert W., '41
1136 W. 6th St., Los Angeles, Calif. ALR

## P

Pace, Errett, '30...City Savings Bank bldg., Paducah, Ky. ALR
Pachtman, Isadore, '37..Jenkins Arcade, Pittsburgh 22, Pa. OP
Packard, Louis A., '26.523 W. 6th, Los Angeles 14, Calif. OALR
Page, Donald Franklin, '46
Boulder-Colorado Sanitarium, Boulder, Colorado ALR
Page, John R., '36.....127 E. 62nd, New York 21, N. Y. ALR
Page, Robert O., '38....722 Nat'l Bank bldg., Lima, Ohio ALR
Pagliughi, John J., '41,
1915 Palisade av., Union City, N. J. ALR
Palevsky, Samuel N., '44, 22 E. Los Olivos St.,
Santa Barbara, Calif. ALR
Palmer, Arthur, '26
667 Madison av., New York 21, N. Y. ALR-PI S
Palmer, Bascom H., '28..Huntington bldg., Miami 32, Fla. OALR
Palmer, Bascom W., '36
804 Boston bldg., Salt Lake City 1, Utah OP
Palmer, F. E., '28.....123 N. 3rd St., Sterling, Colo. OALR
Palmer, Mahlon Phelps, '41.....715 Lake, Oak Park, Ill. ALR
Palmer, Milton A., '41, 18 Park Blvd., Lancaster, N. Y. OP
Pannabecker, Charles L., '44,
434 Jefferson bldg., Peoria 2, Ill. OP
Paradis, Charles Ovide, '45
625 Cleveland Ave., N.W., Canton, Ohio ALR
Park, M. Benjamin, '36.325 Ingraham bldg., Miami 32, Fla. OP
Parker, Harry Caunter, '45.....673 Broadway, Gary, Ind. ALR
Parker, Harry L., '31.....88 East av., Lockport, N. Y. ALR
Parker, Ray, '42.....U. S. Bank bldg., Johnstown, Pa. ALR

①Parker, Walter R., '03
David Whitney bldg., Detroit 26, Mich. OP
Parkinson, Harold O., '45
626 Flood bldg., San Francisco, Calif. ALR
Parrish, Robert E., '23
Medical Arts bldg., San Antonio 5, Texas OALR
Parry, Thayer L., '35.....328 Ohio Bldg., Akron 8, Ohio OALR
Parsell, Louis A., '35
Paulsen Med. & Dental bldg., Spokane 8, Wash. ALR
①Parsons, J. G., '14..Crookston Clinic, Crookston, Minn. OALR
③Parsons, Sir John Herbert, '36
Meads, Wigton Lane, Alwoodley, Leeds, England OP
Pastore, Peter Nicholas, '42
5503 Riverside Drive, Richmond 24, Va. ALR
Patch, Orien B., '43..2905 E. Superior St., Duluth, Minn. ALR
Paton, R. Townley, '36.927 Park av., New York 28, N. Y. OP
Paton, Walter M., '43
919 Medical Dental Bldg., Vancouver, B. C., Canada ALR
Pattee, George L., '43.612 Republic bldg., Denver 2, Colo. ALR
①Patterson, Ellen J., '17, (Retired)
504 Neville, Pittsburgh 13, Pa. ALR
③Patterson, Robert U., '33
5801 Roland Ave., Baltimore 10, Md. ALR
Patterson, Sam H., '44.....125 - 15th, Toledo 2, Ohio OP
Paul, Wm. C., '30...Medical Arts bldg., Tacoma 2, Wash. ALR
Pauley, Gerald E., '39,
9460 220 St., Queens Village, L. I., N. Y. ALR
Paulk, James R., '38.....Moultrie, Ga. OP
①Payne, A. L., '08.....119 S. Barstow, Eau Claire, Wis. OALR
Payne, Brittain F., '32
17 E. 72nd St., New York 21, N.Y. OP
Payne, Frank, '44...635 W. 165th, New York 32, N. Y. OP
Payne, R. J., '25...University Club bldg., St. Louis 3, Mo. ALR
Payne, V. LeRoy, '30
Simmons Nat'l bldg., Pine Bluff, Ark. ALR
Pearlman, Louis M., '39...53 E. 93rd, New York 28, N. Y. ALR
Pearlman, S. J., '24....55 E. Washington, Chicago 2, Ill. ALR
Pearson, George J., '43.....320 N. 3rd, Burlington, Iowa ALR
Pearson, Ovid, '37, 277 Alexander St., Rochester, N. Y. OALR
Peck, Howard Burgess, '46
106 S. Girard ave., Albuquerque, New Mexico OP
Peden, Barton Edward, '42.Stimson bldg., Seattle 1, Wash. ALR
Peele, James Clarendon, '46....Box 636, Goldsboro, N.C. ALR
②Peeler, C. N., '22.....106 W. 7th, Charlotte 2, N.C. OALR
Peer, Lyndon A., '31..15 Washington St., Newark 2, N.J. ALR-PIS
Peery, Vance Price, '29....Kinston Clinic, Kinston, N. C. ALR
Pegues, James Cary, '42..301 Howard st., Greenwood, Miss. ALR
Pellettiere, Edmund Victor, '42
1530 N. Damon av., Chicago 22, Ill. ALR
Peluse, Samuel, '41
1930 Wilshire blvd., Los Angeles 5, Calif. ALR
Pelz, Mort D., '29....607 N. Grand blvd., St. Louis, Mo. ALR
Pember, Aubrey H., '26
508 W. Milwaukee, Janesville, Wis. OALR
Pendexter, R. Stevens, '26
Farragut Med. bldg., Washington 6, D. C. OP
Penta, Arthur Quito, '46
1301 Union St., Schenectady, N.Y. ALR
Penta, John J., '43.....237 S. 5th St., Reading, Pa. ALR
Penta, Michael J., '39.....312 N. Fifth, Reading, Pa. ALR
Percy, Archibald Edward, '46
North Raguet & Frank aves., Lufkin, Tex. ALR
Perdue, James D., '34.....P.O. Box 514, Mobile 4, Ala. OP
Perera, Charles A., '36...70 E. 66th, New York 21, N. Y. OP
Perfect, Fred Rolleston, '46
1258 Penn. Ave., Wyomissing, Pa. ALR
Perkins, Edward Williamson, '46
312 Medical Arts Bldg., Richmond, Va. OP
Perkins, Osborn P., '39...121 E. 60th, New York 22, N. Y. OP
Perlman, Henry B., '41.....950 E. 59th, Chicago 37, Ill. ALR
Perritt, Richard A., '39
30 N. Michigan Ave., Chicago 2, Ill OP
Perrone, Joseph A., '39...Mercy Hosp., Pittsburgh 19, Pa. ALR
Perry, Claude S., '32, 40 South 3rd St., Columbus, Ohio OP
Persky, A. H., '30..1737 Chestnut st., Philadelphia 3, Pa. ALR
Peter, Beverly K., '43.....Beckley Hosp., Beckley, W. Va. ALR
Peterson, Joel A., '37
609 Lafayette Life bldg., Lafayette, Ind. ALR
Peterson, John C., '36....702 Sharp Bldg., Lincoln, Nebr. OP
Peterson, N. Vern, '37.....Highland Hall, Rye, N. Y. ALR
Peterson, Roy L., '44....218 Eastman bldg., Boise, Idaho ALR
Peterson, Wm. G., '36.....106 E. 13th, Ada, Okla. ALR
Petheram, Charles C., '31
Medical Arts bldg., Portland 5, Ore. ALR
Petix, Dominick Gilbert, '45
2615 Grand Concourse, Bronx, New York, N.Y. OP
Petruzzi, William Astor, '42
65 Pondfield Road, Bronxville, N. Y. ALR
Petti, George Hyacinth, '40, 4 Berkeley ave., Yonkers, N.Y. ALR
①Pfafflin, C. A., '03
Bankers Trust bldg., Indianapolis 4, Ind. OALR
Pfeiffer, Guy Oliver, '45.19-A South High St., Belleville, Ill. ALR
Pfeiffer, Raymond L., '35
635 W. 165th, New York 32, N. Y. OP
Pfingst, Harry A., '46..332 W. Broadway, Louisville 2, Ky. OP
Pfunder, Malcolm C., '38
Medical Arts bldg., Minneapolis 2, Minn. ALR
②Phelps, Kenneth A., '20
Medical Arts bldg., Minneapolis 2, Minn. OALR
Phillips, Earl H., '44
1704 Harrison Ave., Salt Lake City, Utah ALR
Phillips, James R., '43, 701 Winn Ave., Baton Rouge, La. OP
Phillips, James William, '41
Box 632, Newport News, Va. OP
Phillips, James Winston, '42..Hurst Clinic, Longview, Tex. ALR
Phillips, Josephine Dirion, '35
16130 Parkland Drive, Shaker Heights 20, Ohio OP
Phillips, Samuel A., '39....137 N. Eighth, Allentown, Pa. OP
Phillips, Warren Charles, '45.801 N. Second, Harrisburg, Pa. OP
Philpott, Ivan W., '40
806 Metropolitan bldg., Denver 2, Colo. ALR
Pickard, John C., '44..... 1200 Main st., Dubuque, Ia. ALR
Pierce, George W., '43...490 Post, San Francisco 2, Calif. PIS
Pierce, William W., '40,.....Lockport, N. Y. ALR
Piercy, Frederick F., '37
Home S. & L. bldg., Youngstown 3, Ohio ALR
Piercy, Robert Lee, '46
613 Home Savings & Loan bldg., Youngstown, Ohio ALR
Pierson, William M., '38
Medical Arts bldg., Wilmington 9, Del. ALR
Pike, Melvin H., '38.....Reinhart bldg., Midland, Mich. OALR
Pinkerton, F. J., '29, Young Hotel bldg., Honolulu 9, T.H. OALR
Pino, Ralph H., '35.David Whitney bldg., Detroit 26, Mich. OP
Pischel, D. K., '36....490 Post, San Francisco 2, Calif. OP
③Pischel, Kasper, '42..490 Post, San Francisco 2, Calif. OP
Piszkiewicz, Frank J., '38
6019 N. Menard Ave., Chicago 30, Ill. ALR
①Pitkin, C. E., '17, Carnegie Med. bldg., Cleveland 6, Ohio ALR
Pitkin, York Nones, '45
10515 Carnegie Av., Cleveland, Ohio ALR

**Pitman, Louis K., '44**
1749 Grand Concourse, New York 53, N. Y. **ALR**
**Pitman, Will D., '39**..First Nat'l Bank bldg., Pratt, Kans. **ALR**
**Pittman, John E., '37**
David Whitney bldg., Detroit 26, Mich. **OP**
**Place, E. Clifford, '36**....59 Livingston, Brooklyn 2, N. Y. **OP**
**Platt, E. S., '43**.....4 Hamilton Ave., Auburn, N. Y. **OP**
**Pliss, Aaron, '46**.....40 North St., Buffalo, N.Y. **OP**
**Plumer, John S., '28**.121 University pl., Pittsburgh 13, Pa. **OP**
**Pochert, Rolland C., '35**...Matthews bldg., Owosso, Mich. **OALR**
**Podboy, August J., '43**.....28 N. Queen St., York, Pa. **ALR**
**Pohl, Robert L., '39**
755 Paulsen Medical Dental bldg., Spokane, Wash. **ALR**
**Pohlman, David Alexander, '45**
595 E. Colorado St., Pasadena 1, Calif. **ALR**
**Poindexter, Frank Wilmore, '41**
Medical Arts bldg., Newport News, Va. **ALR**
**Poirier, George H., '31**.60 Bay State rd., Boston 15, Mass. **ALR**
**Polan, Charles M., 44**
1042 6th ave., Huntington 1, W. Va. **OP**
**Pollack, Samuel, '41**, 2909 W. Estes Ave., Chicago 45, Ill. **ALR**
**Pollock, Frederic J., '38**
104 S. Michigan ave., Chicago 3, Ill. **ALR**
**Pond, Casper Whittle, '38**...Carlson bldg., Pocatello, Idaho **ALR**
**Poos, E. E., '27**.....Fisher bldg., Detroit 2, Mich. **OALR**
**Pope, E. C., '30**....2021 Sixth av. N., Birmingham 3, Ala. **ALR**
**Popovich, Stephen John, '45**
1930 Wilshire blvd., Los Angeles 5, Calif. **OP**
**Porter, Byron H., '39**.....18 Bank Row, Pittsfield, Mass. **ALR**
**Porter, George LeRoy, '39**
602 W. University Ave., Urbana, Ill. **OP**
①**Porter, L. B., '13**.....199 Thayer, Providence 6, R. I. **OALR**
**Porter, Whitney C., '35**, 320 Republic Bldg., Denver 2, Colo. **OP**
**Portnoy, Samuel H., '43**
19 Garfield pl., Cincinnati 2, Ohio **ALR**
**Posner, Adolph, '36**, 667 Madison Ave., New York 21, N. Y. **OP**
②**Post, Lawrence T., '20**
Metropolitan bldg., St. Louis 3, Mo. **OP**
②**Post, M. Hayward, '18**
Metropolitan bldg., St. Louis 3, Mo. **OP**
**Post, Winfred L., '39**,....804 Frisco Bldg., Joplin, Mo. **OALR**
**Postle, C. D., '30**.....240 E. State, Columbus 15, Ohio **ALR**
**Potter, Allen B., '29**.Pasteur Med. bldg., St. Louis 8, Mo. **ALR**
**Potter, Jacob J., '35**.....Gas Electric bldg., Rockford, Ill. **ALR**
**Potter, W. A., '30**.7450 Grand River av., Detroit 4, Mich. **ALR**
**Potter, William Bently, '42**
303 Merimas bldg., Galveston, Tex. **OP**
**Potter, Wm. W. P., '27**..601 Walnut, Knoxville 30, Tenn. **OALR**
**Potts, Charles R., '44**, 1304 Walker av., Houston 2, Tex. **OP**
①**Potts, John B., '10**....3817 Dewey av., Omaha 5, Nebr. **ALR**
**Poulin, James E., '44**, Professional Bldg., Waterville, Me. **ALR**
**Powell, Archie C., '44**..... Stimson bldg., Seattle 1, Wash. **ALR**
**Powell, Lyle S., '28**....736 Kentucky St., Lawrence, Kans. **OALR**
①**Powers, Everett, '16**.....305 S. Main, Carthage, Mo. **OALR**
**Powers, Frank P., '37**. . .Professional bldg., Raleigh, N. C. **ALR**
**Powers, Harold W., '36**
705 National Reserve Bldg., Topeka, Kans. **ALR**
**Powers, John T. H., '38**....32 Pleasant st., Gardner, Mass. **ALR**
②**Prangen, Avery de H., '19**
Mayo Clinic, Rochester, Minn. **OP**
①**Pratt, Fred J., '12**
Physicians & Surgeons bldg., Minneapolis 2, Minn. **OALR**
**Preefer, Charles Joseph, '41**
195 Hempstead av., Rockville Centre, L. I., N. Y. **OP**
**Preefer, Raymond R., '44**
303 Gardenia st., West Palm Beach, Fla. **OP**
**Prendergast, John J., '38**.2001 4th av., San Diego 1, Calif. **OP**
**Presberg, Max Harold, '46**
35 Chestnut St., Rochester 4, N.Y. **OP**
**Presnell, C. A., '43**....634 N. Grand av., St. Louis 3, Mo. **ALR**
**Pressman, Joel J., '35**,
615 N. Faring Road, Los Angeles 24, Calif. **ALR**
**Preston, H. Grant, '30**
Professional bldg., Harrisonburg, Va. **ALR**
**Preston, William O., '46**
733 S. 44th St., Louisville, Ky. **ALR**
**Prewitt, Leland H., '40**.....207 E. 2nd St., Ottumwa, Ia. **ALR**
**Price, F. Raymond, '45**
118 Rutledge Ave., Charleston 16, S.C. **OP**
①**Price, Norman W., '08**, (Retired), Hyde Park blvd. &
Lewiston rd., Route 47, Niagara Falls, N. Y. **OALR**
**Priest, Robert E., '42**
302 Medical Arts bldg., Minneapolis, Minn. **ALR**
**Prindle, Ronald Edon, '46**, Capt. (MC)
20 Cortland St., Norwich N.Y. **ALR**
**Pritikin Roland I., '41**..1211 Talcott bldg., Rockford, Ill. **OP**
**Proctor, Bruce, '41**
1419 David Whitney bldg., Detroit 26, Mich. **ALR**
**Proctor, Donald F., '44**
Johns Hopkins Hosp., Baltimore 5, Md. **ALR**
**Proetz, A. W., '23**.....Beaumont bldg., St. Louis 8, Mo. **ALR**
**Prohovnik, Julius, '44**, 9 So. Kedzie av., Chicago, Ill. **ALR**
②**Prout, A. W., '18**.....380 E. Town, Columbus 15, Ohio **OALR**
**Provost, Pierre E., '41**.....48 Green St., Augusta, Maine **ALR**
**Pryor, Charles A., '37**
255 So. 17th St., Philadelphia 3, Pa. **ALR**
**Pryor, Will R., '37**
415 Heyburn Bldg., Louisville 2, Ky. **OP**
**Pugmire, Ralph W., '43**.2404 Washington bldg., Ogden, Utah **OP**
**Pullen, Edward Markey, '41**
30 E. 40th, New York 16, N. Y. **ALR**
**Pulliam, Robert Lee, Jr., '46**, Bodine-Contril Clinic,
1735 N. Wheeler av., Portland 12, Ore. **ALR**
**Pumphrey, Gordon H., '42**.100 N. Maine st., Mt. Vernon, O. **OP**
**Puntenney, Irving, '41**.720 N. Michigan av., Chicago 11, Ill. **OP**
**Putney, Floyd Johnson, '46**
255 S. 17th St., Philadelphia 3, Pa. **ALR**
①**Pyle, Wallace, '15**.15 Exchange pl., Jersey City 2, N. J. **OALR**

## Q

**Qualls, H. W., '43**.1635 Exchange bldg., Memphis 3, Tenn. **ALR**
**Quereau, James V., '44**....206 N. 5th st., Reading, Pa. **OP**
**Quincy, J. E., '27**.270 Commonwealth av., Boston 16, Mass. **ALR**
**Quinlan, Joseph D., '43**....44 Prichard, Fitchburg, Mass. **ALR**
**Quinn, Francis P., '39**,.....Roshek Bldg., Dubuque, Ia. **ALR**
**Quinn, John L., '39**.....401 Market, Steubenville, Ohio **ALR**
**Quinn, Lester H., '37**....4105 Live Oak, Dallas 5, Texas **OP**
**Quinn, Robert E., '41**
126 W. Main St., Chillicothe, Ohio **OP**
**Quiring, Walter O., '35**..100 First West, Hutchinson, Kan. **ALR**
①**Quittner, S. S., '16**.10515 Carnegie av., Cleveland 6, Ohio **OALR**

## R

**Rabbiner, Max, '36**, 358 New York av., Brooklyn 13, N. Y. **ALR**
**Rabinowitz, Paul, '38**..Mountain Sanitarium, Hamilton, Can. **ALR**
**Rafferty, George E., '38**.....173 N. Common, Lynn, Mass. **ALR**
**Raggio, Francis Warren, Jr., '45**
2416 Helen st., Lake Charles, La. **ALR**
**Rahmanop, Walter B., '39**...1426 Elm, Manchester, N. H. **ALR**

**Raines, M. M.,** '43.....226 S. C. St., Oxnard, Calif. **ALR**
①**Ralls, C. T.,** '16.....State Bank bldg., Winfield, Kan. **OALR**
**Ralston, Frank L.,** '33
120 E. Birch St., Walla Walla, Wash. **OALR**
①**Ralston, Wm. W.,** '18.1304 Walker av., Houston 2, Tex. **OP**
**Rambo, Victor Clough,** '46, Christian Hosp.
via Bilaspur B.N. Ry., Mungeli, Prov. India **OP**
**Ramm, Carl H., '44....508 Main st., Bennington, Vt. OP**
**Ramsay, Robert Matthews,** '42
1237 Wellington Crescent, Winnipeg, Can. **OP**
**Ramsey, G. Stuart,** '27....1496 Mountain, Montreal, Can. **OP**
**Randall, Hiram,** '28.....81 Main, Binghamton, N.Y. **ALR**
**Randel, Harvey O.,** '36
Medical Arts bldg., Oklahoma City 2, Okla. **OP**
**Randolph, M. Elliott,** '39..11 E. Chase St., Baltimore 2, Md. **OP**
**Rankin, Charles A.,** '38
Ludlow and Heather Road, Upper Darby, Pa. **OP**
**Rasgorshek, Robert Houston,** '46
425 Aquila Court, Omaha, Nebr. **OP**
**Raskin, Moses,** '40, The Madison,
817 St. Paul st., Baltimore 2, Md. **OALR**
**Rathgeber, Van D.,** '45
1305 Medical Arts bldg., Ft. Worth, Tex. **OP**
**Ratner, Simon H.,** '37..3401 Fifth av., Pittsburgh 13, Pa. **ALR**
②**Ravdin, B. D., '19.....Hulman bldg., Evansville 18, Ind. OALR**
①**Ravdin, M.,** '11, 1666 Bayard Pk. Dr., Evansville 14., Ind. **OALR**
**Ravin, Louis C., '43.....316 Michigan, Toledo 2, Ohio OP**
**Rawlins, Aubrey Gatliff,** '41
884 Post, San Francisco 8, Calif. **ALR**
**Rawlins, Grove A.,** '39.....206A West 3rd, Alton, Ill. **ALR**
**Ray, George H.,** '44, 914 Francis Bldg., Louisville, Ky. **OP**
**Ray, Harold E.,** '44.....Allen Bldg., Xenia, Ohio **OP**
**Ray, Joseph Carr,** '45....914 Francis bldg., Louisville, Ky. **ALR**
**Ray, Victor, Jr.,** '32.....Carew Tower, Cincinnati 2, Ohio **OP**
①**Rector, A. E., '15.....Zuelke bldg., Appleton, Wis. OALR**
**Reed, Charles L., '33...Jenkins Arcade, Pittsburgh 22, Pa. OP**
**Reed, Elmer McCready,** '42
2835 4th av., Beaver Falls, Pa. **ALR**
**Reed, James R.,** '37
201 N. El Molino av., Pasadena 4, Calif. **OP**
**Reed, Paul Henry,** '46
727 W. 7th St., Los Angeles 14, Calif. **OP**
**Reed, Wm. W.,** '30.....Mills bldg., Topeka, Kan. **ALR**
**Reeder, James E., Jr.,** '42
523 Davidson bldg., Sioux City 13, Ia. **OP**
①**Reeder, J. E.,** '16....Davidson bldg., Sioux City 13, Iowa **OALR**
**Reeh, Merrill J.,** '43
640 S. Kingshighway, St. Louis 10, Mo. **OP**
**Reel, Frederick Clifton,** '46
432 Medical Arts bldg., Charleston 1, W. Va. **ALR**
**Rees, Charles W., '38.....4496 Trias, San Diego 3, Calif. ALR**
**Reese, Algernon B.,** '28...73 E. 71st, New York 21, N. Y. **OP**
**Reese, Frederic Miller,** '46
Professional bldg., 330 N. Charles, Baltimore, Md. **OP**
②**Reese, Warren S., '21...1901 Walnut, Philadelphia 3, Pa. OP**
**Reeves, J. Norman,** '44....121 Main st., Western Port, Md. **ALR**
**Regan, James J.,** '32
520 Commonwealth Ave., Boston 15, Mass. **OP**
**Regan, William F., '37.19 Bay State road, Boston 15, Mass. ALR**
②**Reger, Scott N.,** '46..University Hospitals, Iowa City, Ia.
②**Reiche, O. C., '18.....Markle Bank bldg., Hazleton Pa. OP**
**Reid, Frederick Kessinger,** '46
119 Manhattan Ave., Crestwood 7, N.Y. **OP**
**Reid, H. W., '30...Union Central bldg., Cincinnati 2, Ohio OP**
**Reid, Thomas F., '40.....538 Central av., Dover, N. H. ALR**
**Reid, Wesley Grattan,** '46
974 Fisher bldg., Detroit, Mich. **OP**
**Reifsneider, J. Stanley,** '38..Beacon bldg., Wichita 2, Kan. **ALR**
②**Reim, Hugo,** '20....Metropolitan bldg., St. Louis 3, Mo. **OP**
**Reimers, R. S.,** '35....613 Eighth st., Fort Madison, Iowa **ALR**
**Rein, Walter John,** '45
501 E. Franklin St., Richmond 19, Va. **OP**
**Reineke, George F.,** '26...Fritsche bldg., New Ulm, Minn. **OALR**
②**Repass, R. E.,** '20....924 N. E. 78th St., Miami 38, Fla. **ALR**
**Reuling, F. H., '24.....Black bldg., Waterloo, Iowa OALR**
**Reuter, Carl H.,** '39
First Nat'l Bank bldg., Springfield, Ohio **OP**
**Reynolds, Edna M., '37..227 Sixteenth st., Denver 2, Colo. OP**
①**Reynolds, H. G.,** '09
Citizens Sav. Bank bldg., Paducah, Ky. **OALR**
**Rhode, Homer J.,** '38.....220 N. 6th, Reading, Pa. **OP**
**Rhode, Solon L.,** '28
P. O. Box 1385, Santa Barbara, Calif. **OALR**
**Rhodes, David H., '37.....Clark bldg., Pittsburgh 22, Pa. OP**
**Ricchiuti, A. George,** '41
316 E. Centre St., Mahanoy City, Pa. **ALR**
**Rice, Carl E., '38....6500 Maple av., Chevy Chase 15, Md. OP**
**Rice, John E.,** '24.....390 Main, Worcester 8, Mass. **OALR**
**Rice, Theodore A.,** '46.....390 Main St., Worcester, Mass. **OP**
**Richards, Lyman G.,** '24..1101 Beacon st., Brookline, Mass. **ALR**
**Richardson, George A.,** '38
1532 Overington, Philadelphia 24, Pa. **ALR**
**Richardson, J. J.,** '29
Cook Hosp. and Beal Clinic, Ft. Worth 3, Texas **ALR**
**Richardson, John R.,** '37
403 Commonwealth av., Boston 15, Mass. **ALR**
**Richardson, John Roberts,** '40
722 DuPont Bldg., Miami, Fla. **ALR**
**Richardson, Shaler,** '25.111 W. Adams, Jacksonville 2, Fla. **OP**
①**Richie, L. T.,** '18.....101 E. Main, Trinidad, Colo. **OALR**
**Richman, Frances B.,** '35, Lt. Comdr. (MC)
U. S. Naval Hospital, Long Beach 4, Calif. **OP**
**Richter, Harry Joseph,** '40
604 2nd Nat'l. Bank Bldg., Saginaw, Mich. **ALR**
**Ricketts, Floyd Blythe,** '41.....Address Unknown
**Ridall, Earle G.,** '41
91 Durland Ave., Elmira, N. Y. **ALR**
**Riddell, Malcolm M.,** '35.....1123½ Front, Fremont, Ohio **ALR**
**Ridout, George B., '40, 1836 South Ave., La Crosse, Wis. ALR**
①**Ridpath, Robert F.,** '14
1737 Chestnut st., Philadelphia 3, Pa. **LR**
**Rieger, W. H.,** '24.....Hanna bldg., Cleveland 15, Ohio **ALR**
**Riesenbeck, Leo Henry,** '45
831 Carew Tower, Cincinnati, Ohio **ALR**
**Rigby, Ralph G.,** '44
837 Boston Bldg., Salt Lake City, Utah **ALR**
**Rigg, J. P.,** '30.....Box 928, Grand Junction, Colo. **OALR**
**Riggs, Ralph H.,** '42
Medical Arts bldg., Shreveport, La. **ALR**
**Rigley, Edward L.,** '34
408 Sherland bldg., South Bend 9, Ind. **ALR**
**Riker, Aaron D., '34.....Riker bldg., Pontiac 15, Mich. OP**
**Rimer, Frank H.,** '35....Jenkins Arcade, Pittsburgh 22, Pa. **ALR**
①**Rindlaub, Elizabeth P.,** '10
321 Eighth st. S., Fargo, N. D. **OALR**
①**Rindlaub, John H.,** '06..321 Eighth st. S., Fargo, N. D. **OALR**
**Ringer, Merritt Gartley,** '46, Col. (MC)
Wm. Beaumont Gen. Hosp., El Paso, Tex. **ALR**
①**Ringle, Charles A.,** '07
c/o John Blackmore, La Junta, Colo. **ALR**
**Ripsteen, Joel Stanley,** '45..2527 Etna st., Berkeley, Calif. **ALR**

Risch, Otto Carl, '35....104 E. 40th, New York 16, N.Y. ALR
Risdon, E. F., '24.Medical Arts bldg., Toronto 5, Can. ALR-PI S
Riseman, Benjamin, '40..41 Bay State rd., Boston 15, Mass. ALR
Riser, Roy Otis, '38.....120 Main, Park Ridge, Ill. OP
Risser, E. D., '28.423 First Trust bldg., Pasadena 1, Calif. OALR
Riter, Kersey C., '43
The Budge Clinic, 3 North Main, Logan, Utah ALR
Rittenhouse, Emory A., '44
203 Masonic Temple, McKeesport, Pa. ALR
Riwchun, Meyer H., '34....510 Linwood av., Buffalo, N.Y. OP
Rizzuti, A. Benedict, '42..160 Henry st., Brooklyn 2, N Y OP
Robb, James M., '37
David Whitney bldg., Detroit 26, Mich. ALR
Robb, Mayo, '29.....Professional bldg., Phoenix, Ariz. ALR
Robbins, Clarence Edward, '41
111 S. Huron av., Pierre, S. D. ALR
Robert, Joseph X., '36.....170 St. George, Toronto, Can. ALR
Roberts, A. Warren, '37.....209 Pine, Texarkana, Texas OALR
Roberts, Gilbert Jay, '42 .586 N. Main st., Pomona, Calif. ALR
Roberts, John L., '39....316 Michigan st., Toledo, Ohio OP
Roberts, Joseph D. Jr., '37..204 N. Green, Longview, Tex. ALR
①Roberts, S. E., '16.Professional bldg., Kansas City 6, Mo. ALR
Roberts, William H., '03, (Retired)
335 Westbourne St., La Jolla, Calif. OALR
Robertson, E. Norris, Jr., '43
301 Northwest 12th, Oklahoma City, Okla. OP
②Robertson, Edward N., '18
New Caldwell bldg., Concordia, Kan. OALR
Robison, J. M., '43....1304 Walker av., Houston 2, Texas ALR
Robison, J. T., '30
900 Capital Nat'l Bank bldg., Austin 16, Tex. ALR
Rock, John Emmett, '40....Putnam bldg., Davenport, Iowa ALR
Rodenberg, Elmer Julius, '46.319 N. Elm St., Henderson, Ky. ALR
Rodin, Frank H., '37....490 Post, San Francisco 2, Calif. OP
Roemaet, Charles M., '37.....Address Unknown ALR
①Rogers, James T., '12..Medical Arts bldg., Montreal, Can. ALR
Rogers, John B., '38..1401 S. Hope, Los Angeles 15, Calif. OP
Roh, Robert F., '37.....727 Glen Ave., Westfield, N.J. ALR
Rohm, Jack Zeth, '45..7002 Jenkins Arcade, Pittsburgh, Pa. ALR
Rohm, Robert Franklin, '42
7002 Jenkins Arcade, Pittsburgh 22, Pa. OP
Rolett, D. M., '37.....876 Park av., New York 21, N. Y. OP
Roling, Edward A., '38.2753 W. North av., Chicago 47, Ill. OP
Roman, Stanley John, '45
742 Maccabees bldg., Detroit 2, Mich. OALR
Rome, Sol, '41....1917 Wilshire blvd., Los Angeles 5, Calif. OP
②Rommel, John C., '20, 4601 N. Broad,
Philadelphia 40, Pa. OP
Romonek, Philip L., '29
415 N. Camden Dr., Beverly Hills, Calif. OALR
Rones, Benjamin, '35
1610 Eye st. N. W., Washington 6, D. C. OP
Ronis, B. J., '43....7141 Elmwood Ave., Philadelphia, Pa. ALR
②Rooker, A. M., '20..United bldg., Niagara Falls, N. Y. ALR
Roper, Kenneth L., '42
58 E. Washington St., Chicago 2, Ill. OP
Rosedale, Raymond Samuel, '42
610 N. Market av., Canton 2, Ohio ALR
Rosen, David M., '43..19 W. 55th st., New York 19, N. Y. ALR
Rosen, Emanuel, '43.....692 High st., Newark, N.J. OP
Rosen, Sam, '37.....101 E. 73rd, New York 21, N. Y. ALR
Rosenbaum, Harry D., '39
7310 Cornell, University City 5, Mo. OP
①Rosenbaum, J., '15
1396 St. Catherine's st. W., Montreal, Can. OP
Rosenberg, Joseph, '36.....333 Linwood, Buffalo 9, N. Y. ALR
Rosenberg, William, '45
Rm. 1523, 25 E. Washington St., Chicago 2, Ill. OP
Rosenberger, H. C., '29
10515 Carnegie av., Cleveland 6, Ohio ALR
Rosenberger, Henry P., '36
221 Fifth st., Bismarck, N. D. ALR
Rosenbluth, Michael, '31
225 West 106th, New York 25, N. Y. ALR
Rosenthal, Benjamin Charles, '40
140 New York av., Brooklyn 16, N. Y. OP
Rosenwasser, Harry, '36
101 E. 73rd st., New York 21, N. Y. ALR
Ross, Cecelia, '41..710 Wilshire blvd., Santa Monica, Calif. OP
Ross, Ervin Scott, '46....477 Riddle Rd., Cincinnati, Ohio ALR
Ross, John, '36.....6 E. 70th, New York 21, N. Y. ALR
also 37 Oriole av., Bronxville, N. Y.
Ross, Percy J., '39.....104 S. Michigan Ave., Chicago, Ill. ALR
Ross, Perry W., '45....5426 N. Sheridan rd., Chicago, Ill. OP
Rossman, Morris M., '40
115 North Main St., Freeport, N. Y. ALR
Roszell, Leo H., '44..131 Fulton ave., Hempstead, L.I., N.Y. OP
Roth, Aaron, '46.....184 Joralemon st., Brooklyn, N.Y. ALR
Roth, Leslie William, '46
203 Commercial bldg., Belleville, Ill. OP
Rothberg, Maurice, '40
712 Wayne Pharm. Bldg., Ft. Wayne, Ind. OP
Rothenberger, Marvin K., '43.206 N. 8th st., Allentown, Pa. ALR
②Rothschild, Harold J., '20
Lowry Medical bldg., St. Paul 2, Minn. OALR
Rothstein, David Louis, '46
57 W. 57th St. New York 19, N.Y. ALR
①Rott, O. M., '15
1828 W. 1st av., Apt. C, Spokane 9, Wash. OALR
Rouse, H. K., Jr., '41,
McWilliams Clinic, Box 174, Gulfport, Miss. OP
Row, D. Hamilton, '35..23 E. Ohio st., Indianapolis, Ind. OP
Rowen, Ernest Henry, '40.....Box 126, Miles City, Mont. ALR
Rowland, Ivo E., '38.....5th and McKean, Charleroi, Pa. ALR
Rowland, W. D., '23
180 Commonwealth av., Boston 16, Mass. OP
Royals, Walter C., '38, Col. (MC)
Chief, EENT Sec., Oliver Gen. Hosp., Augusta, Ga. OP
Royce, Thomas L., '45.....904 Holman, Houston, Tex. ALR
Royer, Charles A., '43,
717 Medical Arts bldg., Oklahoma City, Okla. OP
①Rubendall, C., '15.Medical Arts bldg., Omaha 2, Nebr. OALR
Rubenstein, A. Harry, '30
Medical Arts bldg., Syracuse 2, N. Y. ALR
Rubin, Henry J., '43
8667½ Burton Way, Los Angeles 36, Cal. ALR
Rubin, Herman G., '43,..159 S. Main St., Akron, Ohio ALR
Rubin, Milton R., '40
504 Broadway, Suite 725, Gary, Ind. ALR
Rubin, Nathan S., '36
404-407 Blount Bldg., Pensacola, Fla. OP
Rubright, LeRoy W., '37.....700 State st., Alton, Ill. ALR
①Ruby, F. McK., '08.....235 W. Pearl, Union City, Ind. OALR
Ruchman, Jacob, '41..301 Brooklyn av., Brooklyn 13, N. Y. ALR
Rucker, C. W., '29.....Mayo Clinic, Rochester, Minn. OP
①Rucker, E. W., Jr., '16
Woodward bldg., Birmingham 3, Ala. ALR
Rucker, Ralph Weller, '45
Union National Bank bldg., Bartlesville, Okla. ALR
Rudolph, Carl J., '39,
2016 East Madison St., South Bend, Ind. OP

**Rudolph, Edward A., '42.624 Citizens bldg., Aberdeen, S. D. OP**
**Rudolphy, J. B., '29.....230 S. 21st, Philadelphia 3, Pa. OP**
**Ruedemann, A. D., '29**
Cleveland Clinic, Cleveland 6, Ohio OP
**Ruehl, William W., '45**
7106 Jenkins Arcade, Pittsburgh 22, Pa. ALR
**Ruggeri, Charles Jr., '44**
1120 Boston bldg., Salt Lake City 1, Utah. OP
**Runk, R. L., '29, Washington Sanitarium**
and Hospital, Tacoma Park, Washington 12, D. C. ALR
**Rusk, Harvey Schenck, '40, Colorado bldg., Pueblo, Colo. OALR**
**Russell, Daniel A.. '36**
Medical Arts bldg., San Antonio 5, Texas ALR
③**Russell, J. A. Merle, '22.....813 Sassafras, Erie, Pa. OALR**
**Russell, Martin V., '42...200 W. Elm st., El Dorado, Ark. ALR**
**Russell, R. E., '24....803 Waterloo bldg., Waterloo, Iowa OALR**
**Russell, Ralph Edward, '44**
1206 E. Ocklawaha ave., Ocala, Fla. OP
**Russell, Roland D., '31**
Room 808, Professional bldg., Elgin, Ill. ALR
**Russell, Sidney Benteen, '42**
Midelfart Clinic, Eau Claire, Wis OALR
**Rutherford, C. W., '25**
4601 N. Pennsylvania st., Indianapolis 5, Ind. OP
**Rutterberg, Max, '39.....303 Cooper, Camden, N. J. ALR**
**Ryall, George W., '37, 17010 Kinsman Road,**
Shaker Heights, Cleveland 20, Ohio ALR
**Ryan, Edward P., '40.....266 Glen St., Glen Falls, N.Y. OP**
**Ryan, L. M.. '27.....Wall bldg., St. Louis 8, Mo. ALR**
**Ryan, Maurice J., '40,**
2016 Elizabeth Ave., Kansas City 2, Kans. ALR
**Ryan, M. D., '36....660 Madison av., New York 21, N. Y. ALR**
**Ryan, Michael J., '44,**
310 Huron Bldg., Kansas City 10, Kans. ALR
**Rychener, Ralph O., '28...130 Madison, Memphis 3, Tenn. OP**
①**Ryder, D. R., '11 .....151 Rock, Fall River, Mass. ALR**
**Ryder, William H., '43**
195 Church St., New Haven 10, Conn. OP
**Ryerson, F. L., '25.....Fine Arts bldg., Detroit 26, Mich. OALR**

## S

②**Sacco, Anthony G., '20**
2200 New York av., Union City, N. J. OALR
**Sacheroff, Louis. '43....1288 Hird St., Lakewood 7, Ohio ALR**
**Sachs, Benjamin, '35.....520 Beacon, Boston 15, Mass. OP**
**Sackin, David, '33**
St. Moritz, 50 Central Pk. S., New York 19, N. Y. ALR
**Sadoff, Irvin E., '43....163 New st., New Brunswick, N.J. ALR**
**Safer, Louis A., '39**
Provident Bank bldg., Cincinnati 2, Ohio ALR
**Sage, Harry M., '37**
2194 N. Parkway Dr., Columbus 8, Ohio OP
**Sage, Russell A., '36..505 23 E. Ohio, Indianapolis 4, Ind. ALR**
**Sage, Thomas, '46**
7815 E. Jefferson Ave., Detroit, Mich. ALR
**St. John, Dewey, '40..Metropolitan bldg., St. Louis 3, Mo. ALR**
**Sakler, Barnet R., '39....Doctors bldg., Cincinnati 2, Ohio OP**
**Sale, George Goble, '46..216 Dixon bldg., Missoula, Mont. ALR**
②**Salinger, Samuel, '20..25 E. Washington, Chicago 2, Ill. ALR**
③**Sallander, F. W., '19.....212 W. Main, Visalia, Calif. OALR**
**Sails, Frederick H., '41..132 Union av., Framingham, Mass. OP**
**Salmon, David L., '40, Major (M.C.)**
Noel Ave., Madisonville, Ky. ALR
**Saltzman, Samuel L., '39.334 W. 86th, New York 24, N. Y. OP**
**Samponaro, Nicholas, '43.231 Calhoun st., Torrington, Conn. ALR**
**Samuels, Bernard, '29....57 W. 57th, New York 19, N. Y. OP**
**Sanders, J. Gillis, '29**
714 Merchants Nat'l Bank bldg., Mobile 11, Ala. ALR
**Sanders, John Lewis, '41.....222 N. Main, Greenville, S. C. ALR**
**Sanders, Sam H., '32..1089 Madison av., Memphis 3, Tenn. ALR**
**Sanders, Theodore E., '38**
Metropolitan bldg., St. Louis 3, Mo. OP
①**Sanderson, David D., '17..Stuart bldg., Lincoln 8, Nebr. OALR**
**Sandt, Karl E., '40**
1750 Medical Arts Bldg., Minneapolis 2, Minn. OP
**Sannella, Lee George, '45..124 Waterman, Providence 6, R.I. OP**
**Sanor, Daniel G.. '28....206 E. State, Columbus 15, Ohio ALR**
**Sarno, J. Frederick, '42**
109 S. William st., Johnstown, N. Y. ALR
**Sarnoff, Jack, '41...31-11 31st av., Long Island City, N.Y. ALR**
**Saskin, Edward, '42..855 St. Marks av., Brooklyn 13, N. Y. OP**
**Saslaw, Lewis Benjamin, '41**
Bank of America bldg., Stockton 5, Calif. ALR
**Sauer, John J., '43....30 E. 40th st., New York 16, N. Y. OP**
①**Sauer, W. E., '04.Beaumont Med. bldg., St. Louis 8, Mo. ALR**
①**Sauer, W. W., '10.....231 Third st., Marietta, Ohio OALR**
**Saunders, Truman L., '36.103 E. 65th, New York 21, N. Y. ALR**
**Savery, C. E., '28...230 Sherland bldg., South Bend 9, Ind. OALR**
**Savitt, Louis, '40.....3403 Lawrence av., Chicago 25, Ill. ALR**
**Sawyer, Leroy L., Jr., '34**
1746 K St., N.W., Washington 6, D.C. ALR
**Sayad, William Y., '32**
Harvey bldg., West Palm Beach, Fla. OALR
**Scal, J. Coleman, '26..10 E. 96th st., New York 28, N. Y. ALR**
①**Scales, J. L., '15**
Medical Arts bldg., Shreveport 45, La. OALR
**Scales, Wm. McVey, '41, 100 N. Main, Hutchinson, Kans. OP**
**Scanio, Joseph Charles, '40**
333 Linwood av., Buffalo 9, N. Y. ALR
**Scanlon, Wilko Gruver, '42**
201 N. El Molino av., Pasadena 4, Calif. ALR
②**Scarlett, Hunter W., '19, 825 Montgomery av.,**
Bryn Mawr, Pa. OP
**Scarney, Herman D., '33**
573 Fisher Bldg., Detroit 2, Mich., OP
**Schachat, Walter S., '45..30 E. 60th st., New York 22, N.Y. OP**
**Schall, LeRoy A., '27....243 Charles st., Boston 14, Mass. ALR**
①**Schappert, N. Louis, '16**
59 S. Washington, Wilkes Barre, Pa. OALR
**Schattner, Alfred, '44..115 E. 61st St., New York, N. Y. ALR**
**Schechter, Solomon P., '35**
1749 Grand Concourse, New York 53, N. Y. ALR
**Schechtman, Charles T., '37**
81 W. Main, New Britain, Conn. ALR
**Scheer, Henry M., '32**
522 West End av., New York 24, N. Y. ALR
**Scheffler, Milton Morris, '43**
55 E. Washington st., Chicago 2, Ill. OP
**Scheie, Harold G., '41**
313 S. 17th St., Philadelphia 3, Pa. OP
②**Schenck, C. P., '20**
Med. Arts bldg., Fort Worth 2, Texas OALR
**Schenck, Harry P., '37**
1912 Spruce St., Philadelphia 3, Pa. ALR
**Scheribel, Karl J., '39....55 E. Washington, Chicago 2, Ill. OP**
**Schiefelbein, Harry T., '31**
Stevens Clinic Hosp., Welch, W. Va. OP
**Schillinger, Raphael, '34..858 Summit Ave., Westfield, N.J. ALR**
**Schirack, C. J., '36.817 Cleveland av. N.W., Canton 3, Ohio ALR**
②**Schlanser, A. E., '21, Col. (M.C.)**
2245 Mt. View Place S. E., Washington, D. C. OALR

Schlessel, Louis, '39....101 E. Main st., Middletown, N.Y. ALR
Schlivek, Kaufman, '25.1016 Fifth av., New York 28, N. Y. OP
Schluer, Elmer P., '30
7317 Stanford Ave., University City 5, Mo. ALR
Schmidt, Herbert G., '34
411 E. Mason st., Milwaukee 2, Wis. OALR
Schmidt, James M., '37..40 S. Oxford, Brooklyn 17, N. Y. ALR
Schmidt, Otto V. M., '32.568 Park av., New York 21, N. Y. ALR
Schmidtke, Reinhardt L., '46
141 Lowry Med. Arts bldg., St. Paul 2, Minn. OP
Schnebly, John Thomas, '41
900 17th St., N. W., Washington 6, D. C. OP
Schneck, Nathan, '31, Rock Is. Bank bldg., Rock Island, Ill. ALR
Scholz, Roy O., '43
1515 Pentridge Rd., Baltimore 12, Md. OP
①Scholz, R. P., '16...Metropolitan bldg., St. Louis 3, Mo. ALR
Schoofs, O. P., '36
324 E. Wisconsin av., Milwaukee 2, Wis. ALR
Schoolman, Joseph G., '42,
104 S. Michigan, Chicago 3, Ill. ALR
Schrimpf, Cyril Edward, '41
229 Doctors Bldg., Cincinnati, Ohio OP
Schroeder, Milo Carl, '40,
2112 Colorado blvd., Los Angeles 41, Calif. ALR
Schubert, Clarence K., '33
1 S. Pinckney, Madison 3, Wis. OP
Schugt, Henry P., '33..122 E. 76th, New York 21, N. Y. ALR
Schultz, Abraham, '40...30 N. Michigan ave., Chicago, Ill. OP
Schultz, Jacob F., '39, 1304 Walker av., Houston, Texas OP
Schuster, Frank P., '27
1st Nat'l Bank bldg., El Paso, Texas OALR
Schuster, S. A., '24..1st Nat'l Bank bldg., El Paso, Texas OALR
Schutz, Joseph A.. '40..2804 Main st., Buffalo 14, N. Y. OP
Schutz, Sigmund, '44
105 West 55th St., New York, N. Y. OP
Schwartz, A. A., '27.....7 W. 71st, New York 23, N. Y. ALR
①Schwartz, Frederick O., '19, 508 N. Grand blvd.,
St. Louis 3, Mo., and 230 E. Main, Belleville, Ill. OP
Schwartz, Herbert Norman, '46, 99 Pratt St., Hartford, Conn. OP
Schwartz, Hugh W., '39
183 S. Park, Halifax, Nova Scotia ALR
Schwartz, Leo, '35.....136 E. 57th, New York 22, N. Y. ALR
Schwartz, V. J., '29 Med. Arts bldg., Minneapolis 2, Minn. ALR
Schwartz, William A., '28
320 Professional bldg., Phoenix, Ariz. OALR
Schwarz, Gerald T., '43
10515 Carnegie av., Cleveland 6, Ohio OP
Sciarretta, Sylvio A., '31.55 E. Washington, Chicago 2, Ill. ALR
Scobee, J. Edwin, '40,
6253 Hollywood blvd., Los Angeles 28, Calif. ALR
Scobee, Richard G., '43
640 S. Kingshighway, St. Louis 10, Mo. OP
Scovis, I. John, '45
U. S. National Bank bldg., The Dalles, Ore. OP
Scurti, Bernard V., '43, Lt. (M.C.),
51 Harmon st., Brooklyn 21, N. Y. ALR
①Searcy, H. B., '12.....2501 Sixth st., Tuscaloosa, Ala. OALR
Secoy, Harry R., '35.....Central bldg., Everett, Wash. ALR
①Sedwick, W. A., '16.....950 Pearl st., Denver 3, Colo. OP
Seeley, Ralph Hunt, Jr., '46..79 Center St., Rutland, Vt. ALR
Seeley, Robert Charles, '46.133 E. 58th St., New York, N.Y. ALR
Seelig, Charles Arthur, '40
104 E. 40th St., New York 16, N.Y. ALR
Segal, Samuel, Jr., '33....111 Maple st., Springfield, Mass. ALR
Seip, Walter R., '43, Professional Bldg., Pittsburgh, Pa. OP
Seitz, Howard M., '46....Coronado bldg., Santa Fe, N.M. ALR
Seletz, Abraham, '36.Med. Arts bldg., Charleston 1, W. Va. ALR
①Selfridge, G. L., '08
1000 California st., San Francisco 8, Calif. ALR
Seligman, Albert E., '39
41 Forest av., Staten Island 1, N. Y. ALR
Seligman, Maurice, '46
405 N. Bedford Drive, Beverly Hills, Calif. ALR
Seligstein, Milton B., '26
Union Planters Bank bldg., Memphis 3, Tenn. OALR
Selinger, Elias, '35....104 S. Michigan av., Chicago 3, Ill. OALR
Selinger, Samuel, '33.230 Princeton Drive, Lake Worth, Fla. OALR
②Sellers, L. M., '20.....1719 Pacific, Dallas 1, Texas ALR
Seltz, Samuel, '31.....Doctors bldg., Cincinnati 2, Ohio ALR
Seltzer, Albert Pincus, '42
1332 N. Franklin st., Philadelphia 22, Pa. ALR
Semenov, Herman, '35
415 N. Camden dr., Beverly Hills, Calif. ALR
Senecal, Alphonse L., '44, 1 Monument Sq., Beverly, Mass. ALR
Senturia, Ben H., '39, 6355 Waterman, St. Louis, Mo. ALR
①Sewall, E. C., '08
430 El Escarpado, Stanford U., Palo Alto, Calif. ALR
Seward, Jackson A., '31..115 E. 61st, New York 21, N. Y. ALR
①Seydell, Ernest M., '13
First Nat'l Bank bldg., Wichita 2, Kan. ALR
Shafer, Albert H., '36..E. Greenman Rd., Haddonfield, N. J. ALR
Shaffer, D. Howard, '37
7036 Jenkins Arcade, Pittsburgh 22, Pa. OP
Shaffer, Harry Gard, '40....225 Cherry st., Clearfield, Pa. ALR
①Shahan, W. E., '17..Metropolitan bldg., St. Louis 3, Mo. OP
Shambaugh, George E. Jr., '38
55 E. Washington St., Chicago 2, Ill. ALR
Shanedling, Philip D., '43, Lt. (M.C.),
6832 Paxton, Chicago 49, Ill. OP
①Shannon, C. E. G., '10.1930 Chestnut, Philadelphia 3, Pa. OP
Shannon, Clarence William, '31
New 4th and Pike bldg., Seattle 1, Wash. OP
Shannon, James B., '36
66 S. Fullerton av., Montclair, N. J. ALR
Shannon, L. M., '36..66 S. Fullerton av., Montclair, N. J. ALR
②Shapero, I. M., '19...289 Alexander, Rochester 7, N. Y. OALR
Shapira, Theo. M., '34....58 E. Washington, Chicago 2, Ill. OP
Shapiro, S. L., '30
25 E. Washington Street, Chicago 2, Ill. ALR
Sharp, Benjamin S., '26..339 Thayer St., Providence, R.I. OALR
①Shawhan, Glenn E., '13
Dewey Scales bldg., Nampa, Idaho OALR
①Shea, J. J., '16...1018 Madison av., Memphis 3, Tenn. ALR
Sheahan, William L., '26.59 College, New Haven 10, Conn. OALR
Shearer, George M., '37, 25 Spruce St., Jamestown, N. Y. ALR
Shearer, Harry A., '39.....405 E. Grand av., Beloit, Wis. ALR
①Shellman, John L., '17...Box 2, Pacific Palisades, Calif. OP
②Shemeley, William G., Jr., '19
7 Haddan av., Camden, N. J. OALR
Shepard, Emerson P., '37..128 W. Main St., Chillicothe, O. ALR
Shepard, Warren Brundage, '41
Jenkins Arcade, Pittsburgh 22, Pa. ALR
Shepard, Willis B., '45.....Miner bldg., Eugene, Ore. ALR
Shepherd, Elmer Roberts, '41
1746 K st. N.W., Washington 6, D. C. ALR
Sheppard, Ernest A. W., '36
George Washington Uni., Dept. of Oph., 1801 K st. N.W.
Washington, D.C. OP

**Sheppard, Louis Benjamin, '37**
Suite 301 Medical Arts Bldg., Richmond, Va. OP

**Sher, Maurice A., '38**
5022 Fifteenth av., Brooklyn 19, N. Y. ALR

①**Sherman, A. Edward, '08,** Lt. Col. (M.C.), (retired),
Route 1, Box 1401, La Mesa, Calif. OALR

**Sherman, Arthur E., '39,** Major (MC)
Valley Forge General Hospital, Phoenixville, Pa. OP PIS

**Sherman, A. Russell, '33**.....671 Broad, Newark 2, N. J. OP
①**Sherman, Elbert S., '12**.....671 Broad, Newark 2, N. J. OP

**Sherman, R. Newton, '34**
Phoenix bldg., Bay City, Mich. OALR

①**Shields, J. M., '15**..Metropolitan bldg., Denver 2, Colo. OP

**Shilling, Raymond L., '42**....408 Center st., Ashland, Ohio ALR

**Shipley, J. L., '33,** Capt. (MC) USN (Retired)
309 W. Church st., Elizabeth City, N.C. OALR

**Shipman, James S., '35**.....514 Cooper, Camden, N.J. OP

②**Shiras, Howard H., '20,**
13302 Cormere av., Cleveland 20, Ohio OP

**Shlossberg, Frank Ronald, '41**
116 Emerson st., Haverhill, Mass. ALR

**Shoemaker, Robert Eugene, '46**
1248 Hamilton St., Allentown, Pa. OP

**Shofstall, Charles Kirkpatrick, '45**
Balcony bldg., Kansas City 2, Mo. ALR

**Shope, Edward Pierce L., '32**
807 Wilson bldg., Camden, N. J. OP

**Shorr, Abraham, '40**
225 Eastern Parkway, Brooklyn 16, N. Y. ALR

**Shroder, Luther Null, '45**..144 W. 4th St., Greenville, Ohio ALR
**Shulruff, Harry I., '46**..3701 Main St., East Chicago, Ind. OP

**Shumaker, Phil W., '39**
9730 Wilshire blvd., Beverly Hills, Calif. ALR

①**Shuman, G. H., '17**....351 Fifth av., Pittsburgh 22, Pa. OP
③**Shurly, Burt R., '03**..62 Adams av. W., Detroit 26, Mich. ALR

**Shurtleff, Raymond S., '41**
2224 Harrison st., Davenport, Ia. ALR

**Shuster, B. H., '29**.....1824 Pine, Philadelphia 3, Pa. ALR
**Sidwell, C. E., '29**.....608 Fourth av., Longmont, Colo. ALR

②**Siegel, Francis X., '22**
Union Central bldg., Cincinnati 2, Ohio OALR

**Siewert, Otto Lucht, '42**.....33 Island Ave., Aurora, Ill. OP

**Silberman, Meyer, '38,**
266 Brooklyn av., Brooklyn 13, N. Y. ALR

②**Silbernagel, C. E., '18**..247 E. State, Columbus 15, Ohio OALR
**Silcox, Louis Edward, '40,**
255 S. 17th St., Philadelphia 3, Pa. ALR

**Silverberg, Joseph S., '39**
755 Ocean av., Brooklyn 26, N. Y. ALR

**Silverberg, Samuel Joshua, '40,**
315 Whitney av., New Haven 11, Conn. ALR

**Simkins, James J., '37**..1737 Chestnut, Philadelphia 3, Pa. ALR
**Simmonds, Noel T., '38**.....1404 Murray, Alexandria, La. OP
**Simmons, J. Thomas, '43**.....316 W. 10th st., Erie, Pa. OP
**Simmons, John T., '46**....1504 Alabama Ave., Jasper, Ala. ALR
**Simon, Emanuel, '32**.....377 State St., Albany, N.Y. ALR
**Simons, Harold, '44**..10 California St., Redwood City, Calif. OP

**Simons, Leander T., '42,**
1039 Lowry Medical Arts bldg., St. Paul 2, Minn. OP

**Simons, Max, '41**
2580 N. W. Mildred st., Portland 10, Ore. ALR

**Simons, N. S., '30**....MacCornack Clinic, Whitehall, Wis. ALR

**Simonton, Kinsey M., '41**....Mayo Clinic, Rochester, Minn. ALR

**Simpson, G. Victor, '31,**
1712 Rhode Island ave, Washington 6, D. C. OP

②**Simpson, H. Lee, '20**
David Whitney bldg., Detroit 26, Mich. ALR
**Simpson, John R., '29,** Med. Arts bldg., Pittsburgh 13, Pa. ALR
**Simpson, M. B., '30**..Professional bldg., Kansas City 6, Mo. ALR
**Simpson, Robert K., '39**.....P.O. Box 789, Kerrville, Tex. OP

①**Simpson, W. Likely, '11**
Physicians and Surgeons bldg., Memphis 3, Tenn. OALR
**Sims, Lawrence, '42**....3723 N. Gale, Indianapolis 1, Ind. ALR
**Sinamark, Andrew, '28**.....Power bldg., Hibbing, Minn. OALR
**Singleton, John D., '37**.....3704 Dickason, Dallas, Texas ALR
**Siniscal, Arthur Alfred, '45**..M. Trachoma Hosp., Rolla, Mo. OP
**Sitchevska, Olga L., '32**..30 Fifth av., New York 11, N. Y. OP
**Sitney, Julian Jay, '43**
2007 Wilshire blvd., Los Angeles 5, Calif. OP

**Skillern, S. R., Jr., '25**.....1734 Pine, Philadelphia 3, Pa. ALR
**Skinner, Merton Byron, '45**.30 N. Michigan av., Chicago, Ill. ALR
**Skirball, Joseph J., '27**
353 Commonwealth av., Boston 15,, Mass. OP

**Sklarow, Louis, '44**.....43 Center st., Gowanda, N. Y. ALR
**Skow, John D., '39**..2001 Collingwood av., Toledo 2, Ohio OP
**Slack, Walter K., '34**...308 Eddy bldg., Saginaw 3, Mich. ALR
**Slaughter, Howard Clarence, '45**
908 Hulman bldg., Evansville, Ind. OP

**Sleight, James Duane, '46**
401 Security Bank bldg., Battle Creek, Mich. OP

②**Sloan, Henry L., '19**.....106 W. 7th, Charlotte 2, N.C. OP
**Sloane, Albert E., '38**..416 Marlborough, Boston 15, Mass. OP
**Sloane, H. O., '30**.....1717 Pine, Philadelphia 3, Pa. OP
**Sly, John L., '33**.....382 Springfield av., Summit, N. J. ALR
**Small, Mary Louise, '38**.....18 W. Read, Baltimore 1, Md. OP
**Smart, Robert Edwin, '42**
Medical Arts bldg., Ottawa, Ontario, Can. OP
**Smedal, Erling A., '44,** 456 W. Park av., Mansfield, Ohio OP
**Smiley, Wm. A., '38,**
104 S. Michigan Ave., Chicago 3, Ill. ALR

**Smit, William M., '28**
5285 Washington blvd., St. Louis 8, Mo. ALR

**Smith, Alma C., '36,** Capt. (MC) USN
U.S. Naval Air Station, San Diego, Calif. OALR
**Smith, Austin T., '39,** 1830 Spruce st., Philadelphia 3, Pa. ALR
**Smith, Carlie Souter, '27**...Holland bldg., Springfield, Mo. OALR
**Smith, Carroll L., '29**
Paulsen Med. & Dental bldg., Spokane 8, Wash. OALR
**Smith, Clarence H., '37**..140 E. 54th, New York 22, N. Y. ALR
②**Smith, D. S., '18**....State Bank bldg., La Crosse, Wis. OALR
**Smith, Dennis V., '44**....110 Pine st., Long Beach, Calif. OP
①**Smith, E. Terry, '12**..P. O. Box 42, West Hartford, Conn. OP
**Smith, Ernest Gerard, '41**.339 N. Duke, Lancaster, Pa. OP
**Smith, Eugene C., '36**
814 Mott Foundation bldg., Flint 3, Mich. OALR

**Smith, Ferris, '33**
Blodgett Med. bldg., Grand Rapids 6, Mich. ALR-PI S

**Smith, Franklin C., '39**
2219 Radcliff av., Charlotte 4, N. C. OP

①**Smith, George B., '14**.....Harbin Hosp., Rome, Ga. OALR
**Smith, George H., Jr., '38**
114 W. Boscawen st., Winchester, Va. ALR

**Smith, Guy William, '46**
1014 Republic bldg., Denver, Colo. ALR

②**Smith, H. A., '18**, 226 N. Greenleaf av., Whittier, Calif. OP
**Smith, H. Brooks, '40**.....303 S. Main, Bluffton, Ind. OP
**Smith, Harold D., '39**.540 Investment bldg., Pomona, Calif. ALR
**Smith, Henry B., '37**.131 Fulton av., Hempstead, N. Y. ALR
**Smith, Henry C., '38**.....Doctors bldg., Nashville 3, Tenn. OP

**Smith, Herschel S., '42**
218 E. Kirkwood ave., Bloomington, Ind. OP

Smith, Hiram J., '38..30 N. Michigan av., Chicago 2, Ill. OP
Smith, Homer Edwards, '46
1005 Medical Arts bldg., Salt Lake City, Utah OP
Smith, I. Ruebert, '39.....116 College st., Toronto, Can. ALR
Smith, J. Allen, '39.....Doctors bldg., Macon, Ga. ALR
Smith, James Morrisset., '31
123 E. 53rd, New York 22, N. Y. ALR
Smith, James W., '37..1016 Fifth av., New York 28, N. Y. OP
Smith, Jay D., '38.....813 N. 2nd st., Harrisburg, Pa. ALR
Smith, Josiah E., '39..60 Meeting st., Charleston 1, S. C. OALR
Smith, Leon, '39.....467 Linwood av., Buffalo 9, N. Y. ALR
Smith, Leslie M., '43, Sr. Surgeon USPHS,
U. S. Marine Hospital, New Orleans 15, La. OP
Smith, Mason, '44..1100 W. Franklin St., Richmond 20, Va. OP
Smith, Maynard P., '44..206 Prof. bldg., Richmond 19, Va. ALR
Smith, Morley T., '36..421 Huguenot, New Rochelle, N. Y. ALR
Smith, Norman N., '43
291 Whitney av., New Haven 11, Conn. ALR
Smith, R. N., '29....Medical Arts bldg., Tulsa 3, Okla. ALR
Smith, R. O., '37.....73 Third av. N. W., Pulaski, Va. ALR
Smith, Robert Arvil, '41
106 N. Main St., New Castle, Ind. ALR
Smith, Rodger Browning, '42
224 W. Washington av., Madison 3, Wis. ALR
Smith, Thomas Leonard, '46..800 Broadway, Lorain, Ohio ALR
Smith, Thomas T., '43, 211 Med. Arts bldg., Omaha 2, Nebr. ALR
①Smith, Victor C., '12
American Bank bldg., New Orleans 12, La. OP
Smith, W. Hamilton, '36
314 Wareham bldg., Hagerstown, Md. ALR
Smith, W. Jewell, '39....2014 Tulare St., Fresno 1, Calif. ALR
Smith, William L., '30
Medical Arts bldg., Salt Lake City 1, Utah OP
Smith, Windsor R., '33.....67 Front, Binghamton, N. Y. ALR
Smithies, Harold R., '37..2241 Central av., Alameda, Calif. ALR
Smoot, Merrill C., '39,
130 W. Washington st., Hagerstown, Md. ALR
Snell, Albert C., '25..53 S. Fitzhugh, Rochester 4, N. Y. OP
Snell, Albert Conrad, Jr., '46
53 S. Fitzhugh St., Rochester 4, N.Y. OP
Snell, George C., '33, Medical Arts bldg., Toronto, Can. ALR
Sneller, Charles D., '44, 320 Jefferson bldg., Peoria, Ill. ALR
Snelling, P. W., '31.....179 Allyn, Hartford 3, Conn. ALR
Snitman, Maurice F., '42
55 E. Washington st., Chicago 2, Ill. ALR
Snow, Harold L., '40.....639 W. 9th, San Pedro, Calif. OP
Snow, William J., '44, 1215 Walker av., Houston, Texas ALR
Snydacker, Daniel, '38..2626 Orrington Ave., Evanston, Ill. OP
Snyder, Eugene R., '40....7 W. 96th st., New York, N. Y. ALR
Snyder, George Gordon, '40
Mermont Apartments, Bryn Mawr, Pa. ALR
Snyder, Jerome, '38.....11 E. Chase St., Baltimore, Md. ALR
Snyder, William S., Jr., '36..324 W. Main, Frankfort, Ky. ALR
Soboroff, Simon H., '40
1608 Milwaukee av., Chicago 47, Ill. ALR
Soldineer, Marshall Y., '44, 333 Linwood Ave, Buffalo, N. Y. ALR
Solo, Adrian, '40.....174 Highland av., Somerville, Mass. ALR
Solomon, Henry D., '39
Power & Light bldg., St. Petersburg 5, Fla. ALR
Somberg, J. S., '23...406 Bulkley bldg., Cleveland 15, Ohio OP
Sommer, Ignatius George, '45
981 S. Westmoreland, Los Angeles 6, Calif. OALR
Sooy, Francis A., '46
Suite 808, 490 Post St., San Francisco, Calif. ALR
Soper, Gail R., '33.....636 Church, Evanston, Ill. OP
Southgate, Paul T., '45
302 Professional Bldg., Long Beach, Calif. OP
Sowers, Alva B., '37
6 N. Michigan av., Sec. 2, Chicago 2, Ill. OP
Spaeth, Edmund B., '28
1930 Chestnut, Philadelphia 3, Pa. OP-PI S
Spain, Marceda L., '44, 629½ Main st., Rapid City, S. D. ALR
②Spake, LaVerne B., '20
Brotherhood bldg., Kansas City 11, Kans. ALR
Spalding, Lansford M., '39
185 Fourteenth st., Astoria, Ore. ALR
Sparber, Herman, '35
4701 Fifteenth av., Brooklyn 19, N. Y. OP
Sparer, William S., '39....50 E. 10th, New York 3, N. Y. ALR
Sparks, Alan Leo, '40
1024 Hume Mansur Bldg., Indianapolis 4, Ind. ALR
Spear, Dean, '44..516 Boston bldg., Salt Lake City 1, Utah OP
Spearman, Maurice P. S., '42,
1315 First Nat'l Bank bldg., El Paso, Texas ALR
Speas, William Paul, '41
Reynolds bldg., Winston-Salem 3, N. C. OP
①Spencer, Frank R., '04..Physicians bldg., Boulder, Colo. ALR
Spencer, George Albert, '28 Rt. 1, Box 42, Placerville, Calif. OALR
Spencer, S. C., '29.....Professional bldg., Waco, Texas OALR
②Spencer, W. H., '18
1830 S. Rittenhouse sq., Philadelphia 3, Pa. ALR
Spencer, Walter Carlton Edward, '40
Medical Arts bldg., Houston 2, Tex. OP
①Spengler, John A., '16.....423 S. Main, Geneva, N. Y. OP
Sperry, F. N., '36..107 Whitney av., New Haven 10, Conn. ALR
Spiegelglass, A. B., '39.....417 Main, Hackensack, N. J. ALR
Spiesman, I. G., '29, 1908 St. Charles road, Maywood, Ill. ALR
Spiller, Arthur Eliot, '46
c/o Sutter Hospital, Sutter St., San Francisco 9, Calif. OP
①Spitze, E. C., '16.....Murphy bldg. East St. Louis, Ill. OP
Sporn, Hyman, '38.....839 Park Place, Brooklyn, N.Y. ALR
Springer, James Earl, '41
Peoples Bank bldg., Akron 8, Ohio ALR
Sproule, Ralph P., '28
208 E. Wisconsin, Milwaukee 2, Wis. OALR
Sputh, Carl B., '40
224 N. Meridian st., No. 301, Indianapolis 4, Ind. ALR
Sputh, Carl Brosius, '46
224 N. Meridian, Indianapolis 4, Ind. ALR
Stack, James Edward, '41
8838 Woodhaven blvd., Woodhaven 21, N. Y. ALR
Stagman, Joseph '45....55 E. Washington St., Chicago, Ill. ALR
Stahl, Richard Holmes, '46
143 Portage Trail, Cuyahoga Falls, Ohio ALR
Staman, Harry H., '45..49 W. Church St., Uniontown, Pa. ALR
Stamm, Carl, '15....1737 Chestnut St., Philadelphia 3, Pa. ALR
①Stanbery, Henry, '10..Doctors bldg., Cincinnati 2, Ohio OP
Stanford, Charles E., '39
Medical Arts bldg., Minneapolis 2, Minn. OP
①Stanford, J. B., '16..899 Madison av., Memphis 3, Tenn. OP
Stanley, C. R., '30....508 Tenth st., Worthington, Minn. ALR
Stark, Jesse B., '25.....46 Park av., New York 16, N. Y. OP
Stark, W. Berkeley, '27.Medical Arts bldg., Toronto 5, Can. ALR
Staton, Younger A., '45
715 Harvey bldg., West Palm Beach, Fla. OALR
Statti, Louis Wm., '37..Jenkins Arcade, Pittsburgh 22, Pa. OP
Stauffer, F. Leaver, '34
52 East South Temple st., Salt Lake City 1, Utah ALR

**Stauffer, Harry B., '43**
234 Madison, Jefferson City, Missouri ALR

**Steele, Clarence Hart, '46**
Ochsner Clinic, New Orleans, La. ALR

**Steele, Wendell M., '46**.....17 E. Locust St., Newark, Ohio OP

**Steele, Willard, '28**.....721 Broad, Chattanooga 2, Tenn. OALR

**Steffens, Lincoln F., '44**.....1200 Main st., Dubuque Ia. OP

**Steffensen, W. H., '37**, Blodgett Medical bldg.,
Wealthy St. & Plymouth Rd., Grand Rapids 6, Mich. ALRPIS

①**Steim, J. M., '14**
First Nat'l Bank bldg., New Kensington, Pa. OP

**Steinbach, Louis, '37**
1882 Grand Concourse, New York 57, N. Y. OP

**Steinberg, Martin R., '41,**
226 W. 70th St., New York 23, N. Y. ALR

**Steinberg, Nathan, '45**
253 Sunrise Highway, Rockville Centre, N.Y. ALR

**Steinbugler, Wm. F. C., '35,**
515 Ocean av., Brooklyn 26, N. Y. OP

**Steine, Ben Zion, '46**
Medical Arts bldg., Montreal, P.Q., Canada ALR

②**Steinfeld, A. L., '22**....125 Fifteenth st., Toledo 2, Ohio OALR

**Stelle, Harry L., '45**..207 Commerce bldg., Pittsburg, Kans. OALR

**Stephan, Lewis B., '41**.....207 E. College, Oberlin, Ohio OP

**Stephens, Henry F., '44, Lt. Comdr. (M.C.),**
295 Rumstick Rd., Barrington, R. I. OP

**Stern, Milton J., '35**
First Nat'l Bank bldg., Lexington 3, Ky. OP

**Stern, Wilhelm E., '41**
1099 Bushwick av., Brooklyn 21, N. Y. ALR

**Sternberg, Paul, '46**.....58 E. Washington, Chicago 2, Ill. OP

**Stevens, Ralph W., '38,**
302-12 Baker bldg., Walla Walla, Wash. OP

①**Stevenson, Walter, '15**.....W. C. U. bldg., Quincy, Ill. OALR

**Stevenson, Walter D., Jr., '43**, 500 W.C. U. bldg., Quincy, Ill. ALR

**Stevenson, William W., '31**, Kresge bldg., Flint 3, Mich. OALR

**Stewart, Charles E., Jr., '43**..Rt. 2, Box 452, Vista, Calif. ALR

**Stewart, James K., '37**...Wheeling Clinic, Wheeling, W. Va. ALR

**Stewart, James V., '42,**
First Nat'l Bank Bldg., Massillon, Ohio ALR

②**Stewart, Walter E., '20**, Tribune bldg., Terre Haute, Ind. OALR

①**Stillson, H., '03**.....Seabord bldg., Seattle 1, Wash. OALR

③**Stimson, George W., '19**.580 Perkins drive, Warren, Ohio OALR

**Stine, George H., '32**..Burns bldg., Colorado Springs, Colo. OP

**Stinson, Wm. D., '28**..899 Madison av., Memphis 3, Tenn. ALR

**Stitt, H. L., '27**.....Doctors bldg., Cincinnati 2, Ohio ALR

**Stockwell, Eunice LeBaron, '42**
2018 Pine st., Philadelphia 3, Pa. OP

**Stoffregen, Charles Henry, '41**
10515 Carnegie av., Cleveland 6, Ohio ALR

**Stokes, Wm. H., '26**.Medical Arts bldg., Omaha 2, Nebr. OP

**Stoll, K. Ludwig, '29**.....19 W. 7th, Cincinnati 2, Ohio OP

**Stolzar, Irwin H., '45**
831 Guardian Bldg., Cleveland, Ohio OP

**Stone, Harry B., '30**,..Medical Arts bldg., Roanoke 11, Va. ALR

**Stone, Harry B., Jr., '39,**
811 Medical Arts bldg., Roanoke 11, Va. ALR

**Stone, Leslie O., '35**, Capt. (MC) USN
U. S. Naval Hospital, Bethesda, Md. ALR

**Stonecypher, D. D., '30**
102½ S. 9th st., Nebraska City, Nebraska ALR

**Stoner, Cyrus Hubert, '42**
1029 Candler bldg., Atlanta, Ga. OP

**Stoner, Donald D., '43**...156 W. Pomfret st., Carlisle, Pa. ALR

①**Stotter, Arthur L., '16,**
1148 Euclid av., Cleveland 15, Ohio OALR

**Stough, John T., '39**....4501 Fannin St., Houston 4, Tex. OP

**Stoutenborough, Wm. A., '27**
21 E. State, Columbus 15, Ohio OALR

**Stovin, Joseph S., '36**
151 Central Park West, New York 23, N. Y. ALR

**Stow, M. Noel, '44**..1746 K. st., NW, Washington 6, D. C. OP

**Straatsma, Clarence R., '32,**
66 E. 79th st., New York 21, N. Y. ALR-PI S

**Strange, Russell H., '44**, 117 S. College av.,
Mt. Pleasant, Mich. ALR

**Straus, Gerhard D., '41**
4459 N. Murray ave., Milwaukee, Wis. ALR

**Strause, Harold L., '43**
826 N. 5th st., Reading, Pennsylvania ALR

**Strauss, Arthur, '43**
130 Pavilion av., Long Branch, New Jersey ALR

**Strauss, J. F., '25**....104 S. Michigan av., Chicago 3, Ill. ALR

**Street, Alvin Murray, '41**.745 5th av., New York 22, N. Y. ALR

**Streicher, Carl J., '44**....217 Park Place, Ashtabula, Ohio OP

**Streit, August J., '31**.....724 Polk, Amarillo, Texas ALR

**Strickland, Horace G., '44**..Jefferson bldg., Greensboro, N.C. OP

**Strickler, Alfred D., '43**.....26 N. 9th st., Lebanon, Pa. OP

**Strickler, Joseph G., '37**.200 W. 57th, New York 19, N. Y. ALR

**Strikol, Albert J., '36**
621 Delaware av., Wilmington 9, Delaware ALR

**Strong, James C., Jr., '42**
617 Majestic bldg., Denver 2, Colo. OP

**Stroud, Sanders Key, '40**
519 Medical Professional bldg., Corpus Christi, Tex. OP

②**Strout, G. Elmer, '19,**
1009 Nicollet av., Minneapolis 2, Minn. OALR

**Struble, Gilbert C., '42**
2921 Glendon ave., West Los Angeles 34, Calif. OP

**Stuard, Charles G., '43**, 310 Med. Arts bldg., Tulsa 3, Okla. OP

①**Stueber, F. G., '05**.....209 W. North, Lima, Ohio OALR

①**Stueber, Paul J., '17**.....209 W. North, Lima, Ohio OALR

**Stump, Robert Byron, '46**..408 Hynds bldg., Cheyenne, Wyo. OP

②**Sturges, Leigh F., '18**..121 E. 60th, New York 22, N. Y. ALR

**Stutsman, Albert C., '37**
539 N. Grand blvd., St. Louis 3, Mo. ALR

**Sudranski, Herbert F., '40**
23 E. Ohio St., Indianapolis 4, Ind. OP

**Suehs, Oliver W., '41**.....120 W. 7th St., Austin, Tex. ALR

**Sugar, H. Saul, '41**
2311 David Broderick Tower, Detroit 26, Mich. OP

**Sullivan, Charles T., '37**..277 Alexander, Rochester 7, N. Y. OP

**Sullivan, Clifford P., '40**, 304 W. 63rd St., Chicago 21, Ill. OP

**Sullivan, Joseph A., '33**, Wing Commander (RCAF),
Medical Arts bldg., Toronto 5, Ontario, Can. ALR

**Sullivan, Robert E., '32**...Doctors bldg., Nashville 3, Tenn. OP

**Sullivan, Timothy, '42**.....104-15th av., Sterling Ill. ALR

②**Sulzman, Frank M., '22**.....1831 Fifth av., Troy, N.Y. OALR

**Sulzman, John H., '39**.....1831 Fifth av., Troy, N.Y. OALR

**Summers, Wm. S. '27**
David Whitney bldg., Detroit 26, Mich. OALR

**Sunde, Einar A., '38**.....627 Second, Brooklyn 15, N. Y. ALR

**Surber, John Roger, '45**..119 E. 7th St., New York 21, N.Y. ALR

**Sutherland, J. M., '29**....Fisher bldg., Detroit 2, Mich. ALR

**Sutherland, William S., '40**
1930 Chestnut st., Philadelphia 3, Pa. OP

**Swab, Charles M., '28**, Medical Arts bldg., Omaha 2, Nebr. OP

Swalm, Charles J., '39
1330 W. Rockland, Philadelphia 41, Pa. ALR
Swan, John R., '39
915 Hume Mansur bldg., Indianapolis 4, Ind. ALR
Swan, Kenneth C., '43, Dep't of Oph., University of Ore. Med. Sch., Marquam Hill Rd., Portland, 1, Ore. OP
Swan, Otis Dwight, '46
1035 Forum bldg., 9th & K, Sacramento 14, Cal. ALR
Swann, Cecil C., '32....Flat Iron bldg., Ashville, N. C. ALR
Swanson, Clifford A., '38, Adm. (MC) USN
The Westchester Apts., 836-B, Washington 16, D. C. OALR
Swenson, Rudolph E., '45....1 Carver St., Plymouth, Mass. ALR
①Swetman, C. R. K., '15
219 S. Mt. Vernon, Prescott, Ariz. ALR
Swick, David A., '37....30 W. 70th, New York 23, N. Y. OP
Swift, Donald E., '39..25 Prospect Park W., Brooklyn, N.Y. OP
Swigert, J. Leonard, '37.....Republic bldg., Denver, Colo. OP
Swing, Frank U., '35...Doctors bldg., Cincinnati 2, Ohio ALR
Sykes, Clarence S., '39..2201 Market, Galveston, Texas OP
Sykes, Edwin Meredith, '42
Nix Professional bldg., San Antonio 5, Tex. ALR
Syracuse, Victor R., '41
29 Washington Sq. W., New York 11, N. Y. OALR

## T

Tabor, Gilbert S., '37....11 Park Row, Millerton, N. Y. ALR
Tait, Edwin Forbes, '45..1324 W. Main St., Norristown, Pa. OP
Tangen, George M., '43,
2640 Glenhurst av., St. Louis Park, Minneapolis 16, Minn. ALR
Tanner, James Wallace, '32
131 S. Barstow St., Eau Claire, Wis. ALR
Tanner, Owen Ralph, '46
627 University Ave., Palo Alto, Calif. OP
Tapia y Fernandez, Ricardo, '46
4a de Londres, 67, Mexico, D.F. ALR
①Tarpley, George J., '20
Canal bldg., New Orleans 12, La. ALR
①Tarun, William, '10.2309 Kenoak rd., Baltimore 9, Md. OP
Tassman, Isaac S., '29
Medical Arts bldg., Philadelphia 2, Pa. OP
Taylor, E. Merle, '31.Medical Arts bldg., Portland 5, Ore. OP
Taylor, H. Marshall, '35
111 W. Adams, Jacksonville 2, Fla. ALR
Taylor, Joseph W., '34.....706 Franklin, Tampa 2, Fla. OALR
①Taylor, M. C., '17, 3000 Knowlson av., Pittsburgh 26, Pa. OP
Taylor, Shahane R., '39, Jefferson bldg., Greensboro, N. C. ALR
Teall, Raymond E. '28
Professional bldg., Kansas City 6, Mo. ALR
Teasdale, Laurie Redmond, '45
311 Professional Bldg., Charleston, S.C. ALR
Tebbutt, H. K., '30.....240 State, Albany 6, N. Y. ALR
Teed, R. Wallace, '36..410 Highland rd., Ann Arbor, Mich. ALR
Tegelberg, Julius J., '39
12 Monterey road, Worcester 6, Mass. ALR
Tegge, Charles W., '37
1801 Eye st. N.W., Washington 6, D.C. ALR
Teixler, Victor Adolph, '45
224 Hume Mansur Bldg., Indianapolis 4, Ind. OP
Ten Eyck, Thomas G., '44,
808 N. E. 39th st., Portland 13, Oregon ALR
Terrell, Vance, '42, Stephenville Hospital, Stephenville, Tex. ALR
Thacker, Edgar A., '43
602 W. University av., Urbana, Illinois ALR
Thaler, Nathan, '40
570 Eastern Parkway, Brooklyn 25, N. Y. ALR
Theis, Franklyn B., '39.....209 S. Broadway, Nyack, N.Y. OALR
Thel, Henry C., '38.....387 Franklin, Aliquippa, Pa. ALR
Theobald, Georgiana Dvorak, '29
715 Lake, Oak Park, Ill. OP
Theobald, John J., '29.....715 Lake, Oak Park, Ill. ALR
Theobald, Walter H., '30
307 N. Michigan av., Chicago 1, Ill. ALR
Theodore, Frederick H., '40
667 Madison Ave., New York 21, N.Y. OP
①Thigpen, C. A., '12....401 S. Court, Montgomery 5, Ala. OALR
Thoburn, Thomas W., '39
1020 Huron road, Cleveland 15, Ohio ALR
Thomas, Charles I., '43
10515 Carnegie av., Cleveland 6, Ohio OP
Thomas, Edward R., '37.....60 Wyoming, Dayton 9, Ohio OP
①Thomas, Francis W., '16, 327 E. State, Columbus 15, Ohio OALR
Thomas, Harry Vernon, '40
Empire Bank bldg., Clarksburg, W. Va. OP
Thomas, James Alanson, '41
Southern Michigan Bank bldg., Coldwater, Mich. ALR
Thomas, James W., '28..1310 Crestdale Rd., Lincoln 8, Neb. OALR
Thomas, W. Maxwell, '35,
206 Medical Arts bldg., Dallas 1, Tex. OP
①Thomason, H. E., '12
Professional bldg., Kansas City 6, Mo. ALR
Thompson, Chase S., '45, The Standefer & Canon Clinic
West Texas Hospital, Lubbock, Tex. ALR
①Thompson, E. H., '07....19 W. 7th, Cincinnati 2, Ohio OALR
①Thompson, G. H., '09....74 North st., Pittsfield, Mass. OALR
Thompson, Howard E., '28.....1100 Main, Dubuque, Iowa OALR
Thompson, James P., '42..532 Miller bldg., Yakima, Wash. OP
Thompson, Lester Emmit, '42
Woolworth bldg., Salida, Colo. ALR
①Thompson, Peter H., '14
308 Commonwealth av., Boston 15, Mass. OP
Thompson, Wm. R., '43..203 W. 2nd st., Lexington 15, Ky. ALR
①Thomsen-von Colditz, G., '06
Dun Huntin, Merritt Island, Fla. OALR
Thorburn, O. L., '39.....Box 451, Ames, Iowa ALR
Thornburgh, Robert Grant, '42
Professional bldg., Long Beach 2, Calif. OP
Thorne, Fred Sanborn, '39
270 Commonwealth av., Boston 16, Mass. OP
Thornell, William Clyde, '45
1002 Carew Tower, Cincinnati, Ohio ALR
Thorpe, Harvey E., '31..Jenkins Arcade, Pittsburgh 22, Pa. OP
Thorson, John A., '36.....Roshek bldg., Dubuque, Iowa OP
Thygeson, Phillips, '34
524 Sainte Claire bldg., San Jose 23, Calif. OP
Tickle, Thomas G., '36.30 E. 40th st., New York 16, N. Y. ALR
Tiedke, Gunther E., '39, 120 N. Michigan av. Saginaw, Mich. OP
②Tilderquist, D. L., '19..Med. Arts bldg., Duluth 2, Minn. OALR
Tillotson, R. S., '30
Medical-Dental bldg., Sacramento 14, Calif. ALR
Tindal, John M., '33.....5517 W. Fulton, Chicago 44, Ill. OALR
①Tingley, Louisa Paine, '13
416 Marlborough, Boston 15, Mass. OP
Tinkess, Donald E., '43....Stanwich Rd., Greenwich, Conn. OALR
Tippin, Ernest E., '34
1st Nat'l Bank bldg., Wichita 2, Kan. ALR
Tisher, Paul Winslow, '45
99 W. Main St., New Britain, Conn. OP
①Tobey, George L. Jr., '17
403 Commonwealth av., Boston 15, Mass. ALR

Tobin, Henry Loughlin, '45..74 Front St., Binghamton, N.Y. OP
②Todd, Grover C., '22...Jenkins Arcade, Pittsburgh 22, Pa. ALR
Togus, Leopold T., '37.....967 Elm, Manchester, N. H. ALR
Tolan, John F., '38.....1115 Terry, Seattle 5, Wash. ALR
Tolan, Thomas L., '33.....Wells bldg., Milwaukee 2, Wis. ALR
Toland, Virgil A., '46....509 McDowell Rd., Phoenix, Ariz. OP
Tolman, Henry, '39.....136 Federal, Salem, Mass. ALR
Tomassene, R. A., '23.....1144 Market, Wheeling, W. Va. OALR
Tomb, E. H., '30.....46 Lexington, Framingham, Mass. ALR
Tomlinson, J. Wesley, '42.900 Chester Pike, Sharon Hill, Pa. OP
Tomlinson, Roland D., '39, 505 E. Broad st.,
Westfield, N. J. ALR
①Tooker, C. W., '17.....Carleton bldg., St. Louis 1, Mo. OP
Toot, J. F., '30.....First Nat'l Bank bldg., Canton 2, Ohio OP
Town, Arno E., '36.....140 E. 54th St., New York, N.Y. OP
Townes, C. Dwight, '32..1014 Heyburn bldg., Louisville, Ky. OP
Townsend, John F., '23.85 Wentworth, Charleston 6, S. C. OALR
Towson, Charles E., '32
Germantown Prof. bldg., Philadelphia 44, Pa. ALR
Tracewell, George L., '41
300 Commerce bldg., Okmulgee, Okla. ALR
Tracht, Robert R., '41.....Lowry bldg., St. Paul 2, Minn. OP
Trainor, George M., '42,
277 Alexander st., Rochester 7, N.Y. ALR
Trapasso, Anthony J., '44
Sault Polyclinic, Sault Ste. Marie, Mich. ALR
Traugh, George H., '33
309 Cleveland av., Fairmont, W. Va. ALR
Tremble, G. Edward, '32
1390 Sherbrooke st. W., Montreal, Can. ALR
Treskow, Frank G., '41...411 E. Mason, Milwaukee 2, Wis. ALR
Trexler, C. W., '37.....50 Dowsett av., Honolulu, T.H. ALR
Treynor, Jack Vernon, '46....532 First Ave., Co. Bluffs, Ia. ALR
②Trible, G. B., '20
1150 Connecticut av. N. W., Washington 6, D. C. OALR
②Tripp, LeRoy R., '20, 909 Badgerow bldg., Sioux City, Ia. OALR
Troncoso, Manuel U., '39
500 West End av., New York 24, N. Y. OP
Trott, Leslie De Nyse, '42
143 North Brand, Glendale 3, Calif. ALR
Trotter, Homer A., '32...40 W. North st., Buffalo 2, N. Y. ALR
Troup, Wilson J., '38.....306 Cherry St., Green Bay, Wis. OALR
Troutman, Erwin W., '40
21 East State st., Columbus 15, Ohio ALR
Trowbridge, Barnard C., '41
300 W. 47th st., Kansas City 2, Mo. ALR
Trowbridge, Dwight H., '39
T. W. Patterson bldg., Fresno 1, Calif. OP
Trueman, Robert H., '42,
600 N. 16th St., Philadelphia 30, Pa. OP
Truex, Edward H., Jr., '44....99 Pratt St., Hartford, Conn. ALR
Truitt, John Wm., '46..161 W. Wisconsin, Milwaukee, Wis. ALR
Trull, Alfred C., '38
3 Washington Square, Haverhill, Mass. OP
Trumbo, James. K., '37....P. O. Box 485, Wausau, Wis. ALR
Tucker, Frank Landale, Jr., '42
464 9th st., Brooklyn 15, N. Y. ALR
Tucker, Gabriel, '23.....250 S. 18th, Philadelphia 3, Pa. LB
①Tuckerman, W. H., '08
1020 Prospect av., Cleveland 15, Ohio OALR
Tulloch, George M., '45
410 McCulloch bldg., Okmulgee, Okla. ALR
Turchik, Frank, '40..1831 Barnum av., Bridgeport 8, Conn. ALR
①Turnbull, F. M., '16
Wilshire Med. Bldg., Los Angeles 5, Calif. ALR
①Turner, H. H., '14.....Clark bldg., Pittsburgh 22, Pa. OP
①Turner, Oliver W., '09,
155 E. 34th St., New York 16, N. Y. OALR
Turnley, William H., '33...1 Atlantic st., Stamford, Conn. ALR
Tusak, Ervin A., '31....115 E. 90th, New York 28, N. Y. OP
Twitchell, Emmus G., '37.....266 Main, Burlington, Vt. OALR
Tygett, Glenn J., '39
215 N. Sprigg st., Cape Girardeau, Mo. ALR
Tyler, Richard E., '43, The Norwood Clinic,
2501 N. 16th av., Birmingham 4, Ala. ALR
Tyler, William H., '46
University of Iowa Hosp., Iowa City, Ia. ALR
Tyson, John McClure, '40.....3 S. Brady st., DuBois, Pa. ALR

## U

Uhler, S. Mann, '30.....104 N. 8th, Allentown, Pa. ALR
Ulmer, David H. B., '39..199 Chestnut, Moorestown, N. J. ALR
Underwood, John H., '38.....Brant bldg., Canton 2, Ohio ALR
Unsworth, Arthur C., '38,
179 Allyn st., Suite 508, Hartford, Conn. OP
①Upham, Helen F., '07..305 Third av., Asbury Park, N. J. OALR
Urban, Joseph T., '44.....204 Bushkill st., Easton, Pa. ALR
U'Ren, Harold Merle, '45
1735 N. Wheeler ave., Portland 12, Pa. OP
Urist, Martin J., '44
Center & Phoenix sts., South Haven, Mich. OP
Urmston, Paul Robert, '46
303 Davidson bldg., Bay City, Mich. ALR
Usen, Louis, '36.....858 Lincoln pl., Brooklyn 16, N.Y. ALR

## V

Vadala, Anthony J., '38, Col. (M.C.),
Station Hospital, Fort Totten, Bayside, N. Y. OALR
Vail, Derrick, '31,
Rm 2605, 55 E. Washington st., Chicago 2, Ill. OP
Vaile, Ivan R., '36.....384 Bloor st. W., Toronto, Can. ALR
Valone, Frank H., '31.....107 W. Court, Rome, N. Y. ALR
Van Alyea, O. E., '35.....135 S. LaSalle, Chicago 3, Ill. ALR
Van Bergen, Thomas M., '44....227 16th st., Denver, Colo. OP
Van Buskirk, Edmund Linford, '41
308-312 N. 8th, Lafayette, Ind. OP
②van der Hoeve, Prof. J., '21
Rijnsburgerweg 14, Leiden, Hollard OALR
Vandevere, William E., '32
1st Nat'l Bank bldg., El Paso, Texas. OP
Vann, Homer King, '41
410 Cedar st. N. W., Washington 12, D. C. ALR
Van Osdol, Harry A., '31
Chamber of Commerce, Indianapolis 4, Ind. ALR
Van Poole, G. McD., '24..Young bldg., Honolulu 9, Hawaii, OALR
Van Wien, Stefan, '43, 104 S. Michigan blvd., Chicago, Ill. OP
Vanzant, T. J., '38.....Medical Arts bldg., Houston 2, Tex. OP
Vaughan, Benjamin H., '38
Adams bldg., Port Arthur, Texas ALR
②Vaughn, E. H., '19
Peoples Nat'l Bank bldg., Tyler, Texas OALR
Veach, Oscar Lloyd, '40
Whitney Trust bldg., Sheridan, Wyo. OP
①Veasey, C. A., Sr., '06
Paulsen Med. and Dental Bldg., Spokane 8, Wash. OALR
Veasey, C. A., Jr., '27
Paulsen Med. and Dental bldg., Spokane 8, Wash. OALR
Veirs, E. R., '40....Scott & White Clinic, Temple, Texas OALR

Veitch, Ambert H., '37....170 St. George, Toronto, Can. ALR
Verhoeff, F. H., '36..61 Monmouth St., Brookline, Mass. OP
Vermeren, John C., '38....4753 Broadway, Chicago 40, Ill. ALR
Vermillion, James S., '41
233 E. Sullivan st., Kingsport, Tenn. ALR
Victor, Karl N., '39
1010 Brown bldg., 321 W. Broadway, Louisville 2, Ky. ALR
Viger, Roland Joseph, '45
1414 Drummond St., Montreal, Quebec, Canada OP
Viviani, John J....'44, 180 Suydam st., Brooklyn 21, N.Y. ALR
Vogel, Felix P. S., '36
1749 Grand Concourse, New York 53, N. Y. ALR
Vogt, George C., '37.....140 Chapin, Binghamton, N. Y. ALR
①Voigt, C. B., '12.....1702 Broadway, Mattoon, Ill. OALR
Volk, Benjamin M., '38
146 Washington Ave., Albany 6, N.Y. ALR
Voltz, Vernon Carl, '46
303 N. Main St., Rockford, Ill. OP
von Lackum, John K., '37
Mer. Nat'l Bank bldg., Cedar Rapids, Iowa ALR
Von Leden, Hans Victor, '46
1046 Loyola Ave., Chicago 26, Ill. ALR
Von Sallmann, Ludwig, J. K., '46
635 W. 165th St., New York 32, N.Y. OP
von Zelinski, Walter Franz, '41,
3015 Hermosa rd., Santa Barbara, Calif. OP
Voorhees, Darrell Graham, '45
135 E. 65th, New York, N.Y. ALR
Voorhees, Earle W., '35
8 Garfield place., Poughkeepsie, N. Y. ALR
Vorisek, Elmer A., '33....1041 Lyman Ave., Oak Park, Ill. OP
Vossler, Albert E., '37
David Whitney bldg., Detroit 26, Mich. OALR
Votaw, Robert E., '37..10 Warson Terrace, Clayton 5, Mo. ALR
Vrooman, C. O., '40..55 Claremont Ave., Mt. Vernon, N.Y. OP

## W

Wachsberger, Alfred, '31..1025 9th St., Sacramento, Calif. ALR
Wagener, Henry P., '26.....Mayo Clinic, Rochester, Minn. OP
②Wagers, Arthur J., '20..1429 Spruce, Philadelphia 2, Pa. ALR
Wagner, W. A., '27...200 Carondelet, New Orleans 12, La. ALR
Wails, Theodore G., '38
Medical Arts bldg., Oklahoma City 2, Okla. ALR
Waldapfel, Richard, '40
De Merschman Gardens, Grand Junction, Colo. ALR
Waldeck, E. A., '34
238 W. Wisconsin av., Milwaukee 3, Wis. OALR
Waldman, Joseph, '37,
216 E. Gorgas Lane, Philadelphia 19, Pa. OP
②Walker, C. E., '18..Rt. 1, Box 47, Colorado Springs, Colo. OP
Walker, George W., '32....630 Peratta Way, Fresno, Calif. ALR
Walker, Glenn L., '44, Lt. Col. (M.C.),
Address Unknown OP
Walker, John R., '32.....P.O. Box M-1, Carmel, Calif. OP
Walker, Oliver Frank, '42
2873 Lewiston road, Niagara Falls, N.Y. OALR
Walker, O. J., '35.Home S. & L. bldg., Youngstown 3, Ohio ALR
Walker, R. M., '40.....115 N. Sumter St., Sumter, S. C. ALR
Wall, John William, '42
509 S. Ogden Dr., Los Angeles 36, Calif. ALR
Wall, Walton B., '45.....Professional bldg., Orlando, Fla. OP
Wallerstein, Emanuel U., '41
Professional bldg., Richmond 19, Va. ALR
Wallner, Linden J., '38....4350 Bobolink Terr., Skokie, Ill. ALR
Walls, Edward G., '35,
603 Maison Blanche bldg., New Orleans 16, La. ALR
Walsh, Francis Mark, '46
330 Lowry Medical Arts bldg., St. Paul 2, Minn. OP
Walsh, Frank B., '44,
Johns Hopkins Hosp., Baltimore 5, Maryland OP
Walsh, Theodore Edwin, '40
640 S. Kingshighway blvd., St. Louis 10, Mo. ALR
Walsh, Thomas J., '39...2059 Parrish, Philadelphia 30, Pa. ALR
Waltz, Harold D., '42
510 Medical Dental bldg., Everett, Wash. ALR
Ward, J. W., '30.....2607 Lee, Greenville, Texas ALR
Warner, Raymond C., '38
1684 N. Prospect Ave., Milwaukee 2, Wis. OP
Warner, Robert J., '29.....706 Church, Nashville, Tenn. OP
Warren, William C., Jr., '28..Doctors bldg., Atlanta 3, Ga. ALR
Washick, Frank, '42.501 Cottman st., Philadelphia 11, Pa. ALR
Watke, Frederic Martin, '41
Medical Arts bldg., Omaha 2, Nebr. ALR
Watkins, H. R., '30....Griesheim bldg., Bloomington, Ill. ALR
Watkins, John O., '41
314 Montgomery bldg., Spartanburg, S.C. OP
Watnick, Joseph Louis, '41
10 W. 96th, New York 25, N. Y. ALR
①Watson, H. de Witt, '15..151 Front, Binghamton, N.Y. OALR
Watson, O. Alton, '37
1200 N. Walker, Oklahoma City 3, Okla. ALR
Wattles, Merrill, '39....1200 S. Kuhl Ave., Orlando, Fla. ALR
②Waugh, J. M., '19
Ridge Road, R.F.D. No. 1, Willoughby, Ohio OALR
Weaver, Delmar Franklin, '41
Henry Ford Hospital, Detroit 2, Mich. ALR
Weaver, Emerson M. F., '43
727 W. 7th St., Los Angeles 14, Calif. OP
Weaver, Homer V., '32
625 Cleveland av. N. W., Canton 3, Ohio ALR
①Weaver, T. W., '14.....201 N. Main, Wichita 2, Kan. OP
Webb, M. A., '43.....4500 Olive st., St. Louis 8, Mo. ALR
Webb, Walter Whayne, '45..3rd Nat'l bldg., Dayton, Ohio ALR
Weber, H. C., '28, Capt. (MC) USN (Retired)
1112 Mason ave., Drexel Hill, Pa. OALR
Weber, Julius A., '39.....Stimson bldg., Seattle 1, Wash. ALR
Webster, David H., '37...140 E. 54th, New York 22, N. Y. OP
Webster, F. R., '30...State Tower bldg., Syracuse 2, N. Y. OP
Wechsler, A. H., '34
340 New York av.; Brooklyn 13, N. Y. ALR
Weeks, Carrol Lamont, '46
1930 Wilshire blvd., Los Angeles 5, Calif. OP
①Weeks, John E., '04, (Retired),
360 Edgecliff rd., Portland 1, Ore. OP
Weeth, C. R., '30.....37 Remsen, Brooklyn 2, N. Y. ALR
Wegner, Edwin A., Jr., '38..1791 Howard St., Chicago, Ill. ALR
Weidlein, Ivan F., '30
10515 Carnegie av., Cleveland 6, Ohio ALR
②Weih, E. P., '20.....217 Fifth av., S., Clinton, Iowa OALR
①Weil, A. I., '16....3439 Prytania, New Orleans 15, La. ALR
Weinberg, Herman Charles, '42
2101 S. Taylor Rd., University Heights 18, Ohio OP
Weinman, Edward B., '39.401 Market St., Steubenville, Ohio OP
Weinstein, Lee, '46.....1104 N. 2nd St., Harrisburg, Pa. ALR
Weinstein, M. A., '27....1530 Locust, Philadelphia 2, Pa. ALR
Weinstein, Philip, '39..400 Ingraham bldg., Miami 32, Fla. ALR
Weinstein, Samuel, '37, 441 Ocean av., Brooklyn 26, N. Y. ALR
Weiss, Harry, '40, ..37-21 Parsons Blvd., Flushing, N. Y. OP
Weiss, J. Allan, '32
58 E. Washington st., Chicago 2, Ill. ALR

Weiss, Leon M., '46....30 E. 60th St., New York 22, N.Y. OP
Weiss, Louis, '46....7085 Jenkins Arcade, Pittsburgh, Pa. ALR
Weiss, Sidney, '44
1930 Chestnut st., Philadelphia 3, Pa. OP
Weisser, Chas. Wm., '42..806 May bldg., Pittsburgh 22, Pa. OP
①Weisser, E. A., '17.....May bldg., Pittsburgh 22, Pa. OP
Weizenblatt, Sprinza, '38
New Medical bldg., Asheville, N. C. OP
Welch, Robert Francis, '46..232 Main St., Brockton, Mass. ALR
②Welch, Robert S. G., '22..86 State Circle, Annapolis, Md. OALR
Weldon, Wm. A., '33.....Glasgow, Ky. OP
Wellford, B. R., '43
510 Medical Arts bldg., Richmond 19, Va. ALR
①Wells, David W., '08, 31 Bay State rd., Boston 15, Mass. OP
Welt, Bernard, '43, 135 New York av., Brooklyn 16, N. Y. ALR
Wenaas, Elmer J., '38....24 Wick av., Youngstown, Ohio OP
Wencke, Carl G., '25
1015 Security Tower, Battle Creek, Mich. ALR
Wendel, J. S., '23.David Whitney bldg., Detroit 26, Mich. ALR
Wenner, W. T., '30....St. Mary's bldg., St. Cloud, Minn. OP
Wescott, Virgil, '35....30 N. Michigan av., Chicago 2, Ill. OP
Weseman, Chester M., '46
2140 Shattuck Ave., Berkley, Calif. ALR
Wesson, Thomas W., '43, 124 S. E. 1st., Evansville 8, Ind. ALR
Westervelt, H. O., '32
239 Pipestone, Benton Harbor, Mich. ALR
②Westlake, Samuel B., '18
Beaumont Med. bldg., St. Louis 8, Mo. ALR
Wetzel, John O., '30
1012 N. Riverside Drive., St. Clair, Mich. OP
Wetzel, Roy J., '44.....301 Broadway, Hanover, Pa. ALR
Wexler, David, '30.....103 E. 86th, New York 28, N. Y. OP
Weymuller, Ernest A., '36
69 E. 76th st., New York 21, N.Y. ALR
Whalen, Edward J., '28.....750 Main, Hartford 3, Conn. ALR
Whalman, Harold Frederic, '40
R. 1147, 727 W. 7th st., Los Angeles 14, Calif. OP
Wheeler, Maynard C., '35, 30 W. 59th, New York 19, N. Y. OP
Wheeler, Merritt W., '23..Lowry bldg., St. Paul 2, Minn. OALR
Whelan, G. L., '25.....2100 Walnut, Philadelphia 3, Pa. ALR
Whelan, William F., '34..2004 Walnut, Philadelphia 3, Pa. ALR
①Whisnant, A. M., '13,
R.F.D. No. 2, Park Rd., Charlotte 3, N. C. OALR
①Whitaker, Joel, '17
807 Hume-Mansur bldg., Indianapolis 4, Ind. OALR
White, Carl Harper, '42....106 River st., Fenton, Mich. OALR
White, Clarence Hunt, '42.....Box 257, Henderson, N. C. ALR
White, Francis W., '37..471 Park av., New York 22, N. Y. ALR
White, Herbert T., '37.....National bldg., Flint 3, Mich. OP
White, J. Warren, '37, Medical Arts bldg., Norfolk 10, Va. OALR
White, Vivian P., '31
524 So. 8th East, Salt Lake City 2, Utah ALR
Whitmer, Kenneth Sweet, '46
711 du Pont bldg., Miami 32, Fla. OP
Whitney, E. L., '30..Henry Ford Hosp., Detroit 2, Mich. OP
Whitney, P. T., '36.....390 Main, Worcester 8, Mass. OP
Whitsell, Fay M., '37.....826 E. 61st St., Chicago, Ill. OP
Whitworth, John Ewing, '41....116 Hammond, Bangor, Me. ALR
Wible, LeRoy E., '35.....3227 5th ave., Pittsburgh, Pa. ALR
Wickstrom, Otto William, '46, Capt. (MC) USN
U.S.N. Hosp. Quarters M, Great Lakes, Ill. ALR
①Wiener, Alfred, '08..550 Park av., New York 21, N. Y. OALR
①Wiener, Meyer, '12
Box 685, 321 Alameda blvd., Coronado, Calif. OP
Wieselthier, Maurice L., '42
1322 Union st., Brooklyn 13, N. Y. OP
Wiest, Philip R., '43.....1036 Franklin st., Reading, Pa. ALR
Wilber, Inez Edith, '40
1801 K st., N.W., Washington 6, D.C. OP
①Wilbur, Edward P., '03
438 W. South, Kalamazoo 46, Mich. OALR
Wilcox, John Guinan, '46
624 N. Los Angeles St., Anaheim, Calif. ALR
Wilcox, Malcolm B., '43....5 West 21st St., Kearney, Nebr. ALR
Wilcoxen, William B., '45.....Bowling Green, Mo. ALR
Wild, Joseph Peter, '45
161 W. Wisconsin Ave., Milwaukee 3, Wis. ALR
Wiley, Jason L., '39.....Metcalf bldg., Auburn, N. Y. OP
Wilkerson, Wm. W., Jr., '32
Bennie-Dillon bldg., Nashville 3, Tenn. ALR
Wilkins, Ellis LeRoy, '42
McWilliams bldg., Clarksdale, Miss. ALR
Wilkins, Samuel H., '34
270 Commonwealth av., Boston 16, Mass. OP
Wilkinson, A. P., '30
3001 W. Grand blvd., Detroit 2, Mich. OALR
Wilkinson, Joel A., '37
P. O. Box 1526, Shreveport 93, La. OALR
Wilkinson, Oscar, '30
1408 L st. N. W., Washington 5, D. C. OALR
Wilkinson, Richard W., '34
1408 L st. N. W., Washington 5, D. C. ALR
Wilkinson, Wallace B., '39..521 E. 10th, Dallas 8, Texas ALR
Williams, Henry L., '37....Mayo Clinic, Rochester, Minn. ALR
②Williams, Horace J., '20
5908 Greene, Germantown, Philadelphia 44, Pa. ALR
Williams, John W., '43,
642-660 Medical Arts Bldg., Springfield, Mo. ALR
Williams, M. M., '27..Medical Arts bldg., Scranton 3, Pa. OP
Williams, Mortimer H., '39,
711 Medical Arts bldg., Roanoke, Va. ALR
Williams, Robert J., '41....25 Washington, Monroe, Mich. ALR
Williams, Russell I., '43, 422 Hynds bldg., Cheyenne Wyo. ALR
②Williams, Walton H., '21, Castell bldg., Middletown, Ohio OALR
Willison, Eugene E., '39
Merced Clinic bldg., Merced, Calif. OP
Willson, Wesley W., '28
6777 Hollywood Blvd., Hollywood 28, Calif. OALR
Wilmes, M. N., '43,
960 Paulsen Medical & Dental bldg., Spokane, Wash. ALR
Wilner, Arthur S., '44..225 West State st., Trenton, N. J. ALR
Wilson, Alfred, '39
2910 W st., S. E., Washington 20, D. C. OP
Wilson, Alpheus K., '38
334 St. James bldg., Jacksonville 2, Fla. ALR
Wilson, Clinton A., '40
609 S. Grand av., Rm. 814, Los Angeles 14, Calif. OP
Wilson, Frank E., '33.....302 State, New London, Conn. ALR
Wilson, George E., '38....108 Main, Saranac Lake, N. Y. ALR
Wilson, Harold A., '41, 71 Maiden Lane, Kingston, N. Y. ALR
Wilson, Hudson J., '36
1st National Bank bldg., Ithaca, N. Y. ALR
Wilson, James Edward, '46
899 Madison Ave., Memphis 3, Tenn. OP
Wilson, John McCullough, '46
207 Watauga Ave., Johnson City, Tenn. OP
Wilson, Otis M., '42.....502 3rd st., Wausau, Wis. OP
Wilson, Paul E., '39.....408 Franklin, Clarksville, Tenn. OP
Wilson, Robert G., '31....304 East Main, Batavia, N. Y. ALR
Wilson, Warren Andrew, '46
1930 Wilshire blvd., Los Angeles 5, Calif. OP
Wilson, Wm. Hildebrand, '42,
903 Republic bldg., Denver 2, Colo. ALR
Wimmer, Shirley D., '41
153 So. Lasky Drive, Beverly Hills, Cal. ALR

Wincor, Henry G., '31
1882 Grand Concourse, New York 57, N. Y. OP
Windham, R. E., '28
234 W. Beauregard blvd., San Angelo, Texas OALR
Winer, Albert S., '43
1835 Eye St., N. W., Washington 6, D. C. ALR
Winger, Ira B., '27.....709 Madison av., Toledo 2, Ohio OALR
Winkler, Herman, '34....224 Thayer, Providence 6, R. I. ALR
Winn, Thomas M., '39, 129½ Maple av., C[illegible]gton, Va. OALR
Winn, William R., '38.....614 N. Fourth, Tucson, Ariz. ALR
Winston, Julius, '36..1737 Chestnut st., Philadelphia 3, Pa. ALR
②Winter, George E., '22
61 All States Court, St. Petersburg, Fla. OALR
Winter, John A., '23.....205 W. 2nd, Duluth 2, Minn. OALR
①Wise, Ralph C., '14.....News bldg., Mansfield, Ohio OALR
Wiser, Henry J., '41, Capt. (M.C.)
503 Dream's Landing, Annapolis, Md. ALR
Wishart, David Edmund Staunton, '42
Medical Arts bldg., Toronto 5, Can. ALR
Witchell, Ira S., '46
2021 Grand Concourse, New York 53, N.Y. ALR
Witter, Gordon Lynn, '46..2 W. Fern Ave., Redlands, Calif. OALR
Wojniak, Frank, '43, 4649 S. Ashland av., Chicago 9, Ill. ALR
Wolcott, Charles C., '31..23 Kraft ave., Bronxville 8, N.Y. ALR
Wold, K. C., '24
Lowry Med. Arts bldg., St. Paul 2, Minn. OALR
Wolf, Lewis R., '43
3474 Frankford av., Philadelphia 34, Penn. OP
Wolfe, Hugh C., '37....102 N. Elm, Greensboro, N. C. ALR
②Wolfe, Otis R., '19
Masonic Temple, Marshalltown, Iowa OP
Wolfe, William C., '41
1012 Heyburn bldg., Louisville, Ky. ALR
Wolff, Jerome Marshall, '45..822 Park Ave., Plainfield, N.J. OP
Wolpaw, Benjamin J., '36,
2323 Prospect av., Cleveland, Ohio OP
Womack, David R., '33,
1218 Maison Blanche bldg., New Orleans 16, La. ALR
Wong, Robert Tuck, '46..1147 Union St., Honolulu, T.H. OP
Wood, Earl LeRoy, '28,
225 Ballantine Pkwy., Newark 4, N. J. ALR
Wood, Edward Christopher, '35
175 Main St., White Plains, N. Y. OP
Wood, Everet H., '40.....120 Genesee St., Auburn, N.Y. OP
Wood, George Harden, '45..Petroleum bldg., Big Spring, Tex. ALR
Wood, Maynard A., '44..1037 Stuart bldg., Lincoln, Nebr. OP
Wood, Spencer, '37.....500 Verret, New Orleans 15, La. ALR
Wood, V. Visscher, '28,
6368 Washington, University City 5, Mo. ALR
Woodman, Degraaf, '33, 156 E. 37th, New York 16, N. Y. ALR
Woodrow, Jack H., '41....27 Ludlow st., Yonkers 5, N.Y. ALR
①Woodruff, Frederick E., '13
Metropolitan bldg., St. Louis 3, Mo. OP
Woodruff, George H., '32....250 N. Ottawa, Joliet, Ill. ALR
①Woodruff, H. W., '11.....250 N. Ottawa, Joliet, Ill. OALR
Woodry, Norman Lee, '36
1501 David Whitney bldg., Detroit 26, Mich. ALR
Woods, Alan C., '39
Johns Hopkins Hospital, Baltimore 5, Md. OP
①Woods, R. H., '15.....153 Marquette, La Salle, Ill. OP
Woodson, B. Palmer, '28
817 Scarbrough bldg., Austin 22, Tex. OALR
Woodson, Warren B., '33
601-22 Professional bldg., Temple, Tex. OALR
Woodward, F. D., '29
104 E. Market, Charlottesville, Va. ALR
Woodward, John F., Jr., '36
Goodhue bldg., Beaumont, Texas ALR
Woodward, Paul Maxwell, '41
4140 Hamilton av., Cincinnati 23, Ohio ALR
Woodworth, Wm. P., '27
2501 W. Grand blvd., Detroit 8, Mich. OALR
Woolfolk, William L., '43, 115 E. 4th st., Owensboro, Ky. ALR
Woolsey, William Jinkens, '42
212 N. 7th., Waco, Texas OP
Worcester, George F., '41....220 Engle, Englewood, N. J. ALR
Work, Walter P., '43..384 Post st., San Francisco 8, Calif. ALR
Workman, Joseph B. Jr., '46..1515 Bull St., Columbia, S.C. OP
Wotring, John M., '39.....220 N. 6th, Reading, Pa. OP
Wright, Edward S., '38,
410 Medical Arts bldg., Atlanta, Ga. ALR
①Wright, J. Wm., '14
301 Hume-Mansur bldg., Indianapolis 4, Ind. ALR
Wright, R. Percy, '32,
1414 Drummond st., Montreal, Can. ALR
Wright, R. Wesley, '45..300 Homer ave., Palo Alto, Calif. ALR
Wright, Walter W., '26
Medical Arts bldg., Toronto 5, Can. OP
①Wurtz, W. J. M., '13..333 Linwood av., Buffalo 9, N. Y. ALR
①Wylie, C. B., '10.....Box 224, Morgantown, W. Va. OALR
Wynn, Sidney K., '44
759 No. Milwaukee St., Milwaukee 2, Wis. ALR

## Y

Yasuna, Elton R., '44.....44 Pearl St., Worcester, Mass. OP
Yavelow, Charles S., '41,
117 South Second av., Mt. Vernon, N. Y. ALR
②Yazel, Herman E., '20..Rialto bldg., Kansas City 6, Mo. OALR
Yinger, Starling C., '41
First Nat'l Bank bldg., Springfield, Ohio ALR
Yost, Walter M., '28.....Lake av., Greenwich, Conn. OP
Young, Charles A., '26,
409 Medical Arts bldg., Roanoke, Va. OP
Young, Clarence M., '35, P.O. Box 1506, Medford, Ore. ALR
Young, Clifton Aurelius, '46, Capt. (MC) USN
U.S. Naval Hosp., Great Lakes, Ill. ALR
Youngerman, Wm. M., '38
Christie Clinic, 104 W. Clark St., Champaign, Ill. ALR
Youngs, N. A., '36
Grand Forks Clinic, Grand Forks, N. D. ALR
Yudkin, Arthur M., '37, 257 Church, New Haven 10, Conn. OP

## Z

Zacks, Myron A., '39..1737 Chestnut, Philadelphia 3, Pa. ALR
Zavalia, Alberto Urrets, '45
27 de Abril 244, Cordoba, Argentina OP
Zbar, Joseph E., '42..2690 Boulevard, Jersey City 6, N. J. ALR
②Zehnder, A. Charles, '19
188 Roseville av., Newark 7, N. J. OP
Zekman, Theodore N., '38
58 E. Washington St., Chicago 2, Ill. OP
Zeman, Michael S., '43,
202-3 Professional bldg., 179 Allyn st., Hartford 3, Conn. ALR
①Zemer, S. G., '17.....Stuart bldg., Lincoln 8, Nebr. OALR
②Zentmayer, Wm., '21..1930 Chestnut, Philadelphia 3, Pa. OP
Zimmerman, J. Landis, '37....207 State, Harrisburg, Pa. OP
②Zinkhan, A. M., '20
1801 Eye st. N. W., Washington 6, D. C. ALR
Zinn, Waitman Farnsworth, '40
Medical Arts bldg., Baltimore 1, Md. ALR
Zonderman, Bernard, '46..29 Bay State Road, Boston, Mass. ALR
Zubak, Matthew F., '28
704-9 Central Union bldg., Wheeling, W. Va. OALR
Zuckerman, Joshua, '36..875 Park av., New York 21, N. Y. OP
Zwerling, Samuel, '36
789 St. Mark's av., Brooklyn 13, N. Y. ALR

# Geographical

## ALABAMA

### BIRMINGHAM

Callahan, Alston.....Medical College of Alabama, Z5 OP
Clements, Frank Hartman.....806 Medical Arts bldg. OP
Fisher, Gilbert Eugene.....Medical Arts bldg., Z5 ALR
Gordon, George Raoul.....913 S. 19th St. ALR
Hardy, Walter B.....2121 Highland av., Z5 ALR
Kincannon, LeRoy T.....2300 Highland Ave., Z5 OALR
Locke, William Walter.....507 Empire bldg. ALR
Pope, Ernest C.....2021 Sixth av. N., Z3 ALR
Rucker, E. W., Jr.....Woodward bldg., Z3 ALR
Tyler, Richard E.
The Norwood Clinic, 2501 N. 16th av., Z4 ALR

### DOTHAN

Keyton, John A.....304 N. Oates ALR

### GADSDEN

Frank, Herman W.....Medical bldg. OP
Isbell, Euclid A.....948 Forrest av. ALR

### JASPER

Simmons, John T.....1504 Alabama ave. ALR

### MOBILE

Martin, Henry F.....904 Government St. Z20 ALR
Perdue, James D.....P. O. Box 514, Z4 OP
Sanders, J. Gillis...714 Merchants Nat'l Bank bldg., Z11 ALR

### MONTGOMERY

Benkwith, Karl B.....209 Lexington rd. OP
Hagood, Daniel S.....822 First Nat'l Bank bldg., Z4 OP
Holding, Bruce F.....Bell bldg., Z4 ALR
Jones, John Allen.....401 S. Court St. ALR
Mertins, Paul, Jr.....32 Clayton St. ALR
Nodine, Edward R.....1110 Bell bldg., Z4 ALR
Thigpen, C. A.....401 S. Court, Z5 OALR

### TUSCALOOSA

Searcy, H. B.....2501 Sixth OALR

## ARIZONA

### PHOENIX

Brinkerhoff, David E.....Grunow Clinic ALR
Case, Paul H.....15 E. Monroe OP
Cruthirds, Archie Edward.....Professional bldg. ALR
Melton, B. L., Jr.....15 E. Monroe, OP
Robb, Mayo.....Professional bldg. ALR
Schwartz, William A.....320 Professional bldg. OALR
Toland, Virgil A.....509 W. McDowell Rd. OP

### PRESCOTT

Swetman, C. R. K.....219 S. Mt. Vernon ALR

### TUCSON

Brown, Earl Henderson.....130 S. Scott ALR
Kinkade, Joseph M.....45 E. Jackson st. ALR
Winn, William R.....614 N. Fourth ALR

## ARKANSAS

### EL DORADO

Russell, Martin V.....200 W. Elm st. ALR

### FAYETTEVILLE

Ogden, Frederick W.....130 S. Duncan st. ALR

### FORT SMITH

Faier, Samuel Z.....1425 No. 11th st. OALR
Moulton, Everett C.....Merchants National Bank bldg. OALR
Moulton, Herbert.....Merchants National Bank bldg. OALR

### HOT SPRINGS

King, O. H.....Medical Arts bldg. OALR

### LITTLE ROCK

Calcote, Royal J.....541 Donaghey Bldg. OP
Caldwell, Robert.....New Donaghey bldg. OALR
Cook, Raymond C.....1005 Donaghey Bldg. OP
Cosgrove, Kingsley White.....115 East Capitol OALR
Fein, Normal Nathaniel.....Trinity Hospital ALR
Mahoney, Paul Leo.....Donaghey bldg. ALR

### PINE BLUFF

Payne, Virgil LeRoy.....Simmons National bldg. ALR

## CALIFORNIA

### ALAMEDA

Smithies, Harold R.....2241 Central av. ALR

### ALHAMBRA

Johnson, Ernest D.....317 W. Main OALR

### ANAHEIM

Wilcox, John Guinan.....624 N. Los Angeles st. ALR

### BANNING

Harner, Clyde E.....491 W. Gilman St. OALR

### BERKELEY

Ripsteen, Joel Stanley.....2527 Etna st. ALR
Weseman, Chester M.....2140 Shattuck Ave. ALR
Gunderson, Ernest O.....2511 Hearst Ave. OP

### BEVERLY HILLS

Cooperman, Harold O.....416 No. Bedford OP
Dow, Julian Neal.....9730 Wilshire blvd. OP
Goldberg, Sylvan S.....Suite B, 121 N. San Vicente bldg. ALR
Hartzell, Sol M.....137 So. Peck Drive OALR
Irvine, A. Ray.....9730 Wilshire Blvd. OP
Irvine, S. Rodman.....9730 Wilshire Blvd. OP
Lemere, H. B.....9615 Brighton Way OALR
Morris, S. A.....405 N. Bedford dr. OP
Myerson, Mervin C.....416 N. Bedford dr. ALR
Romonek, Philip L.....415 N. Camden dr. OALR
Seligman, Maurice.....405 N. Bedford dr. ALR
Semenov, Herman.....415 N. Camden dr. ALR
Shumaker, Phil W.....9730 Wilshire blvd. ALR
Wimmer, Shirley D.....153 S. Lasky dr ALR

## CALIFORNIA — Continued

CARMEL
Adams, Frederick Wm.....OALR
Walker, John R.....P. O. Box M-1 OP

CORONADO
Wiener, Meyer.....Box 685, 321 Alameda blvd. OP

EL CENTRO
Edwards, S. R.....107 S. Fifth OALR

FONTANA
Hunt, Wm. Remfry.....Southern Permanente Hosp. ALR

FRESNO
Awtrey, Hugh H.....533 Patterson bldg. OALR
Olson, George W.....1759 Fulton, Z1 ALR
Smith, W. Jewell.....2014 Tulare st. Z1 ALR
Trowbridge, Dwight H.....T. W. Patterson bldg., Z1 OP
Walker, George W.....630 Peralta Way ALR

GLENDALE
Donohoe, Edward C.....102 N. Brand blvd. ALR
Trott, Leslie De Nyse.....143 North Brand, Z3 ALR

HOLLYWOOD
Cobb, Edwin.....1680 N. Vine St. OALR
James, Voyle.....6253 Hollywood blvd. ALR
King, Edward.....6777 Hollywood Blvd. Z28 ALR
Mulligan, Harold R.....1680 North Vine St. Z28 ALR
Naftzger, J. B.....6777 Hollywood blvd., Z28 OALR
Willson, Wesley W.....6777 Hollywood Blvd. Z28 OALR

HUNTINGTON PARK
Appelbaum, Alfred.....5905 Pacific Blvd. OP

LA HONDA
McCurdy, Gordon Judson.....General Delivery ALR

LA JOLLA
Roberts, William H. (Retired).....335 Westbourne St. OALR

LA MESA
Sherman, A. Edward
Lt. Col. (M.C.) (Retired), Rt. 1, Box 1401 OALR

LONG BEACH
Brandenburg, Kenneth C.....110 Pine av., Z2 OALR
Godwin, Dean E.....117 E. 8th St., Z2 ALR
Ogden, J. C.....Professional bldg., Z2 OP
Richman, Frances B., Lt. Comdr. (MC) US Naval Hosp., Z4 OP
Smith, Dennis V.....110 Pine st. OP
Southgate, Paul T.....302 Professional bldg. OP
Thornburgh, Robert G.....Professional bldg., Z2 OP

LOS ANGELES
Abraham, Samuel V.....1930 Wilshire blvd., Z5 OP
Albaugh, Clarence H.....727 W. 7th (suite 1131), Z14 OP
Anderson, Ernest Richard V.....1052 W. Sixth st. Z14 ALR
Beauchamp, Mark L.....1136 W Sixth St. Z14 ALR
Boyce, William A.....Roosevelt bldg., Z14 OP
Boyd, Harold M.E.....1136 W. 6th St. Z14 ALR
Brown, J. M.....1136 W. 6th, Z14 ALR
Brownsberger, Sidney B.....727 W. 7th St. Z14 OP
Bryant, Ben L.....3875 Wilshire blvd. Z5 ALR
Bullis, John A. E.....3875 Wilshire blvd, Z5 OALR
Burston, Herschel.....1917 Wilshire blvd., Z5 ALR
Campbell, Glenn S.....340 No. Oxford ave. Z4 ALR
Christensen, Eugene L.....727 W. 7th st., Z14 OP
Colver, Benton Noble.....436 S. Boyle av., Z33 ALR
Crane, Walter Raymond....Room 1027, 727 W. 7th, Z14 ALR
Dayton, G. O.....1401 S. Hope, Z15 OALR
Dexter, Milton A.....1618 Westwood blvd. Z24 ALR
Donoher, William D.....635 S. Westlake av., Z5 OALR
Ellis, Orwyn H.....727 W. 7th St. Z14 OP
Faier, H. I.....1930 Wilshire blvd., Suite 203 Z5 OP
Fairchild, Nora M.....939 S. Figueroa, Z15 OP
Fraser, Robert C.....6777 Hollywood blvd. Z28 ALR
Goodhill, Victor.....676 S. Westlake, Z5 ALR
Gorrilla, Laurence Vincent.....511 So. Bonnie Brae st. OP
Gosney, C. W.....5217 Hollywood blvd., Z27 OP
Greenwood, Glenn J.....154 Medio Dr. Z24 ALR
Guggenheim, Louis K.....321 Glenroy av., Z24 ALR
Gundrum, Lawrence Kramer.....1930 Wilshire blvd., Z5 ALR
Hall, Colby.....1136 W. 6th st. ALR
Hara, H. James.....436 S. Boyd Ave., Z3 ALR
Hartman, Deane C.....727 W. Seventh st., Z14 OP
Hillyer, Ernest C.....1033 Gayley Ave. Z24 OP
Ho, Wayne Y. H....Gotham Hotel, 975 Ingraham st. Z14 ALR
Holden, Harold M.....3875 Wilshire Blvd. Z5 ALR
House, Howard P.....1136 W. 6th st., Z14 ALR
House, Leland R.....1727 New Jersey st., Z33 ALR
Jesberg, Simon.....500 S. Lucas av., Z13 ALR
Koff, Raphael Joseph.....1917 Wilshire blvd. Z5 OP
Kress, George H.....131 S. Rampart blvd., Z4 OALR
Kully, B. M.....3875 Wilshire blvd., Z5 ALR
Landegger, George P. 860 Roosevelt Bldg.,
727 W. 7th St. Z14 OP
Lefler, Anna B.....2419 W. 18th St. Z6 OALR
Lordan, John P.....1930 Wilshire blvd., Z5 OP
Marks, Julian B.....1093 Broxton av., Z24 ALR
McLaughlin, R. C.....3780 Wilshire blvd., Z5 ALR
Miller, Alden H.....500 S. Lucas ave. ALR
Mogan, Richard F.....523 West 6th st., Z14 ALR
Monaco, Louis.....1100 Roosevelt bldg., Z14 OALR
Mumma, C. S.....U. S. Veterans' Facility, Z25 ALR
Nugent, Maurice W.....1930 Wilshire blvd., Z5 OP
Olkon, Dora B.....8015 Beverly blvd., Z36 OP
Owens, Harold.....1052 W. 6th st., Z14 ALR
Owsley, Robert W.....1136 W. 6th st. ALR
Packard, Louis A.....523 W. 6th st., Z14 OALR
Peluse, Samuel.....1930 Wilshire blvd., Z5 ALR
Popovich, Stephen John.....1930 Wilshire blvd. Z5 OP
Pressman, Joel J.....615 N. Faring Road, Z24 ALR
Reed, Paul Henry.....727 W. 7th St. Z14 OP
Rogers, John B.....1401 S. Hope, Z15 OP
Rome, Sol.....1917 Wilshire blvd. Z5 OP
Rubin, Henry J.....8667½ Burton Way Z36 ALR
Schroeder, Milo Carl.....2112 Colorado blvd., Z41 ALR
Scobee, J. Edwin.....6253 Hollywood blvd. Z28 ALR
Sitney, Julian Jay.....2007 Wilshire blvd., Z5 OP
Sommer, Ignatius George.....981 S. Westmoreland Z6 OALR
Turnbull, F. M.....Wilshire Medical bldg., Z5 ALR
Wall, John William.....509 S. Ogden Dr. Z36 ALR
Weaver, Emerson M. F.....727 W. 7th st., Z14 OP
Weeks, Carrol Lamont.....1930 Wilshire blvd., Z5 OP
Whalman, Harold Frederic ....1147-727 W. 7th, Z14 OP
Wilson, Clinton A.....609 S. Grand Ave. Rm. 814, Z14 OP
Wilson, Warren Andrew.....1930 Wilshire blvd., Z5 OP

MERCED
Willison, Eugene E.....Merced Clinic bldg. OP

MONTEREY
Clark, Howard E.....576 Hartnell st. OALR

## CALIFORNIA — Continued

**OAKLAND**

Gump, Millard E.....411 30th St. Z9 OP
Howell, Homer P.....2709 Alida st. OP
Keeler, Nelson S.....411 Thirtieth, Z9 ALR
McClure, George.....411 Thirtieth, Z9 ALR
Nutting, Raymond J.....411 Thirtieth, Z9 OP
O'Connor, Roderic P.....411 30th st. OP

**ONTARIO**

Hooval, J. H.....213 Fallis bldg. ALR

**OXNARD**

Raines, M. M.....226 So. C st. ALR

**PACIFIC PALISADES**

Shellman, John L.....Box 2 OP

**PALM SPRINGS**

Chapman, Vernon A.....435 N. Palm Canyon drive OALR

**PALO ALTO**

Sewall, E. C.....Stanford Univ., 430 El Escarpado ALR
Tanner, Owen Ralph.....627 University ave. OP
Wright, R. Wesley.....300 Homer ave. ALR

**PASADENA**

Armstrong, Richard C.....623 First Trust bldg. Z1 OP
Balding, Laurence Grant.....93 S. Madison ave., Z5 OP
Cady, D. W.....65 N. Madison av., Z1 OP
Currier, Wilber D.....65 N. Madison av., Z1 ALR
Decker, Russell M.....65 N. Madison av., Z1 ALR
Dewey, C. H.....760 S. Oak Knoll ave. OALR
Dysart, Ben Robnett.....65 N. Madison av., Z1 ALR
Esterly, Daniel Beard.....104 No. Madison, Z4 OP
Greetham, James S., Maj. (MC), McCornack Gen. Hosp. Z2 OP
Henninger, L. L.....65 N. Madison av., Z1 OALR
Henry, L. L.....595 E. Colorado st., Z1 OP
Hunnicutt, Leland G.....275 S. Holliston ave. Z5 ALR
Johnson, Fordyce A. H.....98 No. Madison, Z4 ALR
Johnson, Henrietta May.....595 E. Colorado, Z1 OP
Miller, Theodore E.....First Trust bldg., Z1 ALR
Morgan, George E.....65 No. Madison, Z1 OP
Pohlman, David Alexander.....595 E. Colorado st. Z1 ALR
Reed, James Ross.....201 N. El Molino av., Z4 OP
Risser, E. D.....423 First Trust bldg., Z1 OALR
Scanlon, Wilko Gruver.....201 N. El Molino av., Z4 ALR

**PLACERVILLE**

Spencer, George Albert.....Rt. No. 1, Box 42 OALR

**POMONA**

Roberts, Gilbert Jay.....586 N. Main st. ALR
Smith, Harold D.,.....540 Investment bldg. ALR

**REDLANDS**

Witter, Gordon Lynn.....2 W. Fern Ave. OALR

**REDWOOD CITY**

Simons, Harold.....10 California st. OP

**SACRAMENTO**

Goldthwaite, Ralph H., Brig. Gen. (MC) (Retired)
4515 Mead ave. OALR
Holstein, Theodore.....1800 Sutterville road OP
Swan, Otis Dwight....1035 Forum bldg., 9th & K, Z14 ALR
Tillotson, R. S.....Medico-Dental bldg., Z14 ALR
Wachsberger, Alfred.....1025 9th st. ALR

**SALINAS**

Glasgow, S. Condit.....8 E. Alisal st. ALR

**SAN BERNARDINO**

George, Arthur R.....291 "E" st. ALR

**SAN DIEGO**

Bond, Floyd M.....625 Broadway, Z1 OP
Brown, Charles Wm.....Bank of America bldg., Z1 ALR
Brownell, Durwin H.....3520 Third av., Z3 ALR
Dennis, F. L.....2745 Barnson place, Z3 ALR
Durr, S. A.....511 San Fernando, Z6 OP
Henrickson, John William.....2001 Fourth Ave. ALR
Higbee, D. R.....3245 4th av., Z3 ALR
Jones, Edward Earl, San Diego Co. Gen. Hosp.
North End of Front St. Z3 ALR
Kilgore, George L.....411 Thorn St. Z3 OP
Koke, Martin P.....233 A. st. OP
Maghy, Charles A.....Medico Dental bldg. Z1 OP
Merrill, H. G.....3245 4th av., Z3 OP
Monsees, Wayne Emerson.....233 A St. Z1 OP
Prendergast, John J.....2001 4th av., Z1 OP
Rees, Charles W.....4496 Trias st., Z3 ALR
Smith, Alma C., Capt. (MC) USN, U.S. Naval Air Station OALR

**SAN FRANCISCO**

Ashley, Rea.....384 Post st. ALR
Barkan, Hans.....2351 Clay st., Z15 OP
Barkan, Otto.....490 Post, Z2 OP
Baron, Shirley Harold.....516 Sutter st. Z2 ALR
Borley, Wm. E.....450 Sutter bldg. OP
Bunnell, Chester Wm.....1316 27th ave. ALR
Cordes, Frederick C.....384 Post, Z8 OP
Dickey, Clifford A.....450 Sutter st., Z8 OP
Edgerton, Ambrose E.....450 Sutter, Z8 OP
Fishman, Louis Z., Green's Eye Hosp.,
1801 Bush st., Z9 ALR
Fitzhugh, William McP., Jr.....384 Post, Z8 ALR
Gidoll, Sidney H.....516 Sutter, Z2 ALR
Hand, Frank.....450 Sutter, Z8 ALR
Hearn, Paul P.....384 Post St., Z8 ALR
Hebert, Arthur W.....450 Sutter, Z8 OALR
Hogan, Michael John.....450 Sutter St., Z8 OP
Hopp, Eugene S.....2000 Van Ness Ave. Z9 ALR
Horner, Warren D.....490 Post St., Z2 OP
Irvine, Robert Steele .....490 Post, Z2 OALR
Lachman, George S.....450 Sutter st. Z8 OP
Marks, Roland F.....450 Sutter st., Z8 OP
McCool, J. L.....450 Sutter, Z8 OP
Monson, LaFayette.....450 Sutter, Z8 ALR
Nethercut, Ruth A.....450 Sutter, Z8 ALR
Parkinson, Harold O.....626 Flood bldg. ALR
Pierce, George W.....490 Post st., Z2 PI S
Pischel, D. K.....490 Post, Z2 OP
Pischel, Kasper.....490 Post, Z2 OP
Rawlins, Aubrey Gatliff.....384 Post, Z8 ALR
Rodin, Frank Highman.....490 Post, Z2 OP
Selfridge, G. L.....1000 California st., Z8 ALR
Sooy, Francis A.....Suite 808, 490 Post St. ALR
Spiller, Arthur Eliot..c/o Sutter Hospital, Sutter St., Z9 OP
Work, Walter P.....384 Post st. Z8 ALR

**SAN JOSE**

Jordan, Philip J.....910 Medico-Dental bldg., Z20 ALR
Martin, Paul T.....235 E. Santa Clara, Z20 ALR
Thygeson, Phillips.....524 Sainte Claire bldg. Z23 OP

## CALIFORNIA — Continued

SAN MATEO
Alter, Francis William, Jr.....23 Second Ave. ALR
Mohun, Meade.....23 Second av. ALR

SAN PEDRO
Earl, Harry D.....1501 LeGrande Terrace ALR
Snow, Harold L.....639 W. 9th OP

SANTA ANA
Dixon, W. E.....1617 E. 4th OALR
Maxwell, Harvey C.....1718 N. Main st. OALR

SANTA BARBARA
Gibb, William B.....1515 State st. OP
Hombach, Frank J.....1421 State OP
Jean, George W.....1415 Laguna st. OP
Johnston, William H.....1515 State ALR
Johnston, W. R.....1515 State st. ALR
Lewis, J. D.....650 Hot Springs Dr. OALR
Loutfallah, Michel.....1826 State st. OP
Mellinger, William J.....323 E. Sola st. ALR
Olsen, Arthur Roy.....1421 State St. ALR
Palevsky, Samuel N.....22 E. Los Olivos st. ALR
Rhode, Solon L.....P.O. Box 1385 OALR
Von Zelinski, Walter Franz.....3015 Hermosa rd. OP

SANTA MARIA
Mesirow, Maurice E.....117 E. Cook OP

SANTA MONICA
Adams, Eldridge S., Col. (MC), c/o C.A.A., 1500 4th St. ALR
Ross, Cecelia.....710 Wilshire blvd. OP

SANTA PAULA
Howarth, Ervin McIntyre.....705 Main st. ALR

SHERMAN OAKS
Gradle, Harry S.....14060 Valley Vista rd. OP

SOUTH PASADENA
Hall, Lawrence H.....1520 Oak st. OALR

STOCKTON
Brody, Yale.....919 Bank of America bldg. OP
Saslaw, Lewis Benjamin.....Bank of America bldg., Z5 ALR

VALLEJO
Madeley, Howard Randall.....727 Sonoma OP

VENTURA
Helbling, Franklin Kirkland.....34 No. Ash St. ALR
Mosher, Walter F.....34 N. Ash st. ALR

VISALIA
Keiper, Geo. F.....113 N. Church ALR
Sallander, F. W.....212 W. Main OALR

VISTA
Stewart, Charles E., Jr.....Rt. 2, Box 452 ALR

WEST LOS ANGELES
Hess, Wm. L.....2018 Fairburn Z25 OALR
Struble, Gilbert C.....2921 Glendon ave. Z34 OP

WHITTIER
Morris, Francis O'Neill.....820 S. Cainter Ave. ALR
Smith, H. A.....226 N. Greenleaf ave. OP

## COLORADO

BOULDER
Page, Donald Franklin.....Boulder Colorado Sanitarium ALR
Spencer, Frank R.....Physicians bldg. ALR

COLORADO SPRINGS
Bradley, John W.....Burns bldg. ALR
Brobeck, von Haller.....Ferguson bldg. OP
Chapman, Katharine H.....304 Burns bldg. OP
Chapman, S. Jefferson.....315 No. Cascade ave. ALR
Kettelkamp, F. O.....Ferguson bldg. ALR
Lamberson, William Henry....First National Bank bldg. ALR
Neeper, Edward R.....Exchange National Bank bldg. OP
Nelson, Fritz.....1121 N. Tejon st. OP
Stine, George H.....Burns bldg. OP
Walker, C. E.....Rt. 1, Box 17 OP

DENVER
Bane, W. M.....1612 Tremont Place, Z2 OALR
Baum, H. L.....Republic bldg., Z2 ALR
Beyer, Theodore E.....227 Sixteenth st., Z2 ALR
Chessen, James.....510 Republic bldg. ALR
Collins, E. W.....1578 Humboldt, Z6 ALR
Cooper, Kemp G.....Metropolitan bldg., Z2 ALR
Crisp, William H.....Metropolitan bldg, Z2 OP
Culpepper, Wm. S., Col. (MC).....6601 E. 18th ave. ALR
Danielson, Ralph W.....Metropolitan bldg., Z2 OP
Deems, Myers Bowman, Lt. Col.....1601 Leyden st. ALR
Farrington, P. M. .....Colburn Hotel ALR
Filmer, George A.....324 Metropolitan bldg., Z2 OP
Gale, M. Jean.....Republic bldg., Z2 OP
Goldhammer, Samuel.....Republic bldg. Z2 OP
Greene, Laurence W.....Republic bldg., Z2 ALR
Gromer, Terry John.....110 Metropolitan bldg. ALR
Halsted, Fred S.....Metropolitan bldg., Z2 OALR
Hausmann, Gertrude S. ..... 629 16th st., Z2 OP
Hickey, Harold L.....Republic bldg., Z2 ALR
Laff, H. I.....Metropolitan bldg., Z2 ALR
Leyda, James H.....Metropolitan bldg., Z2 ALR
Long, John C.....Metropolitan bldg., Z2 OP
Marcove, Maurice E.....1612 Tremont pl., Z2 OP
McKeown, Elmer E.....Republic bldg., Z2 OALR
Murphy, Rex L.....Metropolitan bldg., Z2 ALR
Ohmart, Walter A.....Republic bldg., Z2 OP
O'Rourke, D. H.....Republic bldg., Z2 OP
Pattee, George L.....612 Republic bldg., Z2 ALR
Philpott, Ivan W.....806 Metropolitan bldg. Z2 ALR
Porter, Whitney C.....320 Republic Bldg. Z2 OP
Reynolds, Edna M.....227 Sixteenth, Z2 OP
Sedwick, W. A.....950 Pearl st. Z3 OP
Shields, J. M.....Metropolitan bldg., Z2 OP
Smith, Guy William.....1014 Republic bldg. ALR
Strong, James C., Jr.....617 Majestic bldg. Z2 OP
Swigert, J. Leonard.....Republic bldg. OP
Van Bergen, Thomas M.....227 16th st. OP
Wilson, Wm. Hildebrand.....903 Republic bldg. Z2 ALR

ENGLEWOOD
Dart, M. O.....2701 So. Marion st. ALR

GRAND JUNCTION
Cary, G. C.....Canon block OALR
Rigg, James P.....Box 928 OALR
Waldapfel, Richard.....De Merschman Gardens ALR

## COLORADO — Continued

GREELEY
Atkinson, Thomas Ernest.....Coronado bldg. ALR
Droegemueller, Wm. H.....1845 11th Ave. OP
Lehan, James W.....Park Place bldg. OALR

LA JUNTA
Farnsworth, M. A.....Opera House Apt. bldg. OALR
Ringle, Charles A.....c/o John Blackmore ALR

LONGMONT
Hageman, George Ricker.....Longmont Hospital ALR
Sidwell, Clarence Edward.....608 Fourth av. ALR

PUEBLO
Earnest, Clarence E.....Thatcher bldg. ALR
Hopkins, Guy H.....702 N. Main OP
Lassen, Fritz.....Pueblo Clinic OALR
Rusk, Harvey Schenck.....Colorado bldg. OALR

SALIDA
Thompson, Lester Emmit.....Woolworth bldg. ALR

STERLING
Elliff, Edgar Alonzo.....216 N. 3rd. ALR
Palmer, Frank E.....123 N. 3rd St. OALR

TRINIDAD
Richie, L. T.....101 E. Main OALR

## CONNECTICUT

BRIDGEPORT
Alpert, Max.....881 Lafayette st. ALR
Cunningham, Bernard P.....106 Center St. Z4 ALR
Dewitt, Edward Nicholas.....881 Lafayette, Z4 OP
Garlick, George B.....144 Golden Hill, Z3 ALR
Kaplan, Leon.....881 Lafayette st., Z4 OP
Kornblut, Alfred.....1539 Fairfield Ave., Z5 ALR
Turchik, Frank.....1831 Barnum av., Z8 ALR

GREENWICH
Miller, John.....63 North ALR
Morrissett, Leslie E.....261 Lake ave. ALR
Tinkess, Donald E.....Stanwich Rd. OALR
Yost, W. M.....Lake av. OP

HARTFORD
Bausch, Carl Philipp.....36 Pearl st. Z3 OP
Birge, Henry L.....179 Allyn st. OP
Chester, Lewis.....179 Allyn St., Z3 ALR
Connor, Joseph Joyce.....750 Main st., Z3 OP
Davis, James Edward.....179 Allyn, Z3 ALR
Harris, Louis D.....242 Trumbull OP
Hogan, W. L.....750 Main, Z3 OP
Katz, Dewey.....99 Pratt, Z3 OP
Lewis, Samuel D.....49 Pearl st., Z3 OP
Little, Milton F.....49 Pearl, Z3 OP
Neidlinger, Wm. J.....576 Farmington ave. Z5 ALR
Schwartz, Herbert Norman.....99 Pratt St. OP
Snelling, P. W.....179 Allyn, Z3 ALR
Truex, Edward H., Jr.....99 Pratt St. ALR
Unsworth, Arthur C.....179 Allyn st., Suite 508 OP
Whalen, Edward J.....750 Main, Z3 ALR
Zeman, Michael S.
202-3 Professional Bldg., 179 Allyn st. Z3 ALR

MIDDLETOWN
Chase, Carl C.....121 Main OP
Joyce, Wm. M.....121 Main st. ALR

NEW BRITAIN
Schechtman, Charles T.....81 W. Main ALR
Tisher, Paul Winslow.....99 W. Main St. OP

NEW HAVEN
Berman, Harry L.....1142 Chapel, Z11 ALR
Canfield, Norton.....789 Howard ave., Z4 ALR
Clarke, Clement C.....Amity Rd., Woodbridge OP
Cobey, James Francis.....1210 Chapel St., Z11 OALR
Flynn, Charles T.....41 Trumbull, Z10 ALR
Latimer, Marvin L.....129 Whitney av. Z10 ALR
MacCready, Paul B.....442 Temple, Z11 ALR
Ryder, William H.....195 Church st., Z10 OP
Sheahan, William L.....59 College, Z10 OALR
Silverberg, Samuel Joshua.....315 Whitney av., Z11 ALR
Smith, Norman N.....291 Whitney av., Z11 ALR
Sperry, Frederick N.....107 Whitney, Z10 ALR
Yudkin, Arthur Meyer.....257 Church, Z10 OP

NEW LONDON
Haines, Henry L.....309 State st. ALR
Wilson, Frank E.....302 State ALR

STAMFORD
Murray, Henry J.....53 South ALR
Turnley, William H.....One Atlantic ALR

TORRINGTON
Hill, E. S.....51 Main ALR
Samponaro, Nicholas.....231 Calhoun St. ALR

WALLINGFORD
Campbell, Sherburne.....270 Center St. OALR

WATERBURY
Good, William Murray.....63 Center, Z18 ALR

WEST HARTFORD
Smith, E. Terry.....P. O. Box 42 OP

WILLIMANTIC
Arnold, Morton.....781 Main ALR

## DELAWARE

REHOBATH BEACH
Daisey, Willard O.....30 Park av. ALR

WILMINGTON
Cutler, Norman L.....1300 Harrison st. OP
LaMotte, William O.....Medical Arts bldg., Z9 OALR
Mayerberg, Emil R.....Medical Arts bldg., Z9 ALR
Pierson, William M.....Medical Arts bldg., Z9 ALR
Strikol, A. J.....621 Delaware av., Z9 ALR

## DISTRICT OF COLUMBIA

WASHINGTON
Albright, Paul M., Rear Adm. (MC) USN
Naval Retiring Board, Navy Dept. Z25 OALR
Alexander, Samuel A.....1830 Eye st. N. W., Z6 ALR
Alfaro, Victor R.....2007 Massachusetts ave. N.W. Z6 ALR

## WASHINGTON, D. C. — Continued

Barnaby, Henry C.....Walter Reed Gen. Hosp. ALR
Bauer, Arthur S.....Suite 812, 1801 Eye st. NW Z6 ALR
Bell, John Gordon.....1835 Eye st. N. W., Z6 ALR
Bockoven, Sterling....1752 Massachusetts av. N. W., Z6 OP
Bradley, Jeter C.....900 Seventeenth st., N. W., Z6 ALR
Breeding, Earle G.....1801 Eye st., N.W., Z6 ALR
Brooks, Richmond C. A.....1150 Connecticut av., Z6 ALR
Burtoff, Samuel.....2025 Eye St. N.W. Z6 ALR
Clayton, Milton B.....Southern R. R. Office bldg., Z13 ALR
Constantinople, P. S.....1801 Eye st., N. W., Z6 ALR
Costenbader, Frank D.....1150 Connecticut av., Z6 OP
Cox, Ronald Atmore..1779 Massachusetts av., N. W., Z6 OP
Cummings, Edward Joseph.....1835 Eye st. N. W., Z6 ALR
Davies, H. F.....1835 Eye st. N. W., Z6 ALR
Davis, David.....1835 Eye st. N. W., Z6 ALR
Dessoff, Joseph.....1726 Eye st. N. W., Z6 OP
Downey, Harold R.....1740 M St., N.W. Z6 OP
Feldman, Irvin.....1722 Eye St. N. W. ALR
Fischer, Aubrey.....1835 Eye st. N. W., Z6 ALR
Flynn, James A.....1511 Rhode Island av. N. W., Z5 ALR
Foote, Wm. D.....1800 Eye St. N.W. OP
Gill, G. Bache.....1726 Eye st. N. W., Z6 ALR
Goodman, E. Leonard.....1801 Eye st. N. W., Z6 OP
Greear, James N., Jr.....1740 M. st., N. W. Z6 OP
Gurwin, Bernard J.....900 17th St. N.W. Z6 OP
Henning, Carl ....1752 Massachusetts av. N. W., Z6 OP
Holmes, Dorothy Behner.....1816 R. St. N.W. Z9 OP
House, Hugh O.....1835 Eye st. N. W., Z6 ALR
Johnson, Don.....1835 Eye st. N. W., Z6 ALR
Kearney, Richard.....2311 Connecticut av., N. W., Z8 ALR
Knowlton, Don S.....408 Columbia Medical bldg., Z6 ALR
Lamb, Robert Scott.....Stoneleigh court, Z6 OP
Lavine, Oscar.....Suite 620, 1801 Eye St. N.W. OP
Luber, Samuel.....1801 Eye st. N. W., Z6 ALR
Meloy, William C.....2019 R. st. N. W., Z9 ALR-PLS
Moffett, Daniel B.....1150 Connecticut av. N. W., Z6 ALR
Morgan, William A.....1835 Eye st. N. W., Z6 ALR
Moss, L. Conner.....2223 Observatory Pl. N.W. OP
Novick, Joel N.....1726 Eye st. N. W., Z6 ALR
Pendexter, R. Stevens.....Farragut Medical bldg., Z6 OP
Rones, Benjamin.....1610 Eye st. N. W., Z6 OP
Runk, R. L.
Washington Sanitarium and Hospital, Takoma Park, Z12 ALR
Sawyer, Leroy Lee, Jr.....1746 K St. N.W. Z6 ALR
Schlanser, A. E., Col. (M.C.)..2245 Mt. View Place S. E. OALR
Schnebly, John Thomas.....900 17th st. N.W. Z6 OP
Shepherd, Elmer Roberts.....1746 K st. N. W., Z6 ALR
Sheppard, Ernest A. W.
George Washington Univ., Dept. of Oph. 1801 K St. N.W. OP
Simpson, G. Victor.....1712 Rhode Island ave. Z6 OP
Stow, M. Noel.....1746 K st. N. W., Z6 OP
Swanson, Clifford A., Adm. (MC) U.S.N.
The Westchester Apts., 836-B, Z16 OALR
Tegge, Charles William.....1801 Eye st. N. W., Z6 ALR
Trible, G. B.....1150 Connecticut av. N. W., Z6 OALR
Vann, Homer King.....410 Cedar st. N. W., Z12 ALR
Wilber, Inez Edith.....1801 K st. N. W., Z6 OP
Wilkinson, Oscar.....1408 L st. N. W., Z5 OALR
Wilkinson, Richard W.....1408 L st. N. W., Z5 ALR
Wilson, Alfred.....2910 W st., S. E., Z20 OP
Winer, Albert S.....1835 Eye st. N.W., Z6 ALR
Zinkhan, A. M.....1801 Eye st. N. W., Z6 ALR

## FLORIDA

BAY PINES
Gross, Julius E.....Veterans Administration ALR

CLERMONT
Middleton, A. B.....OALR

COCONUT GROVE
Hutchinson, Abbott T.....1086 Reynolds av. ALR

CORAL GABLES
Cowell, Edward Huntington.....427 Biltmore Way OP
Hasty, F. E.....1203 Asturia ALR

DANIA
Connor, Arthur B.....P.O. Box 398 ALR

DAYTONA BEACH
Lenholt, Eric H.....101 Lenox av. ALR

FORT LAUDERDALE
Driscoll, Frederick J.....P.O. Box 1653 ALR
Enlows, Ella M. A.....450 N. Victoria Park rd. ALR
McKee, Thomas L.....436 E. Las Olas blvd. OP

FORT MYERS
Merrick, Charles Gordon.....Leon bldg. ALR

JACKSONVILLE
Boyd, Charles Wilson.....111 W. Adams st. OP
Irwin, Thomas M.....Medical Arts bldg.—Park st. ALR
King, Raymond H.....3690 Herschel st. ALR
Knauer, William J.....Buckman bldg., Z2 OALR
McDaniel, Reuben L.....401 St. James bldg. OP
O'Dell, John C., Jr.....Medical Arts bldg., Z4 ALR
Richardson, Shaler.....111 W. Adams, Z2 OP
Taylor, H. Marshall.....111 W. Adams, Z2 ALR
Wilson, Alpheus K.....334 St. James bldg., Z2 ALR

LAKELAND
Hester, Marion William.....317 Marble Arcade bldg. OP

LAKE WORTH
Hughes, Ray W.....803 N. Lakeside Drive OALR
Selinger, Samuel.....230 Princeton Drive OALR

MERRITT ISLAND
Thomsen-von Colditz, G.....Dun Huntin OALR

MIAMI
Black, Nelson Miles.....Huntington bldg., Z32 OP
Brown, Andrew George.....717 DuPont bldg. OP
Chandler, G. E.....Huntington bldg., Z32 ALR
deBoe, M. P.....Huntington bldg., Z32 OALR
Dunaway, Carl E.....711 DuPont bldg. ALR
Hodsdon, B. F.....Box 923, Z6 OALR
Levin, Nathaniel M.....517 DuPont bldg. ALR
McKenzie, George Edward.....602 DuPont bldg. ALR
Mendel, James H.....Ingraham bldg., Z32 ALR
Palmer, Bascom H.....Huntington bldg., Z32 OALR
Park, M. B.....325 Ingraham bldg., Z32 OP
Repass, R. E.....924 N.E. 78th st. Z38 ALR
Richardson, John Roberts.....722 Dupont bldg. ALR
Weinstein, Philip.....Ingraham bldg., Z32 ALR
Whitmer, Kenneth Sweet.....711 duPont bldg., Z32 OP

## FLORIDA — Continued

MIAMI BEACH
- Denman, Francis E.....541 Lincoln road, Z39 ALR
- Edelman, Maurice I.....605 Lincoln rd. Z39 ALR
- Hollender, Abraham R.....605 Lincoln rd. ALR
- Hotchkiss, W. T.....541 Lincoln road, Z39 ALR

OCALA
- Russell, Ralph Edward.....1206 E. Ocklawaha ave. OP

ORLANDO
- Ingram, Hollis Carlyle.....303 Exchange bldg. OP
- Johnston, Hewitt.....303 Exchange bldg. ALR
- McLemore, Carl Sloan.....1202 S. Kuhl av. ALR
- Wall, Walton B.....Professional bldg. OP
- Wattles, Merrill.....1200 S. Kuhl ave. ALR

PALM BEACH
- Constantine, K. W.....229 Barton av. OP

PENSACOLA
- Davis, Oscar, Capt. (M.C.) USN, U. S. Naval Hospital OALR
- Heinberg, Charles J.....Medical Center ALR
- Leamer, Bruce V., Capt. (MC) USN
  School of Aviation Medicine, U. S. Naval Air Station ALR
- Lischkoff, M. A.....Blount bldg. OALR
- McLane, Jesse Newman.....1212 N. Palafax st. ALR
- Rubin, Nathan S.....404-407 Blount bldg. OP

ST AUGUSTINE
- Grace, Charles C.....145 King st. ALR

ST. PETERSBURG
- Heard, Mary K. (Retired).....804 4th Ave. N., Z4 OP
- McNay, Miller Ost.....472 Central av., Z5 ALR
- Nelson, Orville N.....1008 First Federal bldg. OALR
- Solomon, Henry D.....Power and Light bldg., Z5 ALR
- Winter, George E.....61 All States Court OALR

SARASOTA
- Goodell, William.....1066 S. Citrus ave. ALR

TAMPA
- Duke, R. R.....706 Franklin, Z2 ALR
- Edwards, Thomas Marvin.....322 Citizens bldg., Z2 ALR
- Forbes, Sherman B. .....706 Franklin, Z2 OP
- Lowry, B. W.....Citizens Bank bldg., Z2 OALR
- Taylor, Joseph W.....706 Franklin, Z2 OALR

WEST PALM BEACH
- Miller, Alice R.....505 Guaranty bldg. OALR
- Preefer, Raymond R.....303 Gardenia st. OP
- Sayad, William Y.....Harvey bldg. OALR
- Staton, Younger A.....715 Harvey bldg. OALR

WINTER HAVEN
- Odeneal, Thomas H.....Coker bldg. OALR

## GEORGIA

ATLANTA
- Baird, James Mason.....511 Medical Arts bldg. OP
- Brown, Lester A.....815 Doctors bldg. ALR
- Burgess, Taylor S.....310 Medical Arts bldg. Z3 ALR
- Burke, B. Russell.....478 Peachtree st., N.E., Z3 ALR
- Calhoun, F. Phinizy.....478 Peachtree st. N. E., Z3 OP
- Calhoun, Ferdinand Phinizy, Jr.....478 Peachtree St. OP
- Campbell, W. E., Jr.....Medical Arts bldg., Z3 OP
- Equen, M. S.....144 Ponce de Leon av. N. E. ALR
- Gershon, Nathan Isaac.....26 Linden Ave., N.E. ALR
- Hallum, Alton V.....478 Peachtree st. N. E., Z3 OP
- Howell, Stacy C.....478 Peachtree st. N. E., Z3 OP
- King, James Thomas.....803 Medical Arts bldg. ALR
- Lokey, H. M.....Medical Arts bldg., Z3 OALR
- Martin, William O., Jr.....478 Peachtree st. N.E., Z3 OP
- McDougall, J. Calhoun.....Medical Arts bldg., Z3 ALR
- McDougall, William L.....Doctors' bldg., Z3 ALR
- Stoner, Cyrus Hubert.....1029 Candler bldg. OP
- Warren, William C., Jr.....Doctors' bldg., Z3 ALR
- Wright, Edward S.....410 Medical Arts bldg. ALR

AUGUSTA
- Lewis, Samuel J.....S. F. C. bldg. ALR
- Royals, Walter C., Col. (MC)
  Chief, EENT Section, Oliver General Hospital OP

FT. BENNING
- Lippman, Otto, Capt. (M.C.).....Regional Hospital OP

MACON
- Smith, J. Allen.....Doctors' bldg. ALR

MOULTRIE
- Paulk, James R.....OP

ROME
- Smith, George B.....Harbin Hospital OALR

ST. SIMON'S ISLAND
- Brawner, Leon E.....Thelon "By the Sea" ALR

SAVANNAH
- Faggert, George H.....18 W. Oglethorpe av. ALR
- Lang, George Hermanne.....202 E. Liberty ALR

WAYCROSS
- Collins, Braswell E.....701 Elizabeth St. ALR

## IDAHO

BOISE
- Hedemark, Norman.....218 Eastman bldg. OALR
- Jones, Arthur C.....518 Eastman bldg. ALR
- Peterson, Roy L.....218 Eastman bldg. ALR

IDAHO FALLS
- Battles, Newell H.....P.O. Box 1180 OALR

NAMPA
- Shawhan, Glenn E.....Dewey Scales bldg. OALR

POCATELLO
- Clothier, Joseph.....Kane bldg. OALR
- Clothier, William L.....407-11 Kane bldg. OP
- Pond, Casper W.....Carlson bldg. ALR

TWIN FALLS
- Cutler, Morton.....122½ Main, South OP

## ILLINOIS

ALTON

Jones, F. W.....101 E. Broadway OALR
Middleton, Harry E.....206A W. 3rd OALR
Rawlins, Grove A.....206A W. 3rd ALR
Rubright, LeRoy W.....700 State St. ALR

AURORA

Darmer, George A.....Terminal bldg. OALR
Siewert, Otto Lucht.....33 Island Ave. OP

BELLEVILLE

Otrich, Grover C.....Commercial bldg. ALR
Pfeiffer, Guy Oliver.....19-A South High St. ALR
Roth, Leslie William.....203 Commercial bldg. OP
Schwartz, F. O.....230 E. Main OP

BLOOMINGTON

Elliott, J. Norman.....Griesheim bldg. ALR
Gailey, Watson W.....1008 N. Main OP
Watkins, Harold R.....Griesheim bldg. ALR

BLUE ISLAND

Field, Homer Baker.....12757 S. Western ave. OP

CARBONDALE

Brooks, Clyde M.....105 South University av. ALR

CENTRALIA

Hirschfelder, Max Kurt.....408 W. 2nd OP

CHAMPAIGN

Albers, Ed. C.....104 W. Clark OP
Ford, Hanby Lewis.....Robeson bldg. OALR
Youngerman, Wm. M.....Christie Clinic, 104 W. Clark St. ALR

CHICAGO

Ackerman, Walter Gabe.....104 S. Michigan av. Z3 OP
Allen, Thomas Dyer.....122 South Michigan av., Z3 OP
Andrews, Albert H., Jr.....700 N. Michigan av., Z11 ALR
Apple, Carl.....55 East Washington, Z2 OP
Barnett, Irving F.....30 North Michigan av., Z2 OALR
Bau, Hyman William.....3216 W. Roosevelt Rd. Z24 ALR
Becker, Oscar J.....30 N. Michigan av., Z2 ALR
Beem, Ione F.....30 North Michigan av., Sec. 2, Z2 OALR
Bellows, John G.....Rm. 1729, 30 No. Michigan Z2 OP
Berger, Louis.....30 N. Michigan ave. Z2 OP
Bernheimer, L. Benno.....104 S. Michigan av., Z3 ALR
Blonder, Edwin J.....25 E. Washington st., Z2 ALR
Blumklotz, Bruno.....25 E. Washington st., Z2 ALR
Boden, Worthey C.....6537 Kenwood Z37 ALR
Bothman, Louis.....30 N. Michigan av., Z2 OP
Brandenburg, Nora B.....700 N. Michigan av., Z11 ALR
Brawley, F. E.....30 N. Michigan av., Z2 OP
Bredlau, Ernest A.....2753 W. North, Z47 ALR
Brunner, Hans.....307 North Michigan av., Z1 ALR
Campbell, Paul A.....Suite 314, 700 N. Michigan av. ALR
Carelli, Paul Vincent.....3626 W. Chicago av., Z51 OP
Chainski, Edward L.....130 N. Wells, Z6 ALR
Christoph, Carl H.....30 N. Michigan av., Z2 OALR
Clark, James W.....2250 W. 91st st. Z20 OP
Cleff, Oscar.....55 E. Washington, Z2 OALR
Clement, C. C.....30 N. Michigan av., Z2 OALR
Cohen, Bernard M.....25 E. Washington st. Z2 ALR
Coombs, A. J.....30 N. Michigan av., Z2 ALR
Cottle, Maurice H.....30 N. Michigan av., Z2 ALR
Cowen, Jack P.....1421-122 S. Michigan ave. OP
Crage, Francis M.....25 E. Washington st., Z2 OP
Crane, Cyril Vincent.....6810 S. Loomis blvd. Z36 OP
Crossley, E. R.....4144 Sheridan rd., Z13 OALR
Cushman, Beulah.....25 E. Washington, Z2 OP
Cuter, Max Helix.....1010, 307 N. Michigan ALR
Davidson, H. P.....30 N. Michigan av., Z2 OP
Davis, Ralph Allison.....4753 Broadway OP
Delph, John F.....700 N. Michigan av., Z11 ALR
Dougherty, Clifford L.....55 E. Washington, Z2 ALR
Duboe, Sidney H.....4160 W. Madison Z24 ALR
Fabricant, Noah D.....185 N. Wabash av., Z1 ALR
Fields, Jack.....30 No. Michigan ave. Z1 OP
Findlay, E. K.....30 N. Michigan av., Z2 OP
Fitzgerald, James Robert.....3215 W. North Ave. Z47 OP
Fowler, Earle B.....55 E. Washington, Z2 OP
Fox, Nathan H.....4010 W. Madison OP
Fox, Noah.....841 E. 63rd, Z37 ALR
Friedberg, Stanton A.....122 S. Michigan Ave. Z3 ALR
Friedman, Elmer A.....6733 Newgard Ave. Z26 ALR
Gamble, Richard Cotter.....30 N. Michigan av., Z2 OP
Glatt, Morris A.....55 E. Washington, Z2 ALR
Goldenburg, Michael.....104 S. Michigan av., Z3 OP
Gordon, John Simpson.....950 E. 59th St., Z19 ALR
Green, Hyman S.....25 E. Washington st. OALR
Gustafson, Olga Frideberg.....185 N. Wabash av., Z1 OP
Guttman, M. Reese.....30 N. Michigan av., Z2 ALR
Hagens, Elmer William.....30 N. Michigan, Z2 ALR
Hall, Alice K.....25 E. Washington, Z2 ALR
Hayden, Jerome William.....4458 Madison st., Z24 ALR
Henrich, Mary Ingeborg.....6214 Winthrop ave. OP
Hipskind, Myron Maxwell.....6060 Drexel av., Z37 ALR
Hoeltgen, Maurice M.....1607 West 51st st., Z9 OP
Hoffman, L. G.....30 N. Michigan av., Z2 OALR
Hoffman, William A.....30 N. Michigan, Sec. 2., Z2 OP
Holinger, Paul H.....700 N. Michigan av., Z11 L
Holt, Helen.....720 North Michigan av., Z11 OP
Howard, William H.....9152 Commercial av., Z17 OP
Hurwitz, Paul
Suite 1532 Field Annex Bldg., 25 E. Washington st Z2 OP
Jacobs, Milton.....122 S. Michigan av., Z3 OP
Jenkins, Albert H.....2749 W. Arthur ave. Z45 ALR
Jones, William M.....5254 Michigan av. OP
Juers, Arthur L.....55 E. Washington st. Z2 ALR
Kammerling, Theodore S.....616 N. Central av., Z44 OALR
Kaufman, Samuel I.....185 N. Wabash av., Z1 OP
Kerst, John Arthur.....1444 Lake Shore dr. Z10 OP
Kerwin, Raymond W.....310 S. Michigan av., Z4 OALR
Klawans, Harold L.....20 E. Jackson blvd., Z4 ALR
Klien, Bertha A.....58 E. Washington, Sec. 2., Z2 OP
Kobrak, Heinrick..Univ. of Chicago, 950 E. 59th St. Z37 ALR
Koch, Harold J.....6235 S. Kedzie av., Z29 OP
Kraft, Oscar H.....1518 N. Dearborn Parkway, Z10 OP
Krause, Arlington C.....950 E. 59th, Z37 OP
Kronfeld, Peter C.....904 W. Adams, Z7 OP
Kulvin, Max M.....5110 Kenwood av., Z15 OALR
LaReau, Hector G.....4803 Lincoln av., Z25 ALR
Lazar, A. M.....30 N. Michigan av., Z2 ALR
Lazar, Nathan K. ..Room 2025 - 25 E. Washington, Z2 OP
Lebensohn, James E..6200 N. Kenmore av. Apt. 1022 Z40 OALR
Lederer, Francis L.....307 N. Michigan ave. ALR
Leech, Vernon M.....55 E. Washington, Z2 OP
Leshin, Norman.....55 E. Washington st., Z2 ALR
Lewy, Alfred.....25 E. Washington st., Z2 ALR
Lewy, Robert B.....25 E. Washington st., Z2 ALR
Lifschutz, Jacob.....30 N. Michigan av., Z2 OALR
Lindsay, John R.....950 E. 59th, Z37 ALR

## ILLINOIS — Continued

Livingston, George S.....185 N. Wabash av., Z1 ALR
Lyons, H. R.....1301 N. State St. Z10 ALR
Mann, William A.....30 N. Michigan av., Z2 OP
Matzkin, Lloyd L.....1525 E. 53rd St. Z15 ALR
McBean, George M.....25 E. Washington, Z2 OALR
McIntyre, George Fillmore.....25 E. Washington, Z2 ALR
McRoberts, Earl S. .....55 E. Washington, Z2 ALR
Merz, Earl H.....4319 N. Greenview, Z13 OP
Meyer, Samuel J.....58 E. Washington, Z2 OP
Moncreiff, William F.....58 E. Washington, Z2 OP
Morse, Leo M.....25 E. Washington St., Suite 1523 ALR
Morwitz, Samuel M.....55 E. Washington, Z2 ALR
Mundt, G. Henry.....30 N. Michigan av., Z2 OALR
Muskat, Irving.....25 E. Washington, Z2 OALR
Nance, Willis O.....6845 Euclid av., Z19 OALR
Nash, Herbert T.....31 N. State, Z2 ALR
Nethercut, Glenway W.....25 E. Washington, Z2 OP
Newell, Frank W.....2605 Pittsfield bldg., Z2 OP
Noonan, W. J.....4753 Broadway, Z40 OALR
Novak, Frank J., Jr.....30 N. Michigan av., Z2 ALR
Nugent, Oscar B.....231 W. Washington, Z6 OP
O'Connor, Philip D.....30 N. Michigan av., Z2 OP
O'Connor, Thomas P.....30 N. Michigan Z2 ALR
Orcutt, D. C.....55 E. Washington, Z2 OALR
Pearlman, S. J.....55 E. Washington, Z2 ALR
Pellettiere, Edmund V.....1530 N. Damen av., Z22 ALR
Perlman, Henry B.....950 E. 59th, Z37 ALR
Perritt, Richard A.....30 N. Michigan av., Z2 OP
Piszkiewicz, Frank J.....6019 N. Menard Ave. Z30 ALR
Pollack, Samuel.....2909 W. Estes av., Z45 ALR
Pollock, Frederick J.....104 S. Michigan ave. Z3 ALR
Prohovnik, Julius.....9 S. Kedzie av. ALR
Puntenney, Irving.....720 N. Michigan av., Z11 OP
Roling, Edward A.....2753 W. North av., Z47 OP
Roper, Kenneth L.....58 E. Washington, Z2 OP
Rosenberg, William..Rm. 1523, 25 E. Washington St. Z2OP
Ross, Percy J.....104 N. Michigan Ave. ALR
Ross, Perry W.....5426 N. Sheridan rd. OP
Salinger, Samuel.....25 E. Washington, Z2 ALR
Savitt, Louis.....3403 Lawrence av., Z25 ALR
Scheffler, Milton Morris.....55 E. Washington st. Z2 OP
Scheribel, Karl J.....55 E. Washington, Z2 OP
Schoolman, Joseph G.....104 S. Michigan, Z3 ALR
Schultz, Abraham.....30 N. Michigan ave. OP
Sciarretta, S. A.....55 E. Washington, Z2 ALR
Selinger, Elias.....104 S. Michigan av., Z3 OALR
Shambaugh, George E. Jr.....55 E. Washington St. Z2 ALR
Shanedling, Philip D., Lt. (M.C.), 6832 Paxton, Z49 OP
Shapira, Theodore M.....58 E. Washington, Z2 OP
Shapiro, S. L.....25 E. Washington st. ALR
Skinner, Merton Byron.....30 N. Michigan ave. ALR
Smiley, Wm. A.....104 S. Michigan Ave. Z3 ALR
Smith, Hiram J.....30 N. Michigan av., Z2 OP
Snitman, Maurice F.....55 E. Washington st., Z2 ALR
Soboroff, Simon H.....1608 Milwaukee av., Z47 ALR
Sowers, Alva Boyd.....6 N. Michigan av., Sec. 2, Z2 OP
Stagman, Joseph.....55 E. Washington St. ALR
Sternberg, Paul.....58 E. Washington, Z2 OP
Strauss, J. F.....104 S. Michigan av., Z3 ALR
Sullivan, Clifford P.,.....304 W. 63rd St., Z21 OP
Theobald, Walter H.....307 N. Michigan av., Z1 ALR
Tindal, John M.....5517 W. Fulton, Z44 OALR
Vail, Derrick.....Rm. 2605, 55 E. Washington st., Z2 OP
Van Alyea, O. E.....135 S. La Salle, Z3 ALR
Van Wien, Stefan.....104 S. Michigan blvd. OP
Vermeren, John C.....4753 Broadway, Z40 ALR
Von Leden, Hans Victor.....1046 Loyola Ave. ALR
Wegner, Edwin A., Jr.....1791 Howard St. ALR
Weiss, J. Allan.....58 E. Washington st., Z2 ALR
Wescott, Virgil.....30 N. Michigan av., Z2 OP
Whitsell, Fay M.....826 E. 61st st. OP
Wojniak, Frank.....4649 S. Ashland av., Z9 ALR
Zekman, Theodore N.....58 E. Washington st., Z2 OP

### CHICAGO HEIGHTS

Gorrell, T. J. H.....16th and Vincennes av. OALR

### DANVILLE

Baldwin, H. E.....139 N. Vermillion OALR
Baldwin, Stephen Glidden.....Adams bldg. ALR
Fink, Otto E.....9 W. Madison OP

### DECATUR

Hubble, William Fields.....Citizens bldg., Z30 ALR

### DIXON

McNichols, W. A.....101 W. First, Z3 OALR

### EAST ST. LOUIS

Hanson, William Ludwig.....Spivey bldg. ALR
Spitze, E. C.....Murphy bldg. OP

### ELGIN

Russell, Roland D.....Room 808 Professional bldg. ALR

### EVANSTON

Dodd, Oscar.....636 Church st. OP
Eubank-Jones, Miriam D.....944 Maple av. OP
Galloway, T. C.....636 Church st. ALR
Lawson, L. J.....636 Church st. ALR
McBride, Linn Frederick.....1501 Asbury av. ALR
McGarry, H. Isabelle.....636 Church st. OP
Snydacker, Daniel.....2626 Orrington Ave. OP
Soper, Gail Raney.....636 Church st. OP

### FREEPORT

Clark, J. Sheldon .....509 State Bank bldg. OALR

### GREAT LAKES

Wickstrom, Otto William, Capt. (MC) USN
U.S.N. Hosp. Quarters M. ALR
Young, Clifton Aurelius, Capt. (MC) USN
U. S. Naval Hosp. ALR

### HIGHLAND PARK

Henner, Robert, Capt. (M.C.).....2315 Sheridan rd. ALR

### HOMEWOOD

Dillon, Elmo E.....1952 Ridge road OALR

### JACKSONVILLE

Adams, A. L.....Professional bldg. OALR

### JOLIET

Lennon, Robert Watson.....58 N. Chicago st. OP
Woodruff, George H.....250 N. Ottawa ALR
Woodruff, H. W.....250 N. Ottawa OALR

## ILLINOIS — Continued

KANKAKEE
Gernon, William....542 S. Schuyler ave. OP
Judd, Delbert K....420 Arcade bldg. ALR
Kelly, Vincent J....310 Arcade bldg. OALR

LA GRANGE
Ehrmann, Evelina W....327 S. La Grange road OP

LA SALLE
Lenzen, Aloysius Francis....206 Marquette St. OP
Woods, R. H....153 Marquette OP

MACOMB
Duntley, G. S....604 Beverly ave. OALR

MATTOON
Jones, Florentine B....416 South 17th ALR
Voigt, C. B....1702 Broadway OALR

MAYWOOD
Spiesman, Irwin G....1908 St. Charles rd. ALR

MOLINE
Flatley, T. J....1518 Fifth av. OALR

NORMAL
Hartenbower, George E....1212 Broadway ALR

OAK PARK
Flack, Clarence Elbert....715 Lake St. OP
Good, Palmer W....715 Lake st. OALR
Palmer, Mahlon Phelps....715 Lake ALR
Theobald, Georgiana Dvorak....715 Lake OP
Theobald, John J....715 Lake ALR
Vorisek, Elmer A....1041 Lyman Ave. OP

OLNEY
Fisher, H. Noland....106 N. Silver st. OP

OTTAWA
Guibor, George P....303 Central Life bldg. OP

PARK RIDGE
Riser, Roy O....120 Main OP

PEKIN
Glasford, S. T....416 Elizabeth OALR

PEORIA
Blickenstaff, A. J....First Nat'l Bank bldg. Z23 OALR
DeLozier, Leonard Cecil....311 Central Nat'l Bank bldg. ALR
Howard, Henry Sargent....940 Jefferson bldg., Z2 ALR
McGrath, Philip R....842-845 Jefferson bldg. ALR
Owen, Walter E....1242 Jefferson bldg., Z2 ALR
Pannabecker, Charles L....434 Jefferson bldg., Z2 OP
Sneller, Charles D....320 Jefferson bldg. ALR

QUINCY
Gabriel, C. K....Illinois State Bank bldg. OALR
Henderson, James Alexander
The Quincy Clinic, 1416 Maine st. ALR
Mercer, Ray....811-13 W.C.U. bldg. OALR
Stevenson, Walter....W. C. U. bldg. OALR
Stevenson, Walter D., Jr....500 W.C.U. bldg. ALR

ROCKFORD
Blomberg, T. E....501 Seventh ALR
Fringer, Robert Chatterton....216 News Tower OP
Fringer, W. R....109 Sheridan st. OP
Potter, Jacob J....Gas Electric bldg. ALR
Pritikin, Roland I....1211 Talcott Bldg. OP
Voltz, Vernon Carl....303 N. Main St. OP

ROCK ISLAND
Herzog, Max....2815 8½ ave. OP
Ostrom, Louis....124 18th St. ALR
Schneck, Nathan....Rock Island Bank bldg. ALR

SKOKIE
Wallner, Linden J....4350 Bobolink Terr. ALR

SPRINGFIELD
Broadwell, Stuart, Jr....103½ N. 5th ALR
Deal, John Francis....522 E. Capitol av. OALR
Duncan, Perry E....405 E. Capitol av. OP

STERLING
Austin, M. Fletcher....206 Central Trust bldg. ALR
Brodrick, Frank W....302 First av. OALR
Sullivan, Timothy....104 15th av. ALR

STREATOR
Lester, Harry S....Murray bldg. OALR

URBANA
Baldwin, Gertrude Werder
Carle Hosp. Clinic, 602 W. University ave. OP
Cordray, David P.
Carle Hosp. Clinic, 602 W. University ave. ALR
Porter, George LeRoy....602 W. University ave. ALR
Thacker, Edgar A....602 W. University av. ALR

WAUKEGAN
Harrison, Glen H....307 Washington OP
Lennarson, Vincent A. E....4 S. Genesee st. ALR

WINNETKA
Ballenger, Howard C....723 Elm ALR

## INDIANA

ANDERSON
Aagesen, Walter J....615 Anderson Bank bldg. ALR
Larmore, Joseph L....712 Anderson Bank bldg. ALR

BLOOMINGTON
Geiger, Dillon....300 E. Kirkwood ALR
Smith, Herschel S....218 E. Kirkwood ave. OP

BLUFFTON
Smith, H. Brooks....303 S. Main OP

CRAWFORDSVILLE
Lingeman, B. N....Ben Hur bldg. ALR

EAST CHICAGO
Shulruff, Harry I....3701 Main St. OP

EVANSVILLE
Cockrum, William Marion....908 Hulman Bldg. OP
Leich, Charles F....124 S. E. First st. Z8 ALR
Ravdin, B. D....Hulman bldg., Z18 OALR
Ravdin, M....1666 Bayard Pk. dr., Z14 OALR
Slaughter, Howard Clarence....908 Hulman bldg. OP
Wesson, Thomas W....124 S. E. 1st, Z8 ALR

## INDIANA — Continued

FORT WAYNE

Bulson, Eugene Loring.....347 W. Berry, Z2 OALR
Estlick, Richard E.....629 Wayne Pharmacal bldg. Z2 ALR
Gessler, William F.....2902 Fairfield av., Z6 OALR
Mueller, Lawrence W.....3423 S. Washington Rd. ALR
O'Rourke, Carroll.....604 W. Berry st. ALR
Rothberg, Maurice.....712 Wayne Pharm. bldg. OP

GARY

Carmody, Raymond Francis.....504 Broadway OP
Chevigny, Julius James.....504 Broadway ALR
Parker, Harry Caunter.....673 Broadway ALR
Rubin, Milton R.....504 Broadway, Suite 725 ALR

HAMMOND

Kuhn, Hedwig S.....112 Rimbach OP
Kuhn, Hugh A.....112 Rimbach OALR
Lee, Otis S., Jr.....112 Rimbach st. OP
Lloyd, A. W.....8150 Forest Ave. OALR

HARTFORD CITY

Owsley, Guy A., Col. (M.C.).....214 N. High ALR

INDIANAPOLIS

Adams, D. S.....520 Hume-Mansur bldg., Z4 OALR
Alvis, Edmond Ochs.....320 Hume-Mansur bldg., Z4 OP
Aronson, Sidney S.....23 E. Ohio St. ALR
Berry, D. F.....Bankers Trust bldg., Z4 ALR
Blackford, Roger W.....503 Hume-Mansur bldg., Z4 ALR
Brown, David Edward.....520 Hume-Mansur bldg., Z4 ALR
Clark, Cecil P.....922 Hume-Mansur bldg., Z4 OP
Craft, Kenneth L.....1002 Hume-Mansur bldg., Z4 ALR
Cuthbert, Marvin Peare.....5611 N. Delaware st. OP
Dearmin, Robert M.....3440 N. Meridian St. ALR
Dyar, Edwin William.....5407 Kenwood av., Z8 OP
Ellis, Bert E.....303 Hume-Mansur bldg., Z4 ALR
Garrett, John D.....Bar Association bldg., Z4 ALR
Harding, Myron S.....308 Hume-Mansur bldg., Z4 OP
Harris, Carl B.....319 Hume-Mansur bldg., Z4 OP
Hughes, W. F.....401 Hume-Mansur bldg., Z4 OP
Hughes, William Franklin....401 Hume Mansur bldg., Z4 OP
Johnson, Thomas W.....529, 39 East Ohio, Z4 ALR
Jones, David E.....Chamber of Commerce, Z4 ALR
Kuntz, Herman William.....501 Hume Mansur bldg. ALR
Larkin, Bernard J.....305 Hume-Mansur, Z4 OP
Leasure, J. Kent.....611 Hume-Mansur bldg., Z4 ALR
Lingeman, Edward Laurel....411 Hume-Mansur bldg., Z4 ALR
Littell, J. Jerome .....603 Hume-Mansur bldg., Z4 ALR
Manion, Marlow W.....601 Hume-Mansur bldg., Z4 ALR
Mann, Mortimer.....323 Hume-Mansur bldg., Z4 OP
Masters, John M.....805 Hume Mansur bldg. Z4 OP
Masters, Robert J.....805 Hume-Mansur bldg., Z4 OP
McCaskey, C. H.....Guaranty bldg., Z4 ALR
McQuiston, Ralph J.....608 Guaranty bldg. ALR
Molt, William F.....Bankers Trust bldg., Z4 ALR
Norris, Mary Alice.....404 Hume-Mansur bldg., Z4 OP
Pfafflin, C. A.....Bankers Trust bldg., Z4 OALR
Row, D. Hamilton.....23 E. Ohio st., Z4 OP
Rutherford, C. W.....4601 N. Pennsylvania, Z5 OP
Sage, Russell A.....505 Hume-Mansur bldg., Z4 ALR
Sims, Lawrence.....3723 N. Gale, Z1 ALR
Sparks, Alan Leo.....1024 Hume-Mansur bldg. Z4 ALR
Sputh, Carl B.....224 N. Meridian st., No. 301, Z4 ALR
Sputh, Carl Brosius.....224 N. Meridian, Z4 ALR
Sudranski, Herbert F.....23 E. Ohio st., Z4 OP
Swan, John R.....915 Hume-Mansur bldg., Z4 ALR
Teixler, Victor Adolph.....224 Hume-Mansur bldg., Z4 OP
Van Osdol, Harry A.....Chamber of Commerce bldg., Z4 ALR
Whitaker, Joel.....807 Hume-Mansur bldg., Z4 OALR
Wright, J. William.....301 Hume-Mansur bldg., Z4 ALR

KOKOMO

Adams, Charles Joseph.....Armstrong-Landon bldg. OALR
Good, Richard P.....308 Armstrong-Landon bldg. ALR

LAFAYETTE

Calvert, Raymond R.....314 N. 6th st. OP
Peterson, Joel A.....609 Lafayette Life bldg. ALR
Van Buskirk, Edmund Linford .....308-312 N. 8th OP

LOGANSPORT

Adamski, Michael S.....408 North st. OP
Hickman, Warren R.....211 Third ALR
Holmes, Will W.....R.F.D. 4 ALR

MARION

Braunlin, William H.....Marion National Bank bldg. OALR

MICHIGAN CITY

Krieger, G. M.....701 Washington OALR

MUNCIE

Deutsch, William, Jr.....309 Johnson bldg. ALR
Hill, Howard E.....402 W. Jackson st., POB 774 ALR

NEW CASTLE

Smith, Robert Arvil.....106 N. Main st. ALR

RICHMOND

Holland, Emory E.....1907 E. Main st. OP

SOUTH BEND

Cassady, J. Vernal.....Sherland bldg., Z9 OALR
Clark, William H.....120 N. Lafayette blvd. ALR
Dietl, Ernest L.....527 Sherland bldg. ALR
McFarland, Corley B.....122 N. Lafayette blvd. OP
Rigley, Edward L.....108 Sherland bldg., Z9 ALR
Rudolph, Carl J.....2016 E. Madison st. OP
Savery, C. E.....Sherland bldg., Z9 OALR

TERRE HAUTE

Allen, Orris T.....Rose Dispensary bldg. OALR
Gillum, J. R.....221 S. 6th OALR
Mahoney, Charles L.....1438 S. Center st. ALR
McBride, Noel S.....12 S. 7th OP
McEwen, James Woods.....670 Cherry St. OP
Stewart, Walter E.....Tribune bldg. OALR

TIPTON

Overman, Frederick V.....222 W. Jefferson ALR

UNION CITY

Ruby, F. McK.....235 W. Pearl OALR

## IOWA

AMES

Thorburn, O. L.....Box 451 ALR

BLOOMFIELD

Gilfillan, Homer J.....Gilfillan Clinic ALR

## IOWA — Continued

BURLINGTON
La Force, E. F. ..... Iowa State Bank bldg. OALR
Lee, Wayne Robert ..... 613 Medical Arts bldg. ALR
Pearson, George J. ..... 320 N. 3rd ALR

CARROLL
Anneberg, Adrian R. ..... 1332 N. Carroll st. ALR
McConkie, Willis L. ..... ALR

CEDAR FALLS
Huston, Marshall D. ..... 302½ Main st. ALR

CEDAR RAPIDS
Foster, Wayne J. ..... Higley bldg. ALR
Noe, Carl A. ..... 601 Higley bldg. OALR
von Lackum, John Kenneth ... Merchants Nat'l Bank bldg. ALR

CLINTON
Carey, Edward Thomas ..... 415 Howes bldg. ALR
Foster, Warren H. ..... 217 Fifth av. S. ALR
Weih, E. P. ..... 217 Fifth av. S. OALR

COUNCIL BLUFFS
Dean, F. W. ..... 22 S. Sixth OALR
Howard, Lloyd G. ..... Park bldg. OALR
Treynor, Jack Vernon ..... 532 First Ave. ALR

CRESTON
Childs, H. A. ..... 1016 W. Adams OALR

DAVENPORT
Hands, Sidney G. ..... 201 W. 2nd ALR
Harkness, Gordon F. ..... Putnam bldg. OALR
Hofmann, W. P. ..... Kahl bldg. OALR
Lamb, Harry Howe ..... 220 Main ALR
Rock, John Emmett ..... Putnam bldg. ALR
Shurtleff, Raymond S. ..... 2224 Harrison st. ALR

DES MOINES
Downing, Arthur H. ..... 406 6th Ave. OP
Downing, James A. ..... Bankers Trust bldg., Z9 ALR
Gurau, Henry H. ..... 215 Bankers Trust bldg. OP
Jones, Cecil C. ..... Equitable bldg., Z9 OALR
Kirch, Walter ..... 1023 Bankers Trust bldg., Z9 ALR
Kos, Clair M. ..... 534 29th st. ALR
Lynch, Robert J. ..... Equitable bldg., Z9 ALR
Matheson, John Hollis ..... 802 Equitable bldg., Z9 OP
Merkel, Byron M. ..... 112 Equitable bldg., Z9 ALR

DUBUQUE
Connelly, Edgar ..... 7 Roshek bldg. ALR
Langworthy, Henry G. ..... 394 W. 10th st ALR
Pickard, John C. ..... 1200 Main st ALR
Quinn, Francis P. ..... Roshek bldg. ALR
Steffens, Lincoln F. ..... 1200 Main st. OP
Thompson, Howard E. ..... 1100 Main OALR
Thorson, John A. ..... Roshek bldg. OP

FORT DODGE
Chase, S. B. ..... Carver bldg. OALR
Kluever, Herman C. ..... 221 N. 18th St. OP

FORT MADISON
Reimers, R. S. ..... 613 Eighth ALR

IOWA CITY
Albright, George C. ..... 104 S. Clinton ALR
Allen, James Harrill ..... University Hospitals OP
Boiler, William F., Maj. (M.C.), .... 206 Dey bldg. OALR
Huffman, William Clyde ..... University Hospitals ALR
Leinfelder, Placidus J. ..... University Hospital OP
Lierle, Dean M. ..... University Hospital ALR-PI S
O'Brien, C. S. ..... University Hospital OP
Reger, Scott N. ..... University Hospitals
Tyler, William H. ..... University of Iowa Hosp. ALR

LE MARS
Joynt, M. J. ..... Royal bldg. ALR

MARSHALLTOWN
French, R. F. ..... Masonic Temple OALR
Wolfe, Otis R. ..... Masonic Temple OP

MASON CITY
Chenoweth, Charlie Everette ..... Forester's bldg. ALR
Fallows, Howard D. ..... Forester's bldg. OALR
Flickinger, Roger R. ..... Park Hospital Clinic. ALR
O'Brien, Steve A. ..... First Nat'l Bank bldg. OALR

MUSCATINE
Goad, R. R. ..... 110 W. 2nd St. OP

OSKALOOSA
Greenlee, Max R. ..... Mahaska Theatre bldg. ALR

OELWEIN
Howard, W. H. ..... OALR

OTTUMWA
Prewitt, Leland H. ..... 207 E. 2nd St. ALR

SHENANDOAH
Maloy, Wayland H. ..... 608½ W. Sheridan av. OP

SIOUX CITY
Decker, J. C. ..... Frances bldg., Z8 OALR
Dvorak, Joseph E. ..... Davidson bldg., Z13 OP
Gittins, T. R. ..... Davidson bldg., Z13 OALR
Reeder, J. E. ..... Davidson bldg., Z13 OALR
Reeder, James E., Jr. ..... 523 Davidson bldg. Z13 OP
Tripp, LeRoy R. ..... 909 Badgerow bldg. OALR

WATERLOO
Bender, H. A. ..... Black bldg. ALR
Joynt, A. J. ..... 525 Sycamore OALR
Reuling, F. H. ..... Black bldg. OALR
Russell, R. E. ..... 531 Commercial OALR

## KANSAS

ATCHISON
Bribach, Eugene J. ..... Professional bldg. OALR

COFFEYVILLE
Chadwick, Ira B. ..... 717½ Walnut OALR

CONCORDIA
Robertson, Edward N. ..... New Caldwell bldg. OALR

EMPORIA
Granger, W. B. ..... Gazette bldg. OALR

## KANSAS — Continued

**HUTCHINSON**
- Greever, Boyd L. ..... 100 N. Main OALR
- Quiring, Walter O. ..... 100 First West ALR
- Scales, Wm. McVey ..... 100 N. Main OP

**KANSAS CITY**
- Billingsley, John A. ..... Huron bldg., Z10 OP
- Mullen, Clifford J. ..... 424 Brotherhood bldg. Z11 OP
- Ryan, Maurice J. ..... 2016 Elizabeth Ave., Z2 ALR
- Ryan, Michael J. ..... 310 Huron Bldg. Z10 ALR
- Spake, LaVerne B. ..... Brotherhood bldg., Z11 ALR

**LAWRENCE**
- Powell, Lyle S. ..... 736 Kentucky St. OALR

**NEWTON**
- Morgan, Harold E. ..... 314 E. 3rd st OP

**PARSONS**
- Blasdel, Tyler David ..... 110½ S. 18th ALR

**PITTSBURG**
- Haas, Louis Richard ..... Smith Clinic OALR
- Stelle, Harry L. ..... 207 Commerce bldg. OALR

**PRATT**
- Pitman, Will D. ..... First Nat'l Bank bldg. ALR

**SALINA**
- Cheney, Ralph E. ..... United bldg. OP
- Ogus, Alfred, Capt. (M.C.), ..... Army Air Base ALR

**TOPEKA**
- Ashley, Byron J. ..... 2315 W. 13th st. OALR
- Boggs, Frank C. ..... Mills bldg. OP
- Kirkpatrick, Hazen L. ..... 715 Mills bldg. ALR
- Powers, Harold W. ..... 705 National Reserve Bldg. ALR
- Reed, William W. ..... Mills bldg. ALR

**WICHITA**
- Brownell, Morton E. ..... 106 W. Douglas, Z2 OP
- Carter, E. D. ..... Central bldg., Z2 ALR
- Cheney, James W. ..... Schweiter bldg., Z2 OALR
- Francis, Norton L. ..... 4325 E. Lewis, Z8 ALR
- Gillett, Wilbur G. ..... 201½ N. Main, Z2 OP
- Gsell, George F. ..... 911 Beacon bldg., Z2 OP
- Gsell, J. F. ..... Beacon bldg., Z2 OALR
- Maggard, D. I. ..... Orpheum bldg., Z2 OALR
- Marshall, Hal E. ..... 22 Colonial Court, Eastborough ALR
- Reifsneider, J. Stanley ..... Beacon bldg., Z2 ALR
- Seydell, E. M. ..... First Nat'l Bank bldg., Z2 ALR
- Tippin, Ernest E. ..... First Nat'l Bank bldg., Z2 ALR
- Weaver, T. W. ..... 201 N. Main, Z2 OP

**WINFIELD**
- Ralls, C. T. ..... State Bank bldg. OALR

## KENTUCKY

**COVINGTON**
- Bledsoe, R. W. ..... First Nat'l Bank bldg. OALR

**FRANKFORT**
- Snyder, William S., Jr. ..... 324 W. Main ALR

**GLASGOW**
- Weldon, William A. ..... OP

**HARLAN**
- Gunn, Milus L. ..... 107 E. Mound st. ALR

**HENDERSON**
- Rodenberg, Elmer Julius ..... 319 N. Elm St. ALR

**HOPKINSVILLE**
- Harned, John W. ..... 902½ S. Main st. ALR

**LEXINGTON**
- DeWeese, Clarence ..... 201 W. Second st., Z15 OALR
- Frey, Walter D. ..... 200 N. Upper st., Z15 OP
- Marks, Samuel Blackburn ..... 203 W. Second, Z15 ALR
- Stern, Milton J. ..... First National Bank bldg., Z3 OP
- Thompson, Wm. R. ..... 203 W. 2nd st., Z15 ALR

**LOUISVILLE**
- Baker, Melvin C. ..... Heyburn bldg., Z2 OP
- Bass, A. L. ..... Heyburn bldg., Z2 OALR
- Bloch, Charles L. ..... 2905 Dundee rd. OP
- Bornstein, Max ..... 308 Will Sales bldg. ALR
- Dean, Walter ..... 300 Francis bldg., Z2 OALR
- Dulaney, Octavus ..... Brown bldg., Z2 ALR
- Forrester, Alex M. ..... 922 Heyburn bldg. ALR
- Heitger, J. D. ..... Heyburn bldg., Z2 OALR
- Hutcherson, John Kenneth ..... Francis bldg., Z2 OP
- Leggett, Albert E. ..... 614 Fincastle bldg., Z2 OP
- Moran, Charles T. ..... 1014 Heyburn bldg. OP
- Pfingst, Harry A. ..... 332 W. Broadway, Z2 OP
- Preston, William O. ..... 733 S. 44th St. ALR
- Pryor, Will R. ..... 415 Heyburn bldg. Z2 OP
- Ray, George H. ..... 914 Francis bldg. OP
- Ray, Joseph Carr ..... 914 Francis bldg. ALR
- Townes, C. Dwight ..... 1014 Heyburn bldg. OP
- Victor, Karl N. 1010 Brown bldg., 321 West Broadway, Z2 ALR
- Wolfe, William C. ..... 1012 Heyburn bldg. ALR

**MADISONVILLE**
- Garnier, William Hampton ..... Doctors bldg. ALR
- Salmon, David L., Major (M.C.) ..... Noel Ave. ALR

**OWENSBORO**
- Woolfolk, William L. ..... 115 E. 4th st. ALR

**PADUCAH**
- Abell, Harry Davis ..... 333 Broadway OP
- Pace, Errett ..... City Savings Bank bldg. ALR
- Reynolds, H. G. ..... 717 Citizens Savings Bank bldg. OALR

## LOUISIANA

**ALEXANDRIA**
- Simmonds, Noel T. ..... 1404 Murray OP

**BATON ROUGE**
- Marks, Paul L. ..... 131 No. 8th St. OP
- McLaurin, James Walter ..... 251 3rd, Z6 ALR
- Phillips, James R. ..... 701 Winn av. OP

**LAKE CHARLES**
- Moss, Olin W. ..... Weber bldg. ALR
- Raggio, Francis Warren, Jr. ..... 2416 Helen st. ALR

**NEW ORLEANS**
- Alexander, Lucian W. ..... Maison Blanche bldg., Z16 ALR
- Bahn, Charles A. ..... 1028 Maison Blanche bldg., Z16 OP
- Beil, Wallace C. ..... 912 American Bank bldg. OP

## LOUISIANA — Continued

Boles, Wm. M. ..... 4132 St. Charles Ave. Z15 OP
Brewster, H. F. ..... 913 Richards bldg. ALR
Brown, Charles Lafayette ..... 713-15 Richards bldg. ALR
Buffington, Wiley R. ..... Hibernia Bank bldg., Z12 OP
Clark, William B. ..... American Bank bldg., Z12 OP
Cox, Charles L. ..... Pere Marquette bldg., Z12 ALR
Crebbin, Alexander R. ..... Maison Blanche bldg., Z16 OP
Farrior, J. Brown ..... Ochsner Clinic ALR
Fuchs, Valentine Henry ..... American Bank bldg., Z12 ALR
Gaines, Shelley Rice ..... American Bank bldg., Z12 OP
Gooch, John B. ..... Canal Bank bldg., Z12 ALR
Granberry, Carl ..... Maison Blanche bldg., Z16 ALR
Haik, George M. ..... 812 Maison Blanche bldg. OP
Hallaron John J. ..... 3405 St. Charles av. ALR
Hanley, Jeanne C. Roeling ..... Maison Blanche bldg., Z16 ALR
Irwin, John Joseph ..... Baronne bldg., Z12 ALR
Kearney, Harold Leslie ..... 1403 Delachaise, Z15 ALR
Kimata, Harold T. ..... 165 Elk Place, Z13 ALR
Larose, J. Henry ..... 1218 Richards bldg., Z12 OP
LeJeune, F. E. ..... Ochsner Clinic, Z15 ALR
Lynch, Mercer G. ..... Ochsner Clinic, ALR
McComiskey, Arthur James ..... 3420 Prytania, Z15 ALR
Meyer, Monte F. ..... 645 Canal bldg. ALR
Smith, Leslie M., Sr. Surgeon USPHS
U. S. Marine Hospital Z15 OP
Smith, Victor C. ..... American Bank bldg., Z12 OP
Steele, Clarence Hart ..... Ochsner Clinic ALR
Taquino, George J. ..... Canal bldg., Z12 ALR
Wagner, W. A. ..... 200 Carondelet, Z12 ALR
Walls, Edward G. ..... 603 Maison Blanche bldg., Z16 ALR
Weil, A. I. ..... 3439 Prytania, Z15 ALR
Womack, David R. ..... 1218 Maison Blanche bldg., Z 16 ALR
Wood, Spencer ..... 500 Verret, Z15 ALR

PINEVILLE
Ewing, Fayette C. ..... (Retired), "Nosegay,"

SHREVEPORT
Bryant, Frank L. ..... 2622 Greenwood ALR
LaRue, C. L. ..... Highland Clinic, Z50 OALR
Riggs, Ralph H. ..... Medical Arts bldg. ALR
Scales, J. L. ..... Medical Arts bldg., Z45 OALR
Wilkinson, Joel A. ..... P.O. Box 1526, Z93 OALR

## MAINE

AUGUSTA
Provost, Pierre E. ..... 48 Green St. ALR

BANGOR
Butler, Harry ..... 77 Broadway ALR
McQuoid, Robert Moffat ..... 39 Columbia ALR
Whitworth, John Ewing ..... 116 Hammond ALR

BATH
Kershner, W. E. ..... 117 Front OALR

GARDINER
Hurd, Allan C. ..... 62 Church St. ALR

HOULTON
Hogan, Chester F. ..... 62 Main st. ALR

LEWISTON
Chaffers, William H. ..... 190 Bates ALR

PORTLAND
Beach, S. Judd ..... 704 Congress, Z4 OP
Cummings, George O. ..... 47 Deering, Z4 ALR
Gordon, Charles H. ..... 46 Deering, Z4 ALR
Haskell, Alfred W. ..... 142 High, Z3 OP
Johnson, Henry P. ..... 32 Deering, Z3 ALR
McAdams, R. ..... 723 Congress, Z1 OP

WATERVILLE
Flanders, Merton ..... Professional bldg. ALR
Hill, Frederick T. ..... Professional bldg. ALR
Hill, Howard F. ..... 177 Main OP
Poulin, James E. ..... Professional bldg. ALR

## MARYLAND

ANNAPOLIS
Welch, Robert S. G. ..... 86 State Circle OALR
Wiser, Henry J., Capt. (M.C.) .... 503 Dream's Landing ALR

BALTIMORE
Bagley, Cecil Hopkins ..... 2 East Read, Z2 OP
Bodenheimer, Ernst ..... 1212 Eutaw Place, Z17 OP
Bordley, James Jr. ..... 2630 Guilford av., Z18 OALR
Breitstein, M. Lewis ..... 1213 Eutaw Place, Z17 ALR
Clapp, C. A. ..... 513 N. Charles st., Z1 OP
Crowe, Samuel James ..... Johns Hopkins Hospital ALR
Dix, Harold C. ..... 206 Paddington rd., Z12 OP
Fox, Samuel L. ..... 1205 St. Paul st., Z2 ALR
Friedenwald, Harry ..... 1212 Eutaw pl., Z17 OP A
Friedenwald, J. S. ..... 1212 Eutaw pl., Z17 OP
Goldback, L. J. ..... 6 East Eager, Z2 OP
Goldberg, Herman K. ..... 719 N. Charles st. OP
Guyton, Jack Smallwood ..... John Hopkins Hospital, Z5 OP
Hantman, Irvin, Capt. (M.C.)
c/o Greenberg, 3647 Cottage av. ALR
Kemler, Joseph I. ..... 1908 Eutaw pl., Z17 OALR
Kleimen, Bernard S., Capt. (MC) .. 2517 Linden ave., Z17 ALR
Kremen, Abraham ..... 2355 Eutaw Place, Z17 OP
Lieberman, Alfred T. ..... 29 E. Mt. Vernon Pl., Z2 ALR
Looper, Edward Anderson ..... 104 W. Madison, Z1 OALR
MacLean, Angus L. ..... 1201 N. Calvert, Z12 OP
Maumenee, Alfred E. ..... Johns Hopkins Hospital, Z5 OP
McKenzie, William Raymond ..... 101 W. Read, Z1 ALR
Owens, Ella M. Uhler ..... Johns Hopkins Hospital, Z5 OP
Owens, William Councilman .. Johns Hopkins Hospital, Z5 OP
Patterson, Robert U ..... 5801 Roland Ave., Z10 ALR
Proctor, Donald F. ..... c/o Johns Hopkins Hospital, Z5 ALR
Randolph, M. Elliott ..... 11 E. Chase st., Z2 OP
Raskin, Moses ..... The Madison, 817 St. Paul st., Z2 OALR
Reese, Frederic Miller .... Prof. bldg., 330 N. Charles, Z1 OP
Scholz, Roy O. ..... 1515 Pentridge rd., Z12 OP
Small, Mary L. ..... 18 W. Read, Z1 OP
Snyder, Jerome ..... 11 E. Chase St. ALR
Tarun, William ..... 2309 Kenoak rd., Z9 OP
Walsh, Frank B. ..... Johns Hopkins Hosp., Z5 OP
Woods, Alan C. ..... Johns Hopkins Hospital, Z5 OP
Zinn, Waitman Farnsworth ..... Medical Arts bldg., Z1 ALR

BETHESDA
Delaney, Adrian J., Capt. (M.C.), USN,
Nat'l Naval Medical Center ALR
Stone, Leslie O., Capt. (MC) USN .. U.S. Naval Hospital, ALR

CUMBERLAND
Jones, Emmett L., Jr. ..... 15 S. Liberty st. ALR

## MARYLAND—Continued

CHEVY CHASE
Rice, Carl E.....6500 Maple av., Z15 OP

FREDERICK
Brandfield, Ira Irvin.....305 W. 2nd St. ALR

HAGERSTOWN
Smith, W. Hamilton.....314 Wareham bldg. ALR
Smoot, Merrill C.....130 W. Washington st. ALR

SALISBURY
Gillis, Marion H. J.....204 Walnut st. OP

WESTERN PORT
Reeves, J. Norman.....121 Main st. ALR

## MASSACHUSETTS

ARLINGTON
Carroll, Walter J. E.....5 Chestnut, Z4 ALR

BELMONT
Mosher, Henry Adams.....11 Beatrice Circle, Z78 OP

BEVERLY
Senecal, Alphonse L.....1 Monument Square ALR

BOSTON
Allman, Charles H.....520 Commonwealth av. Z15 ALR
Beetham, William P.....108 Bay State Rd. OP
Burian, Hermann M.....520 Commonwealth av. Z15 OP
Carter, Albert A.....419 Boylston, Z16 OP
Casten, Virgil.....412 Beacon st. OP
Chandler, Paul A.....5 Bay State rd., Z15 OP
Chenery, W. E.....377 Commonwealth av., Z15 ALR
Chisholm, Julian F., Jr., 20 W. Cedar, c/o Mrs. Bunting, Z8 OP
Cline, Samuel.....416 Marlboro, Z15 ALR
Cogan, David G.....243 Charles st. OP
Cowan, Clyde R.....69 Bay State rd., Back Bay, Z15 ALR
DeBlois, Elizabeth.....247 Commonwealth av., Z15 ALR
Delman, A. H.....U.S. Vet. Hosp., W. Roxbury ALR
Donahue, Hugh C.....520 Commonwealth av., Z15 OP
Drooker, Joshua C.....20 Charlesgate West ALR
Dunphy, Edwin B.....243 Charles st. OP
Easton, Mahlon T.....264 Beacon st., Z16 OP
Evans, Maurice George.....416 Marlboro, Z15 ALR
Fein, Harry.....483 Beacon st., Z15 ALR
Fineberg, Nathan L.....475 Commonwealth av., Z15 ALR
Frazee, John R.....311 Beacon st. ALR
Fred, Gustave B.....520 Beacon, Z15 ALR
Freedman, L. M.....475 Commonwealth av., Z15 ALR
Garland, Frederick E.....483 Beacon, Z15 LR
Gettes, Charles N.....481 Beacon st., Z15 ALR
Goodale, Robert L.....330 Dartmouth, Z16 ALR
Goodall, Edwin Baker.....71 Bay State rd., Z15 OP
Gorney, Arthur J.....14 Charlesgate West ALR
Gundersen, Trygve.....101 Bay State rd. OP
Haley, William Thomas.....531 Beacon, Z15 ALR
Hickey, Cornelius S.....39 Bay State rd., Z15 ALR
Holmes, Edgar M.....330 Dartmouth, Z16 ALR
Hoover, Walter Boyd.....605 Commonwealth av., Z15 ALR
Inglis, Harry J.....43 Bay State rd., Z15 ALR
Johns, Juanita Pearl.....313 Commonwealth av., Z15 OP
Johnson, Carl Cordes.....5 Bay State rd., Z15 OP
Johnson, Charles I.....403 Commonwealth av. ALR
Johnson, Leighton F.....29 Bay State rd., Z15 ALR
Kaufman, Aaron.....483 Beacon St., Z7 ALR
Kazanjian, V. H.....475 Commonwealth av., Z15 LR-PI S
Kelley, Vincent J.....520 Commonwealth av., Z15 ALR
Kleinert, Margaret N.....23 Bay State rd., Z15 ALR
Lancaster, W. B.....520 Commonwealth av., Z15 OP
Lathrop, Frank D...Lahey Clinic, 605 Commonwealth Ave. ALR
Lentine, Joseph.....395 Commonwealth Ave. ALR
Lewis, James Prince.....412 Beacon st. ALR
Lovesey, Burton E.....76 Bay State rd., Z15 ALR
Lurie, Moses H.....483 Beacon, Z15 ALR
Meltzer, Philip E.....20 Charlesgate West, Z15 ALR
Messenger, Harry K.....313 Commonwealth av., Z15 OP
Miller, Daniel.....20 Charlesgate West, Z15 ALR
Mills, Chester R.....520 Commonwealth av., Z15 ALR
Mueller, Werner.....412 Beacon, Z15 ALR
Offenbach, Bertha.....270 Commonwealth Ave. OP
Poirier, George H.....60 Bay State rd., Z15 ALR
Quincy, J. E.....270 Commonwealth av., Z16 ALR
Regan, James J.....520 Commonwealth Ave., Z15 OP
Regan, William F.....19 Bay State rd., Z15 ALR
Richardson, John R.....403 Commonwealth av., Z15 ALR
Riseman, Benjamin.....41 Bay State rd., Z15 ALR
Rowland, W. D.....180 Commonwealth av., Z16 OP
Sachs, Benjamin.....520 Beacon, Z15 OP
Schall, LeRoy A.....243 Charles st., Z14 ALR
Skirball, J. J.....353 Commonwealth av., Z15 OP
Sloane, Albert E.....416 Marlborough, Z15 OP
Thompson, Peter H.....308 Commonwealth av., Z15 OP
Thorne, Fred S.....270 Commonwealth av., Z16 OP
Tingley, Louisa Paine.....416 Marlborough, Z15 OP
Tobey, George L., Jr.....403 Commonwealth av., Z15 ALR
Wells, David W.....31 Bay State rd., Z15 OP
Wilkins, Samuel Henry....270 Commonwealth av., Z16 OP
Zonderman, Bernard.....29 Bay State Rd. ALR

BRIGHTON
Cammisa, James J. Vincent, Comdr. (M.C.), USN, 44 Leamington rd., Z35 OALR

BROCKTON
Nerbonne, Joseph J.....231 Main, Z1 OALR
Welch, Robert Francis.....232 Main St. ALR

BROOKLINE
Alpert, David R., Maj. (MC).....28 Verndale st. OP
Richards, Lyman G.....1101 Beacon st. ALR
Verhoeff, Frederick H.....61 Monmouth st. OP

CAMBRIDGE
Butler, Edmund John.....25 Garden st., Z38 ALR
Hewitt, Wright P.....51 Brattle st., Z38 ALR

CLINTON
Monahan, J. A.....181 Chestnut OALR
O'Toole, Francis Austin.....181 Chestnut ALR

FALL RIVER
Donovan, Richard J.....422 N. Main OALR
Ryder, D. R.....151 Rock ALR

FITCHBURG
Quinlan, Joseph D.....44 Prichard st. ALR

FRAMINGHAM
Dinan, Thomas Edward.....132 Union av. ALR
Salls, Frederick H. .....132 Union av. OP
Tomb, Everett H.....46 Lexington ALR

## MASSACHUSETTS — Continued

GARDNER
Bone, Herman D.....19 Pleasant OALR
Powers, John T. H., Major (MC).....32 Pleasant st. ALR

GLOUCESTER
Lodge, Edmund Anderson.....79½ Prospect St. ALR

GREENFIELD
Dame, L. R.....78 Federal st. OP
Millett, Frank A.....40 High OALR

HAVERHILL
Birdsall, Clarence H.....30 Sumner ALR
Capeles, T. F.....191 Merrimack OALR
Kapp, Henry.....50 Merrimack ALR
Shlossberg, Frank Ronald.....116 Emerson ALR
Trull, Alfred C.....3 Washington square OP

HOLYOKE
Abbot, Edward A.....225 High st. ALR
Burke, John R.....207 Elm OALR
Halton, Edward P.....1456 Northampton st. ALR

LAWRENCE
Byrne, Harry V.....301 Essex OP
Colson, Z. William.....301 Essex st. OP

LOWELL
Berman, Philip G.....174 Central OP
Leahey, Brendan D.....9 Central OP
Leahey, George A.....45 Merrimack OP

LYNN
McIntire, Frederic J.....63 N. Common ALR
O'Reilly, W. F.....44 S. Common OALR
Rafferty, George E.....173 N. Common ALR

MALDEN
French, Robert Weller.....181 Pleasant OP
Haire, Paul G.....6 Pleasant, Z48 OP

MARBLEHEAD
Mosher, Harris P.....127 Front ALR

MELROSE
DeWolf, Charles W.....8 Porter ALR

NORTHAMPTON
Collins, J. D.....187 Main ALR
Dow, Frank E.....4 West OP

PITTSFIELD
Porter, Byron H.....18 Bank Row ALR
Thompson, G. H.....74 North OALR

PLYMOUTH
Swenson, Rudolph E.....1 Carver St. ALR

QUINCY
Gilmore, Robert O.....1159 Hancock ALR
McClintock, Walter L., Capt. (M.C.), 120 Putnam st. ALR

SALEM
Adams, John G.....24 Winter ALR
Hennessey, W. W.....333 Essex OALR
Tolman, Henry.....136 Federal ALR

SOMERVILLE
Solo, Adrian.....174 Highland av. ALR

SOUTH HARWICK
Haley, Paul J. D..... ALR

SPRINGFIELD
Boss, Eugene George.....1537 Main, Z3 OALR
Byrnes, Harry F.....67 Chestnut, Z5 OALR
Chamberlin, Ira Iowa.....4 Chestnut St. ALR
Dineen, John B.....1537 Main st. ALR
Donoghue, William Francis, Jr.....69 Chestnut St. ALR
Gaudreau, Honore E.....293 Bridge, Z3 OP
Irwin, Vincent J., Jr.....1634 Main, Z3 OALR
Segal, Samuel, Jr.....111 Maple St., Z5 ALR

WAKEFIELD
Jones, Elgin W.....29 Lafayette St. OP A

WALTHAM
Mackenzie, Roland C.....30 Grant, Z54 OP

W. SOMERVILLE
Jellis, Walter.....71 College av. ALR

WORCESTER
Berry, Gordon.....36 Pleasant, Z8 ALR
Caron, Armand L.....36 Pleasant, Z8 ALR
Croissant, Charles A.....390 Main, Z8 ALR
Cross, A. E.....390 Main, Z8 OP
Gibby, Harold James.....27 Elm, Z8 ALR
Goodspeed, Harry B.....36 Pleasant, Z8 ALR
Holzer, William F.....36 Pleasant, Z8 OP
Lieberman, Harold.....175 June St. ALR
Messier, A. Eugene.....390 Main, Z8 OALR
Murphy, William E.....390 Main, Z8 ALR
Rice, John E.....390 Main, Z8 OALR
Rice, Theodore A.....390 Main St. OP
Tegelberg, Julius J.....12 Monterey rd., Z6 ALR
Whitney, Percy T.....390 Main, Z8 OP
Yasuna, Elton R.....44 Pearl St. OP

## MICHIGAN

ANN ARBOR
Barr, Albert S.....First Nat'l Bank bldg. OP
Falls, Harold Francis.....University Hospital OP
Fralick, F. Bruce.....First Nat'l Bank bldg. OP
Furstenberg, A. C.....First Nat'l Bank bldg. ALR
Maxwell, James H.....First Nat'l Bank bldg. ALR
Teed, R. Wallace.....410 Highland rd. ALR

BATTLE CREEK
Dodge, W. M., Jr.....1207 Central National Bank OP
Fraser, R. H.....25 W. Michigan av. ALR
Haughey, Wilfrid H.....Post bldg. OALR
Sleight, James Duane.....401 Security Bank bldg. OP
Wencke, Carl G.....1015 Security Tower ALR

## MICHIGAN — Continued

### BAY CITY

Criswell, Robert Harold.....Phoenix bldg. OP
Kessler, Mana.....311 Center av. ALR
Sherman, R. Newton.....Phoenix bldg. OALR
Urmston, Paul Robert.....303 Davidson bldg. ALR

### BENTON HARBOR

Cawthorne, Harold Jesse.....239 Pipestone ALR
Westervelt, H. O.....239 Pipestone ALR

### BIRMINGHAM

Ferris, Ralph G.....55 W. Maple av ALR

### COLDWATER

Thomas, James Alanson, Southern Michigan Bank bldg ALR

### DETROIT

Amberg, Emil.....1244 W. Boston blvd., Z2 ALR
Barnett, Saul E.....10 Peterboro ALR
Beattie, Robert.....David Whitney bldg., Z26 OALR
Begle, Howell L.....2730 E. Jefferson av. Z7 OP
Bentley, Neil I.....David Whitney bldg., Z26 OALR
Birch, John R.....1010 Maccabees bldg. Z2 ALR
Brown, Audrey O.....5057 Woodward av., Z2 OALR
Brunk, Clifford F.....7815 E. Jefferson av., Z14 ALR
Campbell, Duncan A.....David Whitney bldg., Z26 OALR
Carter, John M.....David Whitney bldg., Z26 ALR
Carter, Leland F.....David Whitney bldg., Z26 OP
Clifford, T. P.....David Whitney bldg., Z26 OALR
Cohoe, Don A.....13535 Woodward av., Z3 ALR
Cooper, Edmond L.....414 David Whitney bldg., Z26 OP
Croll, Leo J.....1326 Maccabees bldg. OALR
Croll, Maurice.....5057 Woodward ave., Z2 OP
Crossen, Henry F.....David Whitney bldg., Z26 ALR
Croushore, James E.....573 Fisher bldg., Z2 ALR
Cusick, Paul L.....1108 Stroh Bldg. Z26 OP
Davies, Windsor S...1302 Stroh Bldg., 28 W. Adams Ave. OP
Defnet, William A.....28 Adams av., West, Z26 ALR
Dill, J. Lewis.....Henry Ford Hospital, Z2 ALR
Dunlap, Henry Arthur.....7815 E. Jefferson av., Z14 OP
Gemeroy, Joseph C.....664 Fisher bldg., Z2 OP
Gleason, John E.....David Whitney bldg., Z26 ALR
Glowacki, Ben Francis.....Maccabee bldg., Z2 OALR
Gonne, William S.....David Whitney bldg., Z26 ALR
Goux, Raymond Salot.....David Whitney bldg., Z26 OALR
Hale, Arthur Stanley.....Eaton Tower, Z26 OALR
Hammond, Arthur.....David Whitney bldg., Z26 ALR
Harrell, Voss.....Hofman bldg, Z1 ALR
Hauser, I. Jerome.....606 David Broderick Tower, Z26 ALR
Hauser, Maurice J.....2311 Broderick Tower, Z26 OP
Heath, Parker.....David Whitney bldg., Z26 OP
Joinville, Euclide V.....Stroh bldg., Z26 ALR
Keating, T. F.....Maccabee bldg., Z2 OALR
Lamberson, Frank Abrom.....19600 Grand River, Z23 ALR
Lauppe, Frederick A....1801 David Whitney bldg., Z26 OP
Lepard, Cecil W.....David Whitney bldg., Z26 OP
Levitt, Edward J.....840-842 Maccabees bldg. ALR
MacFarlane, Howard W.....David Whitney bldg., Z26 ALR
May, Frederick T.....76 W. Adams, Z26 OP
McCullough, Lester E.....1553 Woodward av., Z26 OP
Mercer, R. E.....126 Monterey, Z3 LR
Mond, Edward .....Eaton Tower, Z26 ALR
Morton, J. B.....90 E. Warren av., Z1 OALR
Munson, F. T.....2201 E. Jefferson av., Z7 ALR
O'Hora, James Timothy .....571 Fisher bldg., Z2 ALR
Parker, Walter R.....David Whitney bldg., Z26 OP
Pino, Ralph H.....David Whitney bldg., Z26 OP
Pittman, John E.....David Whitney bldg., Z26 OP
Poos, E. E.....Fisher bldg., Z2 OALR
Potter, Willis A.....7450 Grand River av., Z4 ALR
Proctor, Bruce.....1419 David Whitney bldg., Z26 ALR
Reid, Wesley Grattan.....974 Fisher bldg. OP
Robb, James Milton .....David Whitney bldg., Z26 ALR
Roman, Stanley John.....742 Maccabees bldg., Z2 OALR
Ryerson, F. L .....Fine Arts bldg., Z26 OALR
Sage, Thomas.....7815 E. Jefferson Ave. ALR
Scarney, Herman D.....573 Fisher Bldg., Z2 OP
Shurly, Burt R.....62 Adams av. West, Z26 ALR
Simpson, H. Lee.....David Whitney bldg., Z26 ALR
Sugar, H. Saul.....2311 David Broderick Tower, Z26 OP
Summers, William S.....David Whitney bldg., Z26 OALR
Sutherland, Jacob Marion.....Fisher bldg., Z2 ALR
Vossler, Albert E.....David Whitney bldg., Z26 OALR
Weaver, Delmar Franklin.....Henry Ford Hospital, Z2 ALR
Wendel, J. S.....David Whitney bldg., Z26 ALR
Whitney, E. L.....Henry Ford Hospital, Z2 OP
Wilkinson, A. P. .....3001 W. Grand blvd., Z2 OALR
Woodry, Norman Lee, 1501 David Whitney bldg., Z26 ALR
Woodworth, William P.....2501 W. Grand blvd., Z8 OALR

### FENTON

White, Carl Harper.....106 River st. OALR

### FLINT

Barbour, Fleming A.....342 Bradley Ave. OP
Hubbard, Wm. B.....302 Paterson bldg., Z3 OALR
Smith, Eugene C.....814 Mott Foundation bldg., Z3 OALR
Stevenson, William W.....Kresge bldg., Z3 OALR
White, Herbert T.....National bldg., Z3 OP

### GRAND RAPIDS

Dean, Alfred .....Medical Arts bldg., Z2 OP
Dewar, Murray M.....Medical Arts bldg., Z2 OP
Doran, Frank.....Metz bldg., Z2 ALR
Flynn, J. Donald.....240 Kendall Professional bldg. ALR
Gilbert, Ralph H.....Metz bldg., Z2 OP
Heetderks, D. R.....Medical Arts bldg., Z2 ALR
Laird, Robert G.....114 E. Fulton st., Z2 OP
McRae, J. H.....Medical Arts bldg., Z2 OP
Moll, Arthur M.....Powers Theatre bldg., Z2 ALR
Smith, Ferris.....Blodgett Medical bldg., Z6 ALR-PIS
Steffensen, W. H., Blodgett Medical bldg.,
Wealthy St., Plymouth rd., Z6 ALR-PIS

### GROSSE POINT

Beam, A. Duane.....85 Kercheval ave., Z30 OP
Merkel, Charles C.....85 Kercheval av., Z30 ALR

### HOLLAND

DeVries, Harold G.....36 East 8th ALR

### IRON MOUNTAIN

Boyce, G. H.....First Nat'l Bank bldg. OALR
Menzies, Clifford G.....733 Woodward av. ALR

### JACKSON

Hardie, George C.....290 W. Michigan av. OALR
McGarvey, William Edward....Jackson City Bank bldg. OALR
Newton, Ray E.....1010 Reynolds bldg. OP

## MICHIGAN—Continued

KALAMAZOO
Fast, Ralph B.....136 E. Michigan av., Z4 ALR
Fulkerson, C. B.....107 W. Michigan av., Z9 OALR
Grant, Fred E.....Kalamazoo Nat'l Bank bldg., Z9 OALR
Marshall, Don.....1410 American Bank bldg., Z4 OP
Wilbur, Edward P.....438 W. South, Z46 OALR

LANSING
Behen, William C.....535 S. Capitol av., Z15 ALR
Burdick, Austin F.....P. O. Drawer 1378, Z4 OALR
Ellis, C. Ward.....MO Box 637 ALR
Heckert, Frank B.....1105 Bank of Lansing bldg. OP
McGillicuddy, O. B.....1908 Olds Tower bldg. ALR

MARQUETTE
Hornbogen, Daniel Powell.....401 Savings Bank bldg. OP
Keskev, George I.....Rose bldg. ALR
Nicholson, John Bixby.....401 Savings Bank bldg. ALR

MENOMINEE
Jones, William S.....521 Sheridan road OALR

MIDLAND
Pike, Melvin H.....Reinhart bldg. OALR

MONROE
Williams, Robert J.....25 Washington ALR

MT. CLEMENS
Croman, Joseph M., Jr.....115 Cass av. OALR

MT. PLEASANT
Strange, Russell H.....117 S. College av. ALR

MUSKEGON
Loder, L. L...506 Hackley Union Nat'l Bank bldg., Z11 OP
Mandeville, Clifford Bagley.....Hackley Union Bank ALR

NILES
Moore, Thomas Scott.....107 N. 2nd OP

OWOSSO
Pochert, Rolland C.....Matthews bldg. OALR

PETOSKEY
Conway, Wm. S.....314½ Howard St. OALR

PONTIAC
Baker, Frederick A.....4575 Motorway Dr. OP
Larson, Bertil T.....Riker bldg., Z15 ALR
Riker, Aaron D.....Riker bldg., Z15 OP

RIVER ROUGE
Flaherty, Norman W.....10573 W. Jefferson ave., Z18 OP

ROYAL OAK
Campbell, Mac D.....Washington Square bldg. OALR

SAGINAW
Cady, F. J.....402 S. Jefferson av. OALR
Cortopassi, Andre J.....324 S. Washington av. ALR
Cortopassi, Vital E.....324 S. Washington av. ALR
Curts, James H.....330 South Washington St. OP
Gomon, Louis D.....Eddy bldg. OP
Howell, Donald M.....Wiechmann bldg. ALR
McKinney, A. R.....330 S. Washington av. OALR
Richter, Harry Joseph.....604 2nd Nat'l Bank bldg. ALR
Slack, Walter K.....308 Eddy bldg., Z3 ALR
Tiedke, Gunther E.....120 N. Michigan av. OP

ST. CLAIR
Wetzel, John O.....1012 N. Riverside Dr. OP

ST. JOSEPH
McDermott, John J.....505 Pleasant st. OALR

SAULT ST. MARIE
Trapasso, Anthony J.....Sault Polyclinic ALR

SOUTH HAVEN
Urist, Martin J.....Center & Phoenix sts. OP

TRAVERSE CITY
Beall, John G.....118½ E. Front st. OP

## MINNESOTA

ALBERT LEA
Folken, Frank Garrett.....201 S. Broadway OALR

BEMIDJI
Garlock, Arthur Varney.....217½ 3rd OP

CROOKSTON
Morley, George A.....P. O. Box 452 OP
Oppegaard, C. L.....220 S. Broadway ALR
Parsons, J. G.....Crookston Clinic OALR

DULUTH
Armstrong, E. Leslie.....Medical Arts bldg., Z2 ALR
Athens, Alvin G.....Medical Arts bldg., Z2 OP
Fawcett, Keith R.....205 W. 2nd St. OP
Fellows, M. Fording.....1209 Medical Arts bldg. OP
Hilding, Anderson C.....Medical Arts bldg., Z2 OALR
Knapp, F. N.....Medical Arts bldg., Z2 OP
Olson, Archie.....Medical Arts bldg., Z2 OP
Patch, Orien B.....2905 E. Superior St. ALR
Tilderquist, D. L.....Medical Arts bldg., Z2 OALR
Winter, John A.....205 W. 2nd, Z2 OALR

FERGUS FALLS
Combacker, Leon C.....101½ Lincoln av. W. OP

HIBBING
Morsman, L. W.....Morsman Clinic OP
Sinamark, Andrew.....Power bldg. OALR

MANKATO
Johnson, Hobart Clemens.....839 Belgrade Ave. OP

MINNEAPOLIS
Boies, Lawrence R.....Medical Arts bldg., Z2 ALR
Borgeson, E. J.....Medical Arts bldg., Z2 OP
Cabot, Clyde M.....5629 12th Ave. S ALR
Camp, Walter E.....Medical Arts bldg., Z2 OALR
Cavanor, Frank Tracy.....1818 Medical Arts bldg., Z2 ALR
Dahl, Elmer O.....Medical Arts bldg., Z2 ALR
Dorge, Richard Irving.....1111 Nicollet av., Z2 OALR
Fink, Leo W.....Medical Arts bldg., Z2 ALR
Fink, Walter H.....Medical Arts bldg., Z2 OP
Garten, Joseph L.....502 Medical Arts bldg. OALR
Hansen, Erling W.....Medical Arts bldg., Z2 OP

## MINNESOTA — Continued

Haven, Walter K.....1009 Nicollet av., Z2 OALR
Hoffman, Walter Lees.....Medical Arts bldg., Z2 OP
Hymes, Charles.....Medical Arts bldg., Z2 OP
Jerome, Bourne.....1750 Medical Arts bldg., Z2 OP
Johnson, Malcolm R.....645 Medical Arts bldg., Z2 ALR
Koller, Hermann M.....Medical Arts bldg., Z2 OP
Lindberg, Vernon L.....Physicians & Surgeons bldg. OP
Litchfield, J. T.....Medical Arts bldg., Z2 OALR
Loomis, E. A.....Medical Arts bldg., Z2 OALR
Macnie, J. S.....Medical Arts bldg., Z2 OALR
Matthews, Justus.....Medical Arts bldg., Z2 LR
McGeary, George E.....Medical Arts bldg., Z2 ALR
Minsky, Armen A.....626 LaSalle bldg. ALR
Oberg, Carl M.....300 Physicians & Surg. bldg., Z2 OALR
Pfunder, Malcom C.....Medical Arts bldg., Z2 ALR
Phelps, Kenneth A.....Medical Arts bldg., Z2 OALR
Pratt, Fred J.....Physicians and Surgeons bldg., Z2 OALR
Priest, Robert E.....302 Medical Arts bldg., Z2 ALR
Sandt, Karl E.....1750 Medical Arts bldg., Z2 OP
Schwartz, Virgil Joseph.....Medical Arts bldg., Z2 ALR
Stanford, Charles E.....Medical Arts bldg., Z2 OP
Strout, G. Elmer.....1009 Nicollet av., Z2 OALR
Tangen, George M.,
2640 Glenhurst av., St. Louis Park, Z16 ALR

NEW ULM
Fritsche, Theodore R.....Fritsche bldg. OALR
Reineke, George F.....Fritsche bldg. OALR

RED WING
Brusegard, James F.....414 Main OP

ROCHESTER
Bair, Hugo L.....Mayo Clinic OP
Benedict, William L.....Mayo Clinic OP
Brown, Henry A.....1522 Damon Crt. S.E. ALR
Erich, John B. .....Mayo Clinic PI S
Figi, Frederick A.....Mayo Clinic LR-PI S
Hallberg, Olav Erik.....Mayo Clinic ALR
Havens, Fred Z.....Mayo Clinic ALR-PI S
Hempstead, Bert E.....Mayo Clinic ALR
Lillie, Harold I.....Mayo Clinic ALR
McBean, James Blish.....302 Seventh av. S. W. ALR
New, Gordon B.....Mayo Clinic L-PI S
Prangen, Avery de H.....Mayo Clinic OP
Rucker, C. W.....Mayo Clinic OP
Simonton, Kinsey M.....Mayo Clinic ALR
Wagener, Henry P.....Mayo Clinic OP
Williams, Henry Lane.....Mayo Clinic ALR

ST. CLOUD
Gaida, Joseph Benedict.....St. Mary's bldg. OP
Wenner, W. T.....St. Mary's bldg. OP

ST. PAUL
Binger, H. E.....Lowry Medical bldg., Z2 OALR
Boeckmann, Egil.....Lowry Med. bldg., Z2 OALR
Brown, John C.....Medical Arts bldg., Z2 OALR
Burch, Edward P.....Hamm bldg. OP
Burch, Frank E.....754 Linwood Place, Z5 OP
Connor, Charles E.....Lowry Medical bldg., Z2 ALR
Edwards, Thomas J.....1237 Lowry Medical bldg., Z2 OP
Fogarty, Charles W.....372 St. Peter, Z2 OALR
Grant, H. W.....Lowry Medical bldg., Z2 OP
Hilger, Jerome Andrew.....444 Lowry Medical Arts bldg. ALR
Hochfilzer, John J.....Lowry Medical bldg., Z2 ALR
Holmen, Robert W.....662 Lowry Med. Arts bldg., Z2 ALR
Kennedy, William A.....372 St. Peter st., Z2 OP
Larsen, Carl L.....Lowry Medical bldg., Z2 OALR
Leavenworth, R. O.....Lowry Medical bldg., Z2 OP
Levin, Bert G.....1210 Lowry bldg., Z2 ALR
Nelson, Louis A.....Lowry Medical bldg., Z2 OP
O'Reilley, Bernard E.....641 Lowry Med. Arts bldg., Z2 OP
Rothschild, Harold J.....Lowry Medical bldg., Z2 OALR
Schmidtke, Reinhardt L...441 Lowry Med. Arts bldg., Z2 OP
Simons, Leander T...1039 Lowry Medical Arts bldg., Z2 OP
Tracht, Robert R.....Lowry Medical bldg., Z2 OP
Walsh, Francis Mark.....330 Lowry Med. Arts bldg., Z2 OP
Wheeler, Merritt W.....Lowry Medical bldg., Z2 OALR
Wold, K. C.....Lowry Medical bldg., Z2 OALR

WINONA
Loomis, George L.....310 Choate bldg. ALR

WORTHINGTON
Stanley, C. R.....508 Tenth ALR

## MISSISSIPPI

CLARKSDALE
Wilkins, Ellis LeRoy.....McWilliams bldg. ALR

COLUMBUS
Naugle, Thomas Calvert.....1200 12th St. N. ALR

CORINTH
Hamrick, DeWitt.....601 Jackson ALR

GREENVILLE
Montgomery, D. Cameron.....Weinberg bldg. ALR

GREENWOOD
Pegues, James Cary.....301 Howard st. ALR

GRENADA
Caruthers, Samuel Brooks.....Grenada Clinic OALR

GULFPORT
Hays, Arthur V.....1300 27th ave. ALR
McWilliams, Charles A.....1300 27th av. ALR
Rouse, H. K., Jr.....McWilliams Clinic, Box 174 OP

JACKSON
Adkins, George E.....Jackson Infirmary, Z27 ALR
Anderson, Ross Epting.....Standard Life bldg., Z27 ALR
Hagaman, Van Dyke.....Lamar Life bldg., Z2 ALR
Harris, Robin.....964 N. State, Z6 ALR
Hollowell, Frederick Dixon.....705 Lamar Life bldg. ALR
Hughes, William Lauch.....Lamar Life bldg., Z2 ALR
McNair, Stirling S.....312 N. Congress st. OP

McCOMB
Cotten, Willis Frank.....202 State st. ALR

NATCHEZ
Gaudet, Lucien S.....405 Franklin OALR

OXFORD
Guyton, B. S.....512 Van Buren av. OALR

VICKSBURG
Edwards, C. J.....First Nat'l Bank bldg. OALR
Hall, Donald S.,
The Street Clinic Library, Crawford and Monroe sts. OALR
Jones, Edley H.....1301 Washington OALR

## MISSOURI

BOWLING GREEN
Wilcoxen, Wm. B. ALR

CAPE GIRARDEAU
Tygett, Glenn J. 215 N. Sprigg ALR

CARTHAGE
Powers, Everett 305 S. Main OALR

CLAYTON
Votaw, Robert E. 10 Warson Terrace, Z5 ALR

COLUMBIA
Bruner, Claude R. Exchange Nat'l Bank bldg. OALR

HANNIBAL
Hornback, George A. 500 Broadway ALR

INDEPENDENCE
Green, Stanley L. 129 W. Lexington OP

JEFFERSON CITY
Stauffer, Harry B. 234 Madison ALR

JOPLIN
Post, Winfred L. 804 Frisco bldg. OALR

KANSAS CITY
Allen, Charles E. Waldheim bldg., Z6 OALR
Altringer, A. N. Professional bldg., Z6 ALR
Baer, Alvin J. 1404 Bryant bldg., Z6 OP
Barry, Wm. B. 441 Huntington rd. ALR
Beal, Homer A. 1000 Professional bldg. ALR
Beil, J. Wallace (Retired) 1205 W. 59th st. OALR
Black, W. Byron Professional bldg., Z6 ALR
Bower, Richard L. 1103 Grand av., Z6 OALR
Connell, Evan Shelby Commerce bldg., Z6 OP
Curdy, R. J. 1102 Grand av., Z6 OP
Curran, Desmond 1805 Federal Reserve bldg. OP
Curran, E. J. Federal Reserve Bank bldg., Z6 OP
Curran, Kevin Federal Reserve Bank bldg., Z6 OP
Dixon, O. Jason Professional bldg., Z6 ALR
Eubank, Ambrose E. Bryant bldg., Z6 OALR
Gilliland, O. S. Professional bldg., Z6 ALR
Howard, J. W. (Retired), Residence 6026 McGee, Z2 OALR
Howard, John C. Jr. 1408 Professional bldg. ALR
Keith, W. E. Professional bldg., Z6 OALR
Knight, John S. 5800 High Drive ALR
Lemoine, A. N. Rialto bldg., Z6 OP
Lux, Paul Bryant bldg., Z6 ALR
May, James W. 1016 Rialto bldg., Z6 OP
McAlester, Andrew W., Jr. Bryant bldg., Z6 OP
McAlester, Andrew W., 3rd 2003 Bryant bldg., Z6 OP
McKee, Joseph W. Professional bldg., Z6 OP
McLeod, John, Major (MC)
c/o Dr. Hugh Miller, 40 E. 55th St. OP
Miller, Hugh Argyle bldg., Z6 OALR
Myers, John L. Shukert bldg., Z6 OALR
Roberts, S. E. Professional bldg., Z6 ALR
Shofstall, Charles Kirkpatrick Balcony bldg., Z2 ALR
Simpson, M. B. Professional bldg., Z6 ALR
Teall, Raymond E. Professional bldg., Z6 ALR
Thomason, H. E. Professional bldg., Z6 ALR
Trowbridge, Barnard C. 300 W. 47th st., Z2 ALR
Yazel, Herman E. Rialto bldg., Z6 OALR

KENNETT
Linton, Clair S. c/o Cottonseed Delinting Corp. ALR

ROLLA
Siniscal, Arthur Alfred M. Trachoma Hosp. OP

ST. JOSEPH
Minton, William H. Corby bldg., Z9 OALR

ST. LOUIS
Alden, Arthur M. Frisco bldg., Z1 ALR
Alvis, Bennett Y. 900 Carleton bldg., Z1 OP
Alvis, Edmund B. 308 N. 6th st., Z1 OP
Arbuckle, Millard F., Col. (M.C.), 7118 Washington, Z5 ALR
Bardenheier, F. G. A. Frisco bldg., Z1 ALR
Beisbarth, Carl 3720 Washington blvd., Z8 OP
Birsner, Louis J. 1232 Missouri bldg. ALR
Bisno, Daniel 4500 Olive st., Z8 OP
Black, W. D. Metropolitan bldg., Z3 ALR
Boemer, L. C. 3720 Washington blvd., Z8 ALR
Brown, Clyde O. 3720 Washington blvd., Z8 ALR
Bryan, William T. K. 3720 Washington blvd., Z8 ALR
Burns, S. S. Missouri Theatre bldg., Z3 ALR
Byrnes, Victor A., Col. (MC)
City Hospital, 1515 Lafayette st. OP
Charles, Joseph W. Humboldt bldg., Z3 OP
Cone, Alfred J. 453 N. Taylor, Z8 ALR
Costen, J. B. Beaumont Medical bldg., Z8 ALR
Cutler, Harold M. 640 S. Kingshighway, Z10 ALR
Dean, Lee W., Jr. 3720 Washington blvd., Z8 ALR
Dobbs, Otto Ross 9927 Riverview dr., Z15 ALR
Drews, Leslie C. 516 Metropolitan bldg., Z3 OP
Dyer, C. P. R. 307-508 N. Grand blvd., Z3 OP
Eber, Carl Theodore Carleton bldg., Z1 OP
Eimer, C. E. 462 N. Taylor av., Z8 ALR
Elz, Jule Theodore 3606 Gravois blvd., Z16 OP
Fincharg, Maxwell 607 North Grand blvd., Z3 ALR
Freimuth, L. E. University Club bldg., Z3 ALR
Gissy, C. J. 717 Metropolitan bldg., Z3 OP
Glick, Harry N. 1504 S. Grand blvd., Z4 ALR
Green, John 3720 Washington blvd., Z8 OP
Greene, Maurice L. 501 Missouri Theatre bldg., Z3 OP
Gundelach, C. A. University Club bldg., Z3 ALR
Hamel, French Keller 634 N. Grand av., Z3 ALR
Hanser, Samuel Albert 3424 Longfellow blvd., Z4 OP
Hardesty, J. F. Missouri Theatre bldg., Z3 OP
Hardy, Guerdan 539 N. Grand blvd., Z3 OALR
Hardy, W. F. Humboldt bldg., Z3 OP
Harkins, Wm. Blake 3720 Washington ave., Z8 ALR
Hildreth, H. Rommel Metropolitan bldg., Z3 OP
Hobart, Carl 3601-A Gravois Ave., Z16 OP
Hooss, C. H. A. 2333 S. Grand blvd., Z4 OP
Howard, Harvey J. Park Plaza Hotel, Z8 OP
James, William M. 508 N. Grand av., Z3 OP
Jones, Vincent L. Missouri Theatre bldg, Z3 OALR
Kelley, Isaac D., Jr. 3903 Park Ave., Z10 ALR
Kennedy, Francis L. Missouri Theatre bldg., Z3 ALR
Klaff, Daniel David 3720 Washington blvd., Z8 ALR
Lange, Adolph Charles 3903 Olive, Z8 OP
Leavy, C. A. 9054 Clayton rd., Z17 ALR
Luedde, W. H. Metropolitan bldg., Z3 OP
Lyman, Edward H. 308 North Sixth St., Z1 ALR
Lyman, Harry W. 308 N. 6th, Z1 ALR
Marmor, William A. 607 N. Grand blvd. ALR
Mason, Roy E. Frisco bldg., Z1 OP
Mattis, Robert Dean 375 N. Taylor av., Z8 OP
McCrory, James Lynn 607 No. Grand, Z3 ALR
McMahon, Bernard John Missouri Theatre bldg., Z3 ALR

## MISSOURI — Continued

Meinberg, William H.....906 Olive, Z1 OP
Miller, H. Edward.....634 N. Grand blvd., Z3 ALR
Milligan, Roy H.....607 N. Grand blvd., Room 205 ALR
Morman, William Daniel.....3507 Franklin av., Z6 ALR
Myers, E. Lee.....3903 Olive st., Z8 ALR
Nemours, Paul Roland.....1506 Hodiamont av., Z12 ALR
Payne, R. J.....University Club bldg., Z3 ALR
Pelz, Mort D.....607 N. Grand blvd., Z3 ALR
Post, Lawrence T.....Metropolitan bldg., Z3 OP
Post, M. Hayward.....Metropolitan bldg., Z3 OP
Potter, Allen B.....Pasteur Medical bldg., Z8 ALR
Presnell, C. A.....634 N. Grand av., Z3 ALR
Proetz, A. W.....Beaumont bldg., Z8 ALR
Reeh, Merrill J.....640 S. Kingshighway, Z10 OP
Reim, Hugo.....Metropolitan bldg., Z3 OP
Ryan, L. M.....Wall bldg., Z8 ALR
St. John, Dewey.....Metropolitan bldg., Z3 ALR
Sanders, Theodore E.....Metropolitan bldg., Z3 OP
Sauer, W. E.....Beaumont Medical bldg., Z8 ALR
Scholz, R. P.....Metropolitan bldg., Z3 ALR
Schwartz, Frederick O.....508 N. Grand blvd., Z3 OP
Scobee, Richard G.....640 S. Kingshighway, Z10 OP
Senturia, Ben H.....6355 Waterman ALR
Shahan, W. E.....Metropolitan bldg., Z3 OP
Smit, William M.....5285 Washington blvd., Z8 ALR
Stutsman, Albert C.....539 N. Grand blvd., Z3 ALR
Tooker, C. W.....Carleton bldg., Z1 OP
Walsh, Theodore E.....640 S. Kingshighway blvd., Z10 ALR
Webb, M. A.....4500 Olive st., Z8 ALR
Westlake, Samuel B.....Beaumont Medical bldg., Z8 ALR
Woodruff, Frederick E.....Metropolitan bldg., Z3 OP

### SEDALIA

Brady, Charles Henry.....205 Ilgenfritz bldg. ALR

### SPRINGFIELD

Bailey, Harold.....Landers bldg. OALR
Cheek, Carey.....Medical Arts bldg. OP
Klingner, Thomas O.....951 So. Pickwick OALR
Smith, Carlie Souter.....Holland bldg. OALR
Williams, John W.....642-660 Med. Arts bldg. ALR

### TRENTON

Ambrose, Elmer C.....910 Main OALR

### UNIVERSITY CITY

Milder, Benjamin.....735 Leland ave., Z5 OP
Rosenbaum, Harry D.....7310 Cornell, Z5 OP
Schluer, Elmer P.....7317 Stanford ave., Z5 ALR
Wood, V. Visscher.....6368 Washington, Z5 ALR

### WEBSTER GROVES

Bryan, James H.....242 Bristol rd. OP

## MONTANA

### ANACONDA

Dunlap, Lawrence G.....101 Main OALR

### BILLINGS

Morledge, Roy V.....Hart-Albin bldg. ALR
Morrison, James D.....238 Hart-Albin bldg. OP
Morrison, Wm. R.....Hart-Albin bldg. OALR

### GREAT FALLS

Hurd, Fritz D.....Medical Arts bldg. ALR

### KALISPELL

Huggins, Harrison D.....12 Whipps bldg ALR

### MILES CITY

Harlowe, Harold Douglas..Garberson Clinic, Colony bldg. ALR
Rowen, Ernest Henry.....Box 126 ALR

### MISSOULA

Marshall, William J.....Montana block ALR
Murphy, Edward S.....309 Eddy av. OP
Sale, George Goble.....216 Dixon bldg. ALR

### ROUNDUP

Lewis, George A.....213 Main OALR

## NEBRASKA

### COLUMBUS

Campbell, C. H.....2514½ Thirteenth OALR
Koebbe, E. E.....ALR

### FREMONT

Heine, Lyman H.....635 N. Main ALR
Heine, William Howard.....Odd Fellow bldg. ALR

### GRAND ISLAND

Martin, Roy David.....Masonic bldg. ALR

### HASTINGS

Foote, Charles M.....412 N. Hastings av. OP

### KEARNEY

Hart, Noah Leon
Capt. (M.C.), 0-460831 Station Hospital, AAF ALR
Wilcox, Malcolm B.....5 West 21st St. ALR

### LINCOLN

Black, Paul.....Stuart bldg., Z8 OALR
Brooks, Earl B.....128 North 13th st., Z8 OALR
Hompes, J. J.....Sharp bldg., Z8 OALR
Peterson, John C.....702 Sharp bldg. OP
Sanderson, David D.....Stuart bldg., Z8 OALR
Thomas, James W.....1310 Crestdale rd., Z8 OALR
Wood, Maynard A.....1037 Stuart bldg. OP
Zemer, S. G.....Stuart bldg., Z8 OALR

### NEBRASKA CITY

Stonecypher, D. D.....102½ S. 9th st. ALR

### OMAHA

Alliband, George Thomas.....1020 Medical Arts bldg. OP
Bushman, L. B.....City National Bank bldg., Z2 OALR
Cassidy, W. A.....Medical Arts bldg., Z2 ALR
Coakley, Leo P.....Medical Arts bldg., Z2 ALR
Davis, John Calvin, Jr.....425 Aquila court, Z2 OALR
Finegan, James F.....415 Medical Arts bldg., Z2 OP
Gifford, Harold.....Medical Arts bldg., Z2 OP
Greenburg, Maynard M.....516 Medical Arts bldg., Z2 OP
Hahn, William N.....City Nat'l Bank bldg., Z2 OP
Haney, William P.....Medical Arts bldg., Z2 OALR
Judd, J. H.....Medical Arts bldg., Z2 OP
Kully, Herman E.....Medical Arts bldg., Z2 ALR
Morrison, Wm. Howard.....Medical Arts bldg., Z2 OP
Potts, J. B.....3817 Dewey av., Z5 ALR
Rasgorshek, Robert Houston.....425 Aquila Court OP
Rubendall, C.....Medical Arts bldg., Z2 OALR
Smith, Thomas T.....211 Medical Arts bldg., Z2 ALR
Stokes, William H.....Medical Arts bldg., Z2 OP
Swab, Charles M.....Medical Arts bldg., Z2 OP
Watke, Frederic Martin.....Medical Arts bldg., Z2 ALR

## NEVADA

LAS VEGAS
Allen, Wilmer L.....201 N. 8th St. ALR
Lockwood, Chester C.....218 S. 5th st. OP

RENO
Clarke, Samuel T.....Medical Dental bldg. OP
Creveling, Earle LaMont.....P. O. Box 2446 ALR
Fuller, John Andre.....First Nat'l Bank bldg. OALR
Gillespie, Elmer H.....6 State st. ALR

## NEW HAMPSHIRE

CONCORD
MacMillan, Andrew L., Jr.....46 Pleasant ALR

DOVER
Reid, Thomas F.....538 Central av. ALR

HANOVER
Auten, Hanford Louis, Jr.....Dartmouth Eye Institute OP
Murtagh, John Anthony.....Hitchcock Clinic ALR
O'Brien, James Miles.....Dartmouth Eye Institute OP

KEENE
Ford, Leroy Sargent.....30 School St. ALR

MANCHESTER
Monette, C. J.....814 Elm ALR
Rahmanop, Walter B.....1426 Elm ALR
Togus, Leopold T.....967 Elm ALR

NASHUA
Marcotte, Raymond H.....196 Main ALR
Nutter, C. F.....16 Amherst OP

PORTSMOUTH
Allen, Cary D. .....350 State st. ALR

## NEW JERSEY

ALLENHURST
Baeseman, R. W.....112 Main St. OALR

ASBURY PARK
Fisher, James A.....Jersey Central bldg. OALR
Upham, Helen F.....305 Third av. OALR

ATLANTIC CITY
Charlton, C. Coulter.....1616 Pacific ave. ALR
Dalton, S. Eugene.....101 S. Surrey av., Ventnor ALR
Gordon, Benjamin L.....Professional Arts bldg. OP
Harley, Robison D.....101 S. Indiana Ave. OP

BLOOMFIELD
Harris, Morris.....102 Broad st. OP

CAMDEN
Cox, John R., Capt. (MC).....202 S. 34th st. OP
Kline, Oram R.....514 Cooper ALR
Meyer, George P.....410 Haddon av. OP
Rutterberg, Max.....303 Cooper ALR
Shemeley, William G., Jr.....7 Haddon av. OALR
Shipman, James S.....514 Cooper OP
Shope, Edward Pierce L.....807 Wilson bldg. OP

EAST ORANGE
Buvinger, Charles W.....50 Washington OALR
Campbell, William.....144 S. Harrison ALR
Cregar, John S.....440 Harrison OP
Jaeckle, Charles Eisele.....136 Evergreen Place OP

ELIZABETH
Ehrlich, Max.....721 N. Broad st., Z3 ALR
Gadomski, Casimir Francis.....331 S. Broad st., Z2 OP
Goldmacher, H. B.....113 Elmora av., Z2 ALR
Griesemer, Z. Lawrence.....1145 E. Jersey st., Z1 OP
Kapp, Carl G.....440 Westminster av., Z3 ALR

ENGLEWOOD
Worcester, George F.....220 Engle ALR

GREYSTONE PARK
Gambill, Perry Judson.....New Jersey State Hospital ALR

HACKENSACK
Berke, Raynold N.....430 Union OP
Dezer, Charles N., Jr.....210 Main ALR
Greenfield, William John.....50 Anderson ALR
Hensle, Otto S.....250 Prospect av. ALR
Liva, Arcangelo.....5 Pangborn place OALR
Spiegelglass, A. B.....417 Main ALR

HADDONFIELD
Shafer, A. H.....E. Greenman rd. ALR

JERSEY CITY
Amdur, Louis A.....2540 Boulevard, 15 Exchange Pl., Z4
Jaffe, Benjamin.....566 Bergen Ave., Z4 OP
Pyle, Wallace.....15 Exchange place, Z2 OALR
Zbar, Joseph E.....2690 Boulevard, Z6 ALR

LONG BRANCH
Strauss, Arthur.....130 Pavilion av. ALR

MONTCLAIR
Agnew, Hobart McV.....1f Plymouth OP
Shannon, James B.....66 S. Fullerton av. ALR
Shannon, L. M.....66 S. Fullerton av. ALR

MOORESTOWN
Hebble, Howard Miller.....320 Chester av. ALR
Ulmer, David H. B.....199 Chestnut ALR

MORRISTOWN
Byrne, J. Arthur.....16 Elm ALR
Mathews, Raymond H.....186 South ALR

NEWARK
Adelman, Benjamin B.....186 Clinton av., Z5 OP
Barkhorn, Charles W.....223 Roseville Ave. ALR
Berney, Irving Vincent.....31 Lincoln Park, Z2 ALR
Cardwell, Edgar P.....965 Broad st. ALR
Fischer, Louise.....Med. Towers, 31 Lincoln Park, Z2 ALR
Goldman, Jerome.....1 Johnson Ave., Z8 OP
Goldstein, Henry Z.....31 Lincoln Park ALR
Hahn, William H.....198 Clinton av., Z5 OP
Hughes, L. W.....965 Broad, Z7 OP
Hurff, J. Wallace.....671 Broad st., Z2 OALR
Keim, William Franklin Jr.....15 Washington St., Z2 ALR
Krone, William Frank.....31 Lincoln Park, Z2 ALR
Orton, Henry B.....224 Delavan av., Z4 ALR
Peer, L. A.....15 Washington St., Z2 ALR-PLS
Rosen, Emanuel.....692 High st. OP
Sherman, A. Russell.....671 Broad, Z2 OP

## NEW JERSEY — Continued

Sherman, Elbert S.....671 Broad, Z2 OP
Wood, Earl LeRoy.....225 Ballantine Pkwy. Z4 ALR
Zehnder, A. Charles.....188 Roseville av., Z7 OP

NEW BRUNSWICK
Howley, Bartley Maxwell.....67 Livingston Ave., Z2 ALR
Kler, Joseph Henry.....151 Livingston av. ALR
Sadoff, Irvin E.....163 New St. ALR

ORANGE
Emerson, Linn.....303 Park av. OALR

PASSAIC
Ehrenfeld, Edward.....185 Lexington av. OP

PATERSON
Atwood, Edward A.....360 Park av. Z4 ALR
Denison, Ward C.....316 Broadway ALR
Meer, Nadhim Edward.....400 Park ave. ALR

PERTH AMBOY
Cole, Nathan B.....104 Market OALR
Mann, J. J.....255 State ALR

PLAINFIELD
Childers, Robert Jefferson.....604 Park av. ALR
Wolff, Jerome Marshall.....822 Park Ave. OP

PRINCETON
Abrams, Henry.....195 Nassau St. OP

RIVERSIDE
Geary, Russell Diener.....337 Bridgeboro road ALR

SOUTH ORANGE
Miller, Joseph A.....364 Prospect st. ALR
O'Connor, D. F.....27 S. Kingman rd. OALR

SOUTH PLAINFIELD
Levy, Abram.....1401 Plainfield av. OP

SUMMIT
Burritt, Norman W.....30 Beechwood road ALR
McAlpine, Paul Theodore
Summit Med. Grp, 129 Summit Ave. OP
Sly, John L.....382 Springfield av. ALR

TEANECK
Littwin, Charles.....950 Queen Anne road OALR

TENAFLY
Lewin, Michael.....149 Columbus Ave. ALR-PIS

TRENTON
Ivins, William Clifford.....455 W. State, Z8 ALR
Levy, Irvin.....222 W. State st., Z8 OP
Moriconi, Albert F.....438 Hamilton av. ALR
Wilner, Arthur S.....225 West State st. ALR

UNION CITY
Conty, Anthony J.....318 Forty-eighth ALR
Pagliughi, John J. .....1915 Palisade av. ALR
Sacco, Anthony G.....2200 New York av. OALR

WEEHAWKEN
Ash, Arthur F.....710 Boulevard East ALR

WESTFIELD
Roh, Robert F.....727 Glen av. ALR
Schillinger, Raphael.....858 Summit Ave. ALR
Tomlinson, Rolland Davis.....505 East Broad st. ALR

## NEW MEXICO

ALBUQUERQUE
Brehmer, Harrison L.....First Nat'l Bank bldg. OALR
Peck, Howard Burgess.....106 S. Girard Ave. OP

ROSWELL
Morrison, George S.....308 W. 2nd st. ALR

SANTA FE
Barton, William C.....813 Don Gasper av. OALR
Brooks, Jack E.....c/o P.O. Box 1539 ALR
Seitz, Howard M.....Coronado bldg. ALR

## NEW YORK

ALBANY
Bedell, Arthur J.....344 State, Z6 OP
Cetner, John A.....365 State St..OP
Dowling J. Ivimey.....116 Washington av., Z6 OALR
Dubins, Henry B.....248 State st., Z6 OP
Freund, E. M.....762 Madison av., Z3 ALR
Holohan, Joseph Leo.....330 State, Z6 OALR
Judne, Harry V. .....46 Willett, Z6 OP
Kemp, Roy C.....343 State, Z6 ALR
Mott, Walter C.....75 Willett st., Z6 OP
Simon, Emanuel.....377 State St. ALR
Tebbutt, H. K.....240 State, Z6 ALR
Volk, Benjamin M.....146 Washington Ave., Z6ALR

AUBURN
Bullard, C. W.....120 Genesee ALR
Platt, E. S.....Hamilton Ave. OP
Wiley, Jason L.....Metcalf bldg. OP
Wood, Everet H.....120 Genesee St. OP

BALDWIN
Gaillard, Milton T.....5 Park av. OP

BATAVIA
Wilson, Robert G.....304 East Main st. ALR

BAY SHORE
McGrath, Harry.....12 Shore Lane OP

BAYSIDE
Vadala, Anthony J., Col. (M.C.),
Station Hosp., Fort Totten OALR

BINGHAMTON
Bogdasarian, Robert Michael.....108 Oak St. ALR
Levy, Harry H.....119 Murray OP
Monserrate, Manuel Maria.....73 Main ALR
Randall, Hiram.....81 Main ALR
Smith, Windsor R.....67 Front ALR
Tobin, Henry Loughlin.....74 Front St. OP
Vogt, George C.....140 Chapin ALR
Watson, H. de Witt.....151 Front OALR

## NEW YORK — Continued

### BRONXVILLE

Branigan, C. Hugh....17 Kraft av. OP
Carter, William W....6 Chestnut av. ALR
Kenyon, Byron B....44 Pondfield rd. ALR
Petruzzi, William Astor....65 Pondfield rd., Z8 ALR
Ross, John....37 Oriole av. ALR
Wolcott, Charles C....23 Kraft ave., Z8 ALR

### BROOKLYN

Abrams, Murray....100 Woodruff Ave. ALR
Baker, John Percy....51 S. Oxford, Z17 ALR
Bancks, Howard Raymond....75 Lenox Rd., Z26 ALR
Behrens, Alvin....9 Prospect Park West, Z15 OP
Beinfeld, Henry H. ....760 Eastern Parkway, Z13 ALR
Berger, Meyer D....681 Ocean av., Z26 ALR
Bergida, Jerome J....1615 Dorchester rd. ALR
Bergmann, Milton B....959 Bushwick av., Z21 OP
Bernstein, David....1342 51st St. Z19 ALR
Blankfeld, Nathan....538 9th St. Z15 ALR
Blumenfeld, Louis....221 Brooklyn ave., Z13 ALR
Brooks, Morris J....455 Ocean av., Z26 ALR
Buonaguro, Michael J....589 Lorimer, Z11 OP
Chait, Robert A....20 Plaza St. ALR
Cutino, R. M....79 Pierrepont, Z2 OP
Cutler, Abraham Maurice....750 Ocean av., Z26 ALR
Dan, Julius Martin....1362 President st., Z13 OP
Ebeling, William B....54 Greene av., Z5 OP
Evans, J. N....23 Schermerhorn, Z2 OP
Feldman, Joseph....668 Eastern Parkway, Z13 ALR
Fine, Abraham....925 Ocean av., Z26 OALR
Fisher, George Garthwaite....271 New York av., Z16 ALR
Friedman, Joseph Jay....57 Midwood St., Z25 ALR
Garofalo, Charles Joseph....439 Seventy-seventh, Z9 ALR
Gerzog, Bennett G....891 St. Marks av., Z13 ALR
Gilbert, Joseph G....285 New York ave., Z16 ALR
Gilligan, Alexander....1154 Dean, Z16 ALR
Golding, Joseph E....1 Nevins, Z17 OP
Gottlieb, Bernard N....387 Eastern Parkway, Z16 ALR
Graham, George Albert....842 Carroll St., Z15 OP
Greenfield, Samuel D....169 New York av., Z16 ALR
Griffin, Edwin A....18 Prospect Park, West, Z15 ALR
Gromet, Maurice T....105 Lincoln rd., Z25 ALR
Gubner, Julius....25 Lefferts av., Z25 ALR
Gutkin, Morton Lewis....6734 6th Ave. ALR
Hargitt, Charles A....17 Schermerhorn, Z2 OP
Held, Albert E....732 Eastern Pkwy., Z13 ALR
Holzberger, John C....7005 Freshpond, Z27 ALR
Hull, Allen....401 76th st., Z9 OP
Inciardi, James A....149 Midwood St., Z25 OP
Kaplan, Carl....69-17 Forest av., Z27 ALR
Kasnetz, Joseph P....678 Eastern Parkway, Z13 ALR
Kaufman, Maxwell....85 Maujer, Z6 ALR
Kayser, Reuben....442 Bay Ridge Parkway, 75th st., Z9 ALR
Keil, Peter A....118 Pennsylvania av., Z7 ALR
Knudsen, Arnold F....419 Bay Ridge Parkway, Z9 ALR
Kopp, Mortimer M....79 Argyle road, Z18 ALR
Kravitz, Daniel....861 Park place, Z16 OP
Kreag, Karl A. ....167 Joralemon, Z2 ALR
Krimsky, Emanuel....745 Eastern Pkwy. OP
Kruskal, I. D....101 Lafayette av., Z17 OP
Kuritsky, Isadore....1480 Nostrand av., Z26 ALR
Lasky, Mortimer A....Fox Medical bldg., 1 Nevins st. OP
Levitt, Jesse M....991 Ocean ave., Z26 OP
Lloyd, Ralph I....14 Eighth av., Z17 OP
Lombardo, Melchiore....142 Joralemon st, Z2 OP
Lupo, Carl W....215 St. Johns Place, Z17 OP
Mallon, Frank E., The Williamsburgh Bank bldg., 1 Hanson Pl. Z17 OP
Menger, Louis Charles....202 Weirfield, Z21 ALR
Mensher, Ira Wadsworth....50 Plaza st., Z17 OP
Mezz, David....330 New York av., Z13 ALR
Mochle, Walter....15 Schermerhorn, Z2 OP
Moore, Walter V....1 Nevins, Z17 OP
Moorhead, Robert L....125 Remsen, Z2 ALR
Moyer, John Sanford....901 Washington av., Z25 ALR
Nelson, Nathaniel F....4910 15th av., Z19 OP
O'Brien, G. R. ....307 Sterling pl., Z17 ALR-PIS
Ohly, John H....22 Schermerhorn, Z2 OP
Place, E. Clifford....59 Livingston, Z2 OP
Rabbiner, Max....358 New York av., Z13 ALR
Rizzuti, A. Benedict....160 Henry st., Z2 OP
Rosenthal, Benjamin Charles....140 New York av., Z16 OP
Roth, Aaron....184 Joralemon st. ALR
Ruchman, Jacob....301 Brooklyn av., Z13 ALR
Saskin, Edward....855 St. Marks av., Z13 OP
Schmidt, James M....40 S. Oxford, Z17 ALR
Scurti, Bernard V., Lt. (M.C.)....51 Harmon st., Z21 ALR
Sher, Maurice A....5022 Fifteenth av., Z19 ALR
Shorr, Abraham....225 Eastern Parkway, Z16 ALR
Silberman, Meyer....266 Brooklyn av., Z13 ALR
Silverberg, Joseph S....755 Ocean av., Z26 ALR
Sparber, Herman....4701 Fifteenth av., Z19 OP
Sporn, Hyman....839 Park Place ALR
Steinbugler, William F. C....515 Ocean av., Z26 OP
Stern, Wilhelm E....1099 Bushwick av., Z21 ALR
Sunde, Einar A....627 Second, Z15 ALR
Swift, Donald E....25 Prospect Park W. OP
Thaler, Nathan....570 Eastern Parkway, Z25 ALR
Tucker, Frank Landale, Jr....464 Ninth st., Z15 ALR
Usen, Louis....858 Lincoln Pl., Z16 ALR
Viviani, John J....180 Suydam st., Z21 ALR
Wechsler, A. H....340 New York av., Z13 ALR
Weeth, Charles R....37 Remsen, Z2 ALR
Weinstein, Samuel....441 Ocean av., Z26 ALR
Welt, Bernard....135 New York av., Z16 ALR
Wieselthier, Maurice L....1322 Union st., Z13 OP
Zwerling, Samuel....789 St. Mark's av., Z13 ALR

### BUFFALO

Barone, Michael Horace....468 Delaware av., Z2 ALR
Bender, Rosarie Rossell....195 Linwood Ave. ALR
Beyer, Louis J....449 Delaware av., Z2 ALR
Bozer, Herrmann Eugene....468 Delaware av., Z2 ALR
Brennan, James William....11 Horton Place OP
Brown, C. M....510 Delaware av., Z2 ALR
Buffum, Edward S....40 W. North, Z2 ALR
Cott, C. C....333 Linwood av., Z9 ALR
Cowper, H. W....543 Franklin, Z2 OP
Fairbairn, John F....925 Delaware av., Z9 ALR
Fowler, James Gwynne....412 Linwood av., Z9 OP
Freeman, Sheldon B....196 Linwood av., Z9 OP
Frost, Carl Grover....333 Linwood av., Z9 ALR
Gerstner, Martin Llewellyn....27 Admiral rd. ALR
Glauber, Jerome J....878 Lafayette, Z9 ALR
Hague, Eliott B....454 Franklin, Z2 OP
Harnick, Israel....1132 Delaware av., Z9 ALR
Howard, Wm. M....389 Linwood av., Z9 OP

## NEW YORK — Continued

Jones, W. Yerby....436 Sycamore st., Z4 OP
King, Walter F....519 Delaware av., Z2 OP
Koenig, Ivan J....40 W. North, Z2 OP
Krystaf, Joseph George....1004 Fillmore av., Z11 ALR
Lathbury, Edwin F....333 Linwood ave., Z9 ALR
Le Win, Thurber....112 Linwood av., Z9 OP
Luhr, A. F....40 W. North, Z2 OP
March, Clara A....465 Ashland, Z13 OP
Olmsted, K. Elizabeth Pierce....568 Lafayette av., Z13 OP
Pliss, Aaron....40 North St. OP
Riwchun, Meyer H....510 Linwood Ave. OP
Rosenberg, Joseph....333 Linwood av., Z9 ALR
Scanio, Joseph Charles....333 Linwood av., Z9 ALR
Schutz, Joseph A....2804 Main, Z14 OP
Smith, Leon....467 Linwood av., Z9 ALR
Soldineer, Marshall Y....333 Linwood Ave. ALR
Trotter, Homer A....40 W. North, Z2 ALR
Wurtz, W. J. M....333 Linwood av., Z9 ALR

### COOPERSTOWN

Kieler, Charles B....M. I. B. Hospital ALR

### CORTLAND

Higgins, R. P....20 Court OALR

### CRESTWOOD

Reid, Frederick Kessinger....119 Manhattan Ave., Z7 OP

### EAST AURORA

Andrews, H. D. ....429 Oakwood av. OP

### ELMIRA

Boland, Wm. T....378 W. Church st. OP
Glover, Arthur C....311 W. Church OALR
Monahan, Richard O'Brien....378 W. Church St. ALR
Ridall, Earle G....91 Durland Ave. ALR

### ENDICOTT

Hudock, John P....208 Jefferson Ave. ALR

### FAR ROCKAWAY

Hirschmann, Hans A....1502 Mott av. ALR

### FLUSHING, L. I.

Coleman, Benjamin....36-40 Bowne st. ALR
Goode, Edward Allen....143-28 Sanford av. ALR
Liggett, Harold....44-05 Parson blvd. ALR
Weiss, Harry....37-21 Parsons blvd. OP

### FOREST HILLS, L. I.

Kafka, M. Martyn....109-05 72nd av. ALR

### FREEPORT

Nova, Jules M....164 Pine St. OP
Rossman, Morris M....115 N. Main st. ALR

### GENEVA

Abbott, Henry B....574 So. Main st. OP
Fairfax, Kenneth T....423 Main st. OP
Spengler, John Arthur....423 S. Main OP

### GOWANDA

Sklarow, Louis....43 Center st. ALR

### GLEN FALLS

Buck, Clair A. ....191 Glen ALR
Edmunds, Stanley L....360 Glen St. ALR
Ryan, Edward P....226 Glen St. OP

### HEMPSTEAD, L. I.

Coon, Eugene H....131 Fulton av. ALR
Hughes, Wendell L....Professional bldg. OP-PI S
Jarrott, James T....131 Fulton av. OP
Maier, Marcus M....513 Front st. ALR
Maxwell, Hal W....Professional bldg. OP
Roszell, Leo H....131 Fulton ave. OP
Smith, Henry Buel....131 Fulton av. ALR

### HUNTINGTON

Kimbrig, Max....20 High ALR

### ITHACA

Wilson, Hudson J....First Nat'l Bank bldg. ALR

### JACKSON HEIGHTS, L. I.

Lefkon, Irving M....37-14 73rd ALR

### JAMAICA, L. I.

Bender, M. S....88-05 150th, Z2 ALR
Cirillo, Anchise Anthony....89-21 153rd St. ALR
Douglas, Edward M....87-85 153rd, Z2 OP
Kane, J. T....88-49 163rd, Z3 ALR
Keeler, Lansing H....89-04 148th, Z2 OALR
Miller, Saul....88-05 150th, Z2 OP
Minsky, Arthur....8601 Homelawn st., Z3 OP

### JAMESTOWN

Bourne, Malcolm G....110 W. 3rd OP
Shearer, George M....25 Spruce st. ALR

### JOHNSTOWN

Sarno, J. Frederick....109 S. William st. ALR

### KINGSTON

Cranston, Wm. J., Jr....175 Clinton Ave. OP
Wilson, Harold A....71 Maiden Lane ALR

### LANCASTER

Palmer, Milton A....18 Park blvd. OP

### LITTLE FALLS

Newton, Morris H....Burrell bldg. OP

### LOCKPORT

Morgana, Dante James....13 Main OP
Parker, Harry L....88 East av. ALR
Pierce, William W.... ALR

### LONG ISLAND CITY

Gewanter, Robert....41-44 44th st., Z4 ALR
Hulka, J. H....30-28 36th st. OALR
Sarnoff, Jack....31-11 31st St. ALR

### MIDDLETOWN

Hicks, William J....15 King ALR
Neller, Walter I....121 Wickham av. OP
Schlessel, Louis....101 E. Main St. ALR

### MILLERTON

Tabor, Gilbert Stevens....11 Park Row ALR

### MT. KISCO

Newlands, W. A....359 East Main ALR

### MT. VERNON

Budetti, Joseph A....90 Sherman Ave. ALR
Houlahan, James M....117 S. Second av. OP
Keefe, William John....205 E. Sidney av. OALR
Morrone, Francis X....117 South 2nd av. ALR
Vrooman, C. O....55 Claremont Ave. OP
Yavelow, Charles S....117 South Second av. ALR

## NEW YORK — Continued

### NEWBURGH

Browning, Birt Lee....208 Grand ALR
Burman, Daniel....121 1st St. ALR
McWilliam, Charles S....205 Liberty st. OP
Overton, J. Woodhull....P.O. Box 293 OALR

### NEW ROCHELLE

Beck, A. L....421 Huguenot OALR
Gibson, William J....650 Main st. ALR
Hetrick, L. E....260 North av. ALR
Smith, Morley T....421 Huguenot ALR

### NEW YORK CITY

Aebli, Rudolph....30 E. 40th, Z16 OP
Agatston, Howard James....57 West 57th St., Z19 OP
Allan, William B....410 Park av., Z22 OALR
..Allen, Benjamin Israel....1070 Park Ave. ALR
Almour, Ralph....57 W. 57th st., Z19 ALR
Altmann, Franz....10 E. 85th St., Z28 ALR
Anderson, Orrin E....17 E. 61st st. ALR
Arnold, Leon M....1235 Park Ave., Z28 ALR
Atkinson, Miles....310 Lexington ave., Z16 ALR
Auerbach, Julius....897 Park av., Z21 ALR
Austen, Willard Emerson....10 Park Ave. ALR
Baker, Daniel C., Jr....903 Park av., Z21 ALR
Ballin, M. J....25 E. 77st., Z21 ALR
Barnes, Mark Hopkins....324 E. 41st st., Z17 OALR
Bassen, Edward J....70 E. 66th st., Z21 OP
Bassett, David I....1075 Grand Concourse, Z52 ALR
Bell, Julius W....200 E. 16th St., Z3 ALR
Bellucci, Richard John....121 E. Sixtieth St., Z22 ALR
Bender, M. S....121 E. 60th, Z22 ALR
Berens, Conrad....35 E. 70th, Z21 OP-PI S
Berger, Isidore....888 Grand Concourse, Bronx, Z51 ALR
Berger, Murray....Central Park W., Z24 ALR
Berliner, M. L....57 W. 57th, Z19 OP
Bilchick, Edwin B....133 E. 58th St. ALR
Bloom, Samuel M....121 E. 60th St., Z22 ALR
Bogart, Donald Weeks....20 E. 53rd, Z22 OP
Bonaccolto, Girolamo....123 E. 61st st., Z21 OP
Bowers, W. C....17 E. 61st., Z21 ALR
Boyd, Greydon G., Capt. (MC)....307 E. 44th St. ALR
Boyes, T. L....654 Madison av., Z21 OP
Braley, A. E....635 W. 165th St., Z32 OP
Breuning, Paul H....133 E. 58th, Z22 ALR
Brighton, George R....580 Park av., Z21 ALR
Brikates, Peter....140 E. 54th St. OP
Brodnitz, Friedrich S....667 Madison av., Z21 ALR
Broones, Otto....302 W. 87th, Z24 ALR
Brown, Harold W....15 Park av., Z16 OP
Bruce, G. M....635 W. 165th st., Z32 OP
Bruder, Joseph....57 W. 57th, Z19 OALR
Burchell, Edgar B., New York Eye and Ear Infirmary, 2nd av., Corner 13th st., Z3
Burman, Herman J....1235 Grand Concourse, Z52 ALR
Cappeta, Domenico....125 East 84th St., Z28 OP
Carroll, Frank D....Eye Institute, 635 W. 165th, Z32 OP
Castroviejo, Ramon....635 W. 165th, Z32 OP-PI S
Chace, Robert R....635 W. 165th st., Z32 OP
Chamlin, Max....1840 Grand Concourse OP
Chinn, Chester W....209 W. 125th St. OALR
Cinelli, Albert A....1021 Park av., Z28 ALR
Cohen, Martin....29 E. 64th, Z21 OP
Cole, John Gordon....780 Park Ave. OP
Coleman, Joseph A....5051 Iselin av., Z63 ALR
Coleman, Lester Laudy....162 E. 80th, Z21 ALR
Conboy, John E. ....2265 University av., Z53 OP
Conley, John J....425 E. 51st st. ALR
Constantine, Elizabeth Frederick....340 E. 63rd st., Z21 OP
Corya, Harold W....30 E. 40th, Z16 ALR
Cracovaner, Arthur J....103 E. 78th st., Z21 ALR
Craig, Stuart Lessley....135 E. 65th St., Z21 ALR
Creasy, Raymond C....67 E. 59th, Z22 ALR
Crespo, Jose E....28 West 95th st., Z25 ALR
Cunning, Daniel S....121 E. 60th Z22 OALR
Daley, Jacob....104 E. 40th, Z16 ALR
Daly, John F....140 E. 54th St. ALR
Davidson, Morris....17 Park av., Z16 OP
Davis, G. E....14 E. 90th st., Z28 OALR
Davis, Hugh P....135 E. 65th St. Z21 ALR
Della Rocca, Anthony J....1400 Benson St., Z61 ALR
Depping, Charles William....17 W. 54th, Z19 ALR
DeVoe, Arthur Gerard Inst. of Oph., 635 W. 165th St., Z32 OP
Dillemuth, Frederick Joseph....404 E. 154th, Z55 ALR
Dlugasch, Louis....110 E. 36th, Z16 ALR
Doherty, W. B....150 W. 55th, Z19 OP
Druss, Joseph George....1040 Park av., Z28 ALR
Dunnington, John H., Institute of Ophthalmology, Presbyterian Hosp., 635 W. 165th st., Z32 PLS-OP
Dwyer, James G....375 Park av., Z22 ALR
Ehrlich, Leon Herbert....211 Central Park W., Z24 OP
Eilert, John Harold....391 E. 149th st. Z55 OP
Elwyn, Herman....239 Central Park W. Z24 OP
Epstein, Sidney S....2021 Grand Concourse, Z53 OP
Esterman, Benjamin....983 Park av., Z28 OP
Everett, A. Reginald....11 Wall st., Z5 ALR
Falkson, Kurt F....225 W. 86th st., Z24 ALR
Fernlund, George Byron 8620 115th st., Richmond Hill, Z18 ALR
Fisher, Harry Richard....465 West End Ave. ALR
Fitzgerald, Fred J. C....118 E. 65th, Z21 OP
Flack, J. Vincent Institute of Oph., 635 W. 165th St., Z32 OP
Foster, Harold....92-11 35th av., Jackson Heights ALR
Fowler, Edmund Prince, Jr...180 Ft. Washington av., Z32 ALR
Fowler, Robert H....610 Park av., Z21 ALR
Fowlkes, John Winston....12 E. 63rd, Z21 ALR
Fox, Sidney A....63 E. 75th St. X21 OP
Francis, Charles C....610 West End av., Z24 ALR
Frank, David I....60 Gramercy Park, Z10 ALR
Fread, Bernard....45 E. 85th, Z28 OP
Frey, W. G., Jr....121 E. 60th, Z22 OP
Fried, Joseph John....5 West 86th St., Z24 OP
Friedman, Benjamin....485 Central Park W. OP
Fritz, Milo H....927 Park ave., Z28 OALR
Frothingham, Florie D....133 E. 58th, Z22 ALR
Furman, Martin A....1082 Park ave. Z28 ALR
Garrick, Thomas J....40 E. 61st, Z21 ALR
Gartner, Samuel....521 Park av., Z21 OP
Garvey, Thomas Quincy....115 E. 61st St. ALR
Gatewood, Wm. L....111 E. 61st st., Z21 ALR
Gilmore, Goodlatte Browne..2940 Grand Concourse, Z58 ALR
Givner, Isadore....108 E. 66th, Z21 OP
Glushak, Leopold....1 West 85th, Z24 ALR
Goldman, H. G....57 W. 58th, Z19 ALR
Goldman, Irving Brice....121 E. 60th, Z22 ALR
Goldsmith, Jacob....30 E. 60th st., Z22 OP
Gordon, Dan Morris....525 E. 68th St., Z21 OP

## NEW YORK — Continued

Gould, Nathaniel Jay.....940 Grand Concourse, Z56 OP
Graebner, Herbert
American Airlines, N. Y. M. A., Jackson Heights ALR
Graef, Fred W.....140 East 54th St., Z22 ALR
Grant, Maurice David.....1860 Morris av., Z53 ALR
Greene, James S.....61 Irving place, Z3 ALR
Greenfield, William J.....30 E. 40th av., Z16 ALR
Gresser, Edward Bellamy.....39 E. 75th, Z21 OP
Griesman, Bruno L.....3 East 66 st., Z21 ALR
Grimaldi, Americo John.....100 W. 59th, Z19 OP
Grove, Russell Clark.....60 E. 58th, Z22 ALR
Gurtov, John Jacob.....1285 Grand Concourse, Z52 ALR
Guy, Loren P.....40 E. 62nd, Z21 OP
Hanley, J. Swift.....40 W. 55th, Z19 ALR
Hardy, LeGrand H.....23 E. 79th, Z21 OP
Hartshorne, Isaac.....30 W. 59th, Z19 OP
Haskin, W. H.....375 Park av., Z22 ALR
Hersh, Joseph H.....57 W. 57th st., Z19 ALR
Herzig, Arthur J.....667 Madison av., Z21 ALR
Hetrick, J. A. Werner.....667 Madison av., Z21 ALR
Hetrick, L. E.....40 E. 61st, Z21 ALR
Hipp, Walter.....140 E. 54th St. OP
Holden, William H.....755 Park av. ALR
Hory, Joseph S.....2488 Grand Concourse, Z58 OP
Houlahan, J. M.....653 Park av., Z21 OP
Hunt, Westley Marshall.....907 5th av., Z21 ALR
Hynes, Frank Joseph.....121 E. 60th, Z22 ALR
Ide, David.....1749 Grand Concourse, Z53 ALR
Imperatori, C. J.....108 E. 38th, Z16 ALR
Ingalls, Raymond G., The Institute of Oph.,
Presbyterian Hospital, 635 W. 165th st. OP
Jacobs, S. M.....15 West 81st st., Z24 OALR
Jaffe, Morris.....940 Grand Concourse, Z56 OP
Jessel, Erwin.....1160 Fifth Ave., Z29 OP
Johnson, Thomas Holland.....30 W. 59th, Z19 OP
Jones, David H.....140 E. 54th, Z22 LR
Jones, Marvin F.....121 E. 60th, Z22 ALR
Judd, H. B.....40 E. 61st, Z21 ALR
Kallen, Leo A.....667 Madison Ave., Z21 ALR
Katzin, Herbert Maurice.....1148 Fifth Ave., Z28 OP
Keil, Frank C.....660 Madison av., Z21 OALR
Kelley, Samuel F.....47 E. 61st st. ALR
Kelly, Joseph Dominic.....140 E. 54th, Z22 OALR-PI S
Kend, Lee.....133 E. 58th, Z22 ALR
Kilmann, Martin H.....118 E. 54th St., Z22 OP
Kirby, D. B. .....780 Park av., Z21 OP-PIS
Kirkbright, James C. .....40 E. 61st, Z21 ALR
Kleinfeld, Louis.....100 E. 74th st., Z21 ALR
Knapp, Arnold.....10 E. 54th, Z22 OP
Knapp, Arthur Alexander.....112 E. 73rd St. OP
Knighton, W. S.....121 E. 61st, Z21 OP
Knopf, Saul.....30 E. 40th st., Z16 ALR
Koch, Ferdinand L. Philip.....60 E. 75th st., Z21 OP
Kopetzky, S. J., Col. (M.C.).....71 E. 80th st., Z21 ALR
Kornberg, Maurice.....235 E. 22nd st., Z10 ALR
Kornzweig, Abraham L.....1200 Fifth av., Z29 OP
Kronenberg, Bernard.....737 Park av., Z21 OP
Krug, Ernest F.....988 Fifth av., Z21 OP
LaGattuta, Frank.....2488 Grand Concourse, Bronx 58 OP
Last, Murray A.....1095 Park av., Z28 OP
Laval, Joseph.....136 E. 64th, Z21 OP
La Vigne, A. A.....667 Madison av., Z21 OALR
Lempert, Julius.....119 E. 74th st., Z21 ALR
Levbarg, John J.....176 W. 87 St. Z24 ALR
Limbach, Earl Frederick..30 W. 59th, Station "G", Z19 ALR
Lisman, Jack Victor.....654 Madison Ave. OP
Loewe, Walter R.....1114 Madison av., Z28 OP
Lore, John M.....325 West End av., Z23 ALR
Lubart, Joseph.....310 West 86th st. ALR
Macdonald, James O.....67 E. 59th st., Z22 ALR
MacDonald, Philip G.....115 E. 61st st.,Z21 ALR
Macnie, John P.....2501 Palisade av., Z63 OP
Macpherson, Duncan.....920 Fifth av., Z21 ALR
Maliniac, Jacques W.....106 E. 78th, Z21 ALR-PI S
Martin, Ashby G.....895 Lexington av., Z21 ALR
Masor, Philip L.....306 E. 15th St. Z3 OP
Matusow, Abraham P.....1100 Grand Concourse, Z56 ALR
Maybaum, J. L.....1070 Park av., Z28 ALR
McAuliffe, G. W.....630 Park av., Z21 ALR-PI S
McClanahan, Robert.....60 E. 75th, Z21 OP
McCollom, Robert L.....115 E. 61st st. ALR
McCuaig, Duncan Roderick.....40 E. 61st st. ALR
McCuaig, Victor C.....40 E. 61st, Z21 ALR
McDannald, C. E.....100 W. 59th, Z19 OP
McGrath, James B.....2615 Grand Concourse, Z58 ALR
McLean, John M.....525 E. 68th, Z21 OP
McNally, John Joseph.....121 E. 60th St. ALR
Meek, Raymond E.....729 Park Ave., Z21 OP
Miller, Harold Rexford.....114 E. 54th St., Z22 OP
Miller, Isidore.....2021 Grand Concourse, Z53 ALR
Miller, Joseph William.....242 E. 19th, Z3 ALR
Minsky, Henry.....2 E. 95th, Z28 OP
Mirow, David.....237 E. 20th, Z3 ALR
Mittendorf, A. D.....399 Park av., Z22 OP
Moore, James A.....530 E. 90th st., Z28 ALR
Morrison, W. Wallace.....140 E. 54th st., Z22 ALR
Moshinsky, Alexander L.....237 E. 20th, Z3 ALR
Neer, Edmonde DeWitte.....115 E. 61st, Z21 ALR
Neivert, Harry.....555 Park Ave., Z21 ALR
Neuberger, Julius Frederick.....325 E. 57th st., Z22 ALR
Neuschatz, Gerald.....327 Central Park West, Z25 ALR
Newman, Saul C.....1082 Park Ave., Z28 ALR
Nisselson, Max.....250 Riverside drive, Z25 ALR
Northcott, Thomas A.....40 E. 61st, Z21 OP
O'Kane, George H.....55 E. 86th St., Z28 ALR
Opin, Edward.....2065 Grand Concourse, Z53 ALR
Page, John R.....127 E. 62nd, Z21 ALR
Palmer, Arthur.....667 Madison av., Z21 ALR-PI S
Paton, R. T.....927 Park av., Z28 OP
Payne, Brittain F.....17 E. 72nd st., Z21 OP
Payne, Frank.....635 W. 165th st., Z32 OP
Pearlman, Louis M.....53 E. 93rd, Z28 ALR
Perera, Charles A.....70 E. 66th, Z21 OP
Perkins, Osborn P.....121 E. 60th, Z22 OP
Petix, Dominick Gilbert....2615 Grand Concourse, Bronx OP
Pfeiffer, Raymond L.....635 W. 165th, Z32 OP
Pitman, Louis K.....1749 Grand Concourse, Z53 ALR
Posner, Adolph.....667 Madison av., Z21 OP
Pullen, Edward Markey.....30 E. 40th, Z16 ALR
Reese, Algernon B.....73 E. 71st, Z21 OP
Risch, Otto C.....104 E. 40th, Z16 ALR
Rolett, D. M.....876 Park av., Z21 OP
Rosen, David M.....19 W. 55 st., Z19 ALR
Rosen, Sam.....101 E. 73rd, Z21 ALR
Rosenbluff, Michael.....225 W. 106th, Z25 ALR
Rosenwasser, Harry.....101 E. 73rd st., Z21 ALR
Ross, John.....6 E. 70th, Z21 ALR
Rothstein, David Louis.....57 W. 57th St., Z19 ALR

## NEW YORK — Continued

Ryan, Maxwell D.....660 Madison av., Z21 ALR
Sackin, David..."St. Moritz," 50 Central Park South, Z19 ALR
Saltzman, Samuel L.....334 W. 86th, Z24 OP
Samuels, Bernard.....57 W. 57th, Z19 OP
Sauer, John J.....30 E. 40 st., Z16 OP
Saunders, Truman L.....103 E. 65th, Z21 ALR
Scal, Joseph Coleman.....10 E. 96th, Z28 ALR
Schachat, Walter S.....30 E. 60th st., Z22 OP
Schattner, Alfred ..... 115 E. 61st., Z21 ALR
Schechter, Solomon P.....1749 Grand Concourse, Z53 ALR
Scheer, Henry M.....522 West End av., Z24 ALR
Schlivek, Kaufman.....1016 Fifth av., Z28 OP
Schmidt, Otto V. M.....568 Park av., Z21 ALR
Schugt, Henry P.....122 E. 76th, Z21 ALR
Schutz, Sigmund.....105 W. 55th st. OP
Schwartz, A. A.....7 W. 71st, Z23 ALR
Schwartz, Leo.....136 E. 57th, Z22 ALR
Seeley, Robert Charles.....133 E. 58th St. ALR
Seelig, Charles Arthur.....104 E. 40th St., Z16 ALR
Seward, J. A.....115 E. 61st, Z21 ALR
Sitchevska, Olga L.....30 Fifth av., Z11 OP
Smith, Clarence Henry.....140 E. 54th, Z22 ALR
Smith, James Morrisset.....123 E. 53rd, Z22 ALR
Smith, James W.....1016 Fifth av., Z28 OP
Snyder, Eugene R.....7 W. 96th st. ALR
Sparer, William S.....50 E. 10th, Z3 ALR
Stark, Jessie B.....46 Park av., Z16 OP
Steinbach, Louis.....1882 Grand Concourse, Z57 OP
Steinberg, Martin R.....226 W. 70th st., Z23 ALR
Stovin, Joseph S.....151 Central Park West, Z23 ALR
Straatsma, Clarence R.....66 E. 79th, Z 21 ALR-PLS
Street, Alvin Murray.....745 5th av., Z22 ALR
Strickler, Joseph George.....200 W. 57th, Z19 ALR
Sturges, Leigh F.....121 E. 60th, Z22 ALR
Surber, John Roger.....119 E. 74th St., Z21 ALR
Swick, David A.....30 W. 70th, Z23 OP
Syracuse, Victor R.....29 Washington Sq. W., Z11 OALR
Theodore, Frederick H.....667 Madison Ave., Z21 OP
Tickle, Thomas G.....30 E. 40th st., Z16 ALR
Town, Arno E.....140 E. 54th st. OP
Troncoso, Manuel U.....500 West End av., Z24 OP
Turner, Oliver W.....155 E. 34th st., Z16 OALR
Tusak, E. A.....115 E. 90th, Z28 OP
Vogel, Felix P. S.....1749 Grand Concourse, Z53 ALR
Von Sallmann, Ludwig J. K.....635 W. 165th St., Z32 ALR
Voorhees, Darrell Graham.....135 E. 65th St. ALR
Watnick, Joseph Louis.....10 W. 96th, Z25 ALR
Webster, David H.....140 E. 54th, Z22 OP
Weiss, Leon M.....30 East 60th St., Z22 OP
Wexler, David.....103 E. 86th, Z28 OP
Weymuller, Ernest A.....69 E. 76th St., Z21 ALR
Wheeler, Maynard C.....30 W. 59th, Z19 OP
White, Francis W.....471 Park av., Z22 ALR
Wiener, Alfred.....550 Park av., Z21 OALR
Wincor, Henry G.....1882 Grand Concourse, Z57 OP
Witchell, Ira S.....2021 Grand Concourse, Z53 ALR
Woodman, DeGraaf.....156 E. 37th, Z16 ALR
Zuckerman, Joshua.....875 Park av., Z21 OP

### NIAGARA FALLS

Goetzman, Arthur C.....308 United Office bldg. OP
Price, Norman W. (Retired)
Hyde Park Blvd. & Lewiston Rd., Route No. 47 OALR
Rooker, A. M.....United bldg. ALR
Walker, Oliver Frank.....2873 Lewiston road OALR

### NORWICH

Prindle, Ronald Edon.....20 Cortland St. (Home) ALR

### NYACK

Theis, Franklyn B.....209 S. Broadway OALR

### OGDENSBURG

Baker, Lauchlin J.....310 Franklin St. ALR

### OLEAN

Moore, Ellis Columbus.....Nat'l Bank bldg. ALR
Mountain, Edward R.....128 S. Union st. ALR

### ONEONTA

Hall, Edwin P.....Box 212 ALR

### OZONE PARK

Buchbinder, Murray W.....94-12 109th Ave. ALR

### POUGHKEEPSIE

Bauer, Charles E.....11 Cannon St. OP
Miller, Sidney N.....113 Hooker av. ALR
Ostrow, Irving.....41 Hooker av. ALR
Voorhees, Earle W.....8 Garfield place ALR

### QUEENS VILLAGE, L. I.

Pauley, Gerald E.....9460-220 St. ALR

### ROCHESTER

Avery, Elroy J.....11 N. Goodman, Z7 ALR
Barber, F.....75 S. Fitzhugh, Z8 OP
Clark, L. H.....337 Monroe av., Z7 OALR
Elliott, Raymond E.....2150 St. Paul st., Z5 ALR
Fischel, Abraham.....922 Sibley Tower OP
Frazer, John Paul.....224 Aberdeen St., Z11 ALR
Gipner, J. F.....Medical Arts bldg., Z7 OP
Gorin, Meyer.....35 Chestnut st., Z4 ALR
Hert, Cecil B.....29 N. Goodman st. ALR
Hilleman, George A.....267 Alexander, Z7 ALR
Honiss, John Ralph.....277 Alexander, Z7 ALR
Jones, Leonard W.....16 N. Goodman, Z7 OALR
Kennedy, Eldred Weston....Gas and Electric bldg., Z4 OP
Lerner, Macy Levi.....332 Park av., Z7 OP
Lowy, Karl.....187 S. Goodman st., Z7 ALR
McDowell, N. D.....275 Alexander, Z7 OALR
Morris, A. G.....Medical Arts bldg., Z7 ALR
Nacey, Lawrence J.....221 Alexander ALR
Nash, C. Stewart.....277 Alexander, Z7 ALR
O'Brien, Elmer W.....5 N. Goodman, Z7 ALR
Pearson, Ovid .....277 Alexander st. OALR
Presberg, Max Harold.....35 Chestnut St., Z4 OP
Shapero, I. M.....289 Alexander, Z7 OALR
Snell, Albert C.....53 S. Fitzhugh, Z4 OP
Snell, Albert Conrad, Jr.....53 S. Fitzhugh St., Z4 OP
Sullivan, Charles T.....277 Alexander, Z7 OP
Trainor, George M.....277 Alexander st., Z7 ALR

### ROCKVILLE CENTER, L. I.

Donlon, John J.....141 Hempstead av. ALR
Goldman, A. Milton.....1 N. Village av. OP
Preefer, Charles Joseph.....195 Hempstead av. OP
Steinberg, Nathan.....253 Sunrise Highway ALR

### ROME

Valone, Frank H.....107 W. Court ALR

## NEW YORK — Continued

**RYE**
- Carroll, Frank D. ..... Blind Brook Lodge OP
- Peterson, N. Vern ..... Highland Hall ALR

**ST. ALBANS, L. I.**
- Berkley, William Leneave Capt. (MC) USN U. S. Naval Hospital OP
- Brown, C. J., Capt. (MC) USN .... U. S. Naval Hospital ALR

**SARANAC LAKE**
- Beck, Robert J. ..... 19 Hope St. OP
- Wilson, George E. ..... 108 Main ALR

**SARATOGA SPRINGS**
- Goodfellow, Thomas J. ..... Arcade bldg. OALR

**SCHENECTADY**
- Furlong, Frank C. ..... 713 Union st. ALR
- Gulick, John Duncan ..... 704 Union, Z5 ALR
- Livshin, Norman ..... 631 University bldg. ALR
- McGraw, James Lorenz ..... 619 University bldg. OP
- Ostrom, William C. ..... 1350 Union, Z8 OP
- Penta, Arthur Quito ..... 1301 Union St. ALR

**SKANEATELES**
- Gerstley, Manfred J. ..... 83 E. Genesee St. ALR

**STATEN ISLAND**
- Griffey, Walter P. ..... U. S. Marine Hospital OP
- Kahn, K. M. ..... 204 Pape bldg. ALR
- Odom, Stanley G., Col. (MC) Staten Island Area Station Hospital, Z6 OALR
- Seligman, Albert E. ..... 41 Forest av., Z1 ALR

**SYRACUSE**
- Blaisdell, Irl Hazard ..... Medical Arts bldg., Z2 ALR
- Brown, Mortimer G. ..... State Tower bldg., Z2 ALR
- Brust, H. O. ..... 820 S. Crouse av., Z1 ALR
- Cahill, James F. ..... 428 S. Salina, Z2 OP
- Cave, Linus S. ..... State Tower bldg., Z2 ALR
- Dickinson, William G. ..... State Tower bldg., Z2 OP
- Doust, Alfred W. ..... 965 Lancaster ave. ALR
- Gillette, D. F. ..... State Tower bldg., Z2 OP
- Hoople, Gordon D. ..... Medical Arts bldg. ALR
- Joy, Harold Henry ..... State Tower bldg., Z2 OP
- Katz, I. H. ..... 713 E. Genesee st. OP
- Kauffman, A. B. ..... Chimes bldg., Z2 ALR
- Kline, H. G. ..... State Tower bldg., Z2 ALR
- Moore, Donald E. ..... 713 Genesee st., Z2 OP
- Moore, Roy S. ..... State Tower bldg., Z2 ALR
- Rubenstein, A. H. ..... Medical Arts bldg., Z2 ALR
- Webster, F. R. ..... State Tower bldg., Z2 OP

**TARRYTOWN**
- Newlands, W. Alexander ..... 19 S. Broadway ALR

**TROY**
- Sulzman, Frank Malcolm ..... 1831 Fifth av. OALR
- Sulzman, John H. ..... 1831 Fifth av. OALR

**UTICA**
- Beattie, William ..... 5 Derbyshire Place, Z3 OALR
- Farrell, James I. ..... 250 Genesee, Z2 OP
- Girard, Paul H. ..... 258 Genesee st. OP
- Gruppe, Karl ..... 258 Genesee st., Z2 ALR
- Jones, Fred G. ..... 258 Genesee, Z2 ALR

**WARSAW**
- Nairn, George Waverly Wyoming County Community Hospital OALR

**WATERTOWN**
- Atkinson, John H. ..... 168-70 Sterling ALR
- Atkinson, Walter S. ..... 129 Clinton OP
- Henderson, L. E. ..... 129 Clinton OP
- McAskill, James E. ..... Woolworth bldg. ALR

**WELLSVILLE**
- Glosser, Herbert H. ..... 47 N. Broad OP

**WHITE PLAINS**
- Emery, John R. ..... Medical Centre ALR
- Knight, Granville F. ..... Medical Centre bldg. ALR
- Matusow, Meyer ..... 6 Old Mamaroneck rd. OP
- Moriarty, Richard W. ..... 8 Mamaroneck road OALR
- Wood, Edward Christopher ..... 175 Main St. OP

**WOODHAVEN, L. I.**
- Frothingham, Edward ..... 8626 94th st., Z21 OALR
- Stack, James Edward ..... 8838 Woodhaven blvd., Z21 ALR

**YONKERS**
- Bullwinkel, H. Griffin, Capt. (M.C.), USNR, Glenwood Gardens, Z2 ALR
- Fitzgerald, Wm. W. ..... 27 Ludlow OP
- Petti, George H. ..... 4 Berkeley av., Z5 ALR
- Woodrow, Jack H. ..... 27 Ludlow st., Z5 ALR

## NORTH CAROLINA

**ASHEVILLE**
- Briggs, Henry Harrison, Jr. ..... 611 City bldg. OP
- Chapman, E. J. ..... 331 Haywood bldg. ALR
- McGowan, Joseph Francis ..... New Medical bldg. ALR
- Swann, Cecil C. ..... Flat Iron bldg. ALR
- Weizenblatt, Sprinza ..... 709 New Medical bldg. OP

**CHARLOTTE**
- Foster, Clarence Batchelder ..... 520 Professional bldg. OP
- Hart, Verling Kersey ..... 108 W. 7th St. ALR
- Johnston, James G. ..... 1520 E. 4th, Z4 OP
- Motley, Fred E. ..... 106 W. 7th, Z2 ALR
- Neblett, Herbert Clarence ..... 401 N. Tryon, Z2 OP
- Peeler, C. N. ..... 106 W. 7th, Z2 OALR
- Sloan, Henry L. ..... 106 W. 7th, Z2 OP
- Smith, Franklin C. ..... 2219 Radcliff av., Z4 OP
- Whisnant, A. M. ..... RFD No. 2, Park rd. OALR

**DURHAM**
- Anderson, William Banks ..... Duke Hospital OALR
- Arnold, Ralph A. ..... Duke Hospital ALR
- Eagle, Watt Weems ..... Duke Hospital ALR
- Ferguson, George Burton ..... 1110 West Main ALR
- Kerns, Thomas C. ..... 1110 W. Main ALR
- McPherson, Samuel D. ..... McPherson Hospital ALR

**ELIZABETH CITY**
- Shipley, J. L., Capt. (MC) USN (Retired) 309 W. Church st. OALR

**GOLDSBORO**
- Bizzell, Marcus Edward ..... 139 W. Walnut St. ALR
- Peele, James Clarendon ..... Box 636 ALR

**GREENSBORO**
- Farmer, William Dempsey ..... 119 North Elm St. ALR
- Strickland, Horace G. ..... Jefferson bldg. OP
- Taylor, Shahane R. ..... Jefferson bldg. ALR
- Wolfe, Hugh C. ..... 102 N. Elm ALR

## NORTH CAROLINA — Continued

HENDERSON
White, Clarence Hunt.....Box 257 ALR

HIGH POINT
Leath, Maclean B.....511 N. Main st. ALR

KINSTON
Peery, Vance Price .....Kinston Clinic ALR

LEXINGTON
Leonard, Jacob C., Jr.....3½ E. First st. ALR

MORGANTON
Billings, Gilbert M.....110 S. Sterling ALR

RALEIGH
Hicks, Vonnie Monroe.....Professional bldg. OP
Powers, Frank P.....Professional bldg. ALR

SALISBURY
McCutchan, Frank.....100 W. Innes ALR

WINSTON-SALEM
Bradford, George Edwin.....Nissen bldg. ALR
Combs, Fielding.....Nissen bldg., Z3 ALR
Davis, Thomas W.....O'Hanlon bldg., Z3 OALR
Harrill, James Albert.....Bowman Gray School of Med. ALR
Speas, William Paul.....Reynolds bldg., Z3 OP

## NORTH DAKOTA

BISMARCK
Buckingham, Tracy W.....405 Broadway ALR
Constans, George Maurice.....Clinic bldg. OP
Diven, Wilbur Lester.....406½ Main ALR
Rosenberger, Henry P.....221 Fifth ALR

FARGO
Foster, George C.....Fargo Clinic OALR
Hawn, Hugh W.....807 Broadway, Fargo Clinic OP
Larson, G. Arthur.....608 Black bldg. OALR
Rindlaub, Elizabeth P.....321 Eighth South OALR
Rindlaub, John H.....321 Eighth South OALR

GRAND FORKS
Alger, L. James.....Box 328 OP
Jensen, August F.....1119 Cottonwood st. OP
Youngs, Nelson A.....Grand Forks Clinic ALR

MINOT
Combs, Arnold B.....Northwest Clinic ALR
Lampert, Max T.....First Nat'l bldg. ALR
McCannel, Archie D.
Corner 1st st., S. W. and Central av. OALR

## OHIO

AKRON
Arbuckle, David S.....Akron Savings & Loan bldg., Z8 OALR
Brumbaugh, John D.....75 North Wheaton road, Z13 OALR
Damitz, John Cyril.....2nd National Bank bldg. OALR
Fowler, Floyd J.....Akron Clinic, 513 W. Market st., Z3 ALR
Mathias, Daniel F.
819 First Central Tower bldg., Z8 ALR
Parry, Thayer L.....328 Ohio bldg., Z8 OALR
Rubin, Herman G.....159 S. Main st. ALR
Springer, James E.....409 Peoples Bank bldg., Z8 ALR

ALLIANCE
King, George L.....537 E. Market St. OP

ASHLAND
Shilling, Raymond L.....408 Center st. ALR

ASHTABULA
O'Connor, Timothy Francis.....4634 Main av. ALR
Streicher, Carl J.....217 Park Place OP

BELLEFONTAINE
Harbert, John P.....135 N. Main OALR

CANTON
Brandon, James R.....800 First Nat'l Bank bldg. ALR
Hoffmann, John N.....Brant bldg., Z2 ALR
King, Clair B.....409 3rd St., N.W., Z2 OP
Paradis, Charles Ovide.....625 Cleveland Ave., N.W. ALR
Rosedale, Raymond Samuel....610 N. Market av., Z2 ALR
Schirack, Clarence J.....817 Cleveland av. N. W., Z3 ALR
Toot, Joseph F.....First Nat'l Bank bldg., Z2 OP
Underwood, John H.....Brant bldg., Z2 ALR
Weaver, Homer V.....625 Cleveland av. N. W., Z3 ALR

CHILLICOTHE
Quinn, Robert E.....126 W. Main St. OP
Shepard, Emerson P.....128 W. Main st. ALR

CINCINNNATI
Abrahamson, Ira A.....925 Union Trust bldg., Z2 OP
Asbury, Mary K.....3549 Holly Lane, Z8 OP
Beyer, A. G.....Carew Tower, Z2 OALR
Brown, Albert Louis.....Carew Tower, Z2 OP
Brumm, Lewis Patrick.....726 Carew Tower OP
Craddock, Wm. H.....703 Carew Tower, Z2 ALR
Finney, Nancy E.....2453 Gilbert av., Z6 OP
Fischbach, Victor W.....Union Central bldg., Z2 ALR
Goodyear, H. M.....Doctors bldg., Z2 ALR
Haggart, Harry H.....920 Carew Tower, Z2 ALR
Hans, C. L.....Provident Bank bldg., Z2 OP
Heidelman, George.....Union Central bldg., Z2 OP
Hendricks, Louis J.....Carew Tower, Z2 OP
Hofling, Charles A.....19 W. 8th st., Z2 OP
Hofmann, Albert P.....3149 Vine st., Z19 ALR
Howard, Robert E.....Union Central bldg., Z2 ALR
Hyman, Morris.....305 Doctors bldg., Z2 ALR
Levin, Abram Ascher.....Carew Tower, Z2 OP
Lowenthal, Gerson.....277 Doctors' bldg., Z2 ALR
Lyle, Donald J.....Carew Tower, Z2 OP
Miller, Ralph H.,
6257 Rogers Park Place, Pleasant Ridge, Z13 OP
Mithoefer, W.....Carew Tower, Z2 ALR
Nantz, Frank A.....225 Bodmann ave. OP
Osherwitz, Morris Stanford.....456 Doctors bldg., Z2 OP
Portnoy, Samuel H.....19 Garfield Place, Z2 ALR
Ray, Victor, Jr.....Carew Tower, Z2 OP
Reid, H. W.....Union Central bldg., Z2 OP
Riesenbeck, Leo Henry.....831 Carew Tower ALR
Ross, Ervin Scott.....477 Riddle Rd. ALR
Safer, Louis A.....Provident Bank bldg., Z2 ALR
Sakler, Barnet R.....Doctors bldg., Z2 OP
Schrimpf, Cyril Edward.....229 Doctors' bldg., OP

## OHIO — Continued

**Seitz, Samuel**.....Doctors bldg., Z2 **ALR**
**Siegel, Francis X**.....Union Central bldg., Z2 **OALR**
**Stanbery, Henry**.....Doctors bldg., Z2 **OP**
**Stitt, H. L**.....Doctors bldg., Z2 **ALR**
**Stoll, Karl Ludwig**.....19 W. 7th, Z2 **OP**
**Swing, Frank U**.....Doctors bldg., Z2 **ALR**
Thompson, E. H.....19 W. 7th, Z2 **OALR**
**Thornell, William Clyde**.....1002 Carew Tower **ALR**
Woodward, Paul Maxwell.....**4140 Hamilton av., Z23 ALR**

### CLEVELAND

Adelstein, Fred.....Rose bldg., Z15 **ALR**
Alexander, Fred Wm.....10515 Carnegie ave., Z6 **ALR**
Allen, Donald Gray.....1021 Prospect av., Z15 **OP**
Berns, Charles.....11408 Whitmore av., Z8 **ALR**
Brandwan, Samuel R.....Rose bldg., Z15 **OP**
Bruner, Abram Breneman.....Guardian bldg., Z14 **OP**
Bruner, William E.....Guardian bldg., Z14 **OP**
Bucher, Walter M.....4187 Pearl road, Z9 **ALR**
Burke, T. A.....Rose bldg., Z15 **OALR**
**Chamberlain, Webb Parks**.....7405 Detroit av., Z2 **OP**
Chamberlain, Webb, Jr.....7405 Detroit av., Z2 **OP**
Collins, Arthur D.....1007 Guardian bldg., Z14 **OP**
Collins, James T.....Republic bldg., Z15 **ALR**
Deeton, William L.....**13125** Shaker square, Z20 **ALR**
Dixon, Fred Willis.....Rose bldg., Z15 **ALR**
Engler, Clarence William.....2323 Prospect av., Z15 **ALR**
Ersay, Emil Francis.....1030 Euclid Av., Z14 **ALR**
Forsythe, S. T.....Rose bldg., Z15 **OALR**
Freeman, Marvin S.....409 Osborn bldg., Z15 **ALR**
Gans, Jerome A.....922 Keith bldg. **OP**
Garrett, Ernest W.....Hanna bldg., Z15 **ALR**
Hamilton, Howard Eugene.....10515 Carnegie av., Z6 **ALR**
Hannon, R. Emmet.....364 Hanna bldg., Z15 **OP**
Harris, Harold Earl.....Cleveland Clinic, Z6 **ALR**
Harvey, Elmer Frank.....1422 Euclid Ave. **OP**
Jackson, Clarence C. R.....2065 Adelbert rd., Z6 **ALR**
Jacoby, Mark W.....Hanna bldg., Z15 **OP**
Johnson, Lorand V.....10515 Carnegie, Z6 **OP**
Kennedy, Roscoe J.
2020 E. 93rd st., c/o Cleveland Clinic, Z6 **OP**
Keyes, John E. L.
22199 McCauley rd., Shaker Heights Z22 **OP**
Kinney, Charles Ed.....10515 Carnegie av., Z6 **ALR**
Kogut, Lewis V.....5466 Broadway Ave., Z4 **OP**
Lamb, Andrew J.....10006 Carnegie Ave. **ALR**
Large, S. H.....170 Eddy rd. **ALR**
Lemel, Samuel L.....1030 Euclid av., Z15 **ALR**
Loeb, Wm. J.....1125 Schofield bldg., Z15 **ALR**
Marks, Moses I.....10515 Carnegie Ave., Z6 **ALR**
Master, John R.....1058 Rose bldg. **ALR**
McCall, J. W.....610 Rose bldg., Z15 **ALR**
McDonald, C. L.....Rose bldg., Z15 **OALR**
Metz, Roy B.....1422 Euclid av., Z15 **OP**
Missal, Sylvester Clemens.....2323 Prospect Ave., Z15 **ALR**
Mitchell, Horace E.....14805 Detroit av., Z7 **ALR**
Monson, S. H.....B. F. Keith bldg., Z15 **OALR**
Moore, Paul G.....Republic bldg., Z15 **OP**
Moore, Paul M., Jr.....Cleveland Clinic, Z6 **ALR**
Motto, M. Paul.....Rose bldg., Z15 **OP**
Mussun, W. G.....Rose bldg., Z15 **ALR**
Nelson, Charles F.....Schofield bldg., Z15 **OALR**
Pitkin, C. E.....Carnegie Medical bldg., Z6 **ALR**
Pitkin, York Nones.....10515 Carnegie Ave. **ALR**
Quittner, S. S.....10515 Carnegie av., Z6 **OALR**
Rieger, W. H.....Hanna bldg., Z15 **ALR**
Rosenberger, Harry Clifford.....10515 Carnegie av., Z6 **ALR**
Ruedemann, Albert D.....Cleveland Clinic, Z6 **OP**
Ryall, George W.
17010 Kinsman road, Shaker Heights, Z20 **ALR**
Schwarz, Gerald T.....10515 Carnegie av., Z6 **OP**
Shiras, Howard H.....13302 Cormere av., Z20 **OP**
Somberg, Joseph S.....406 Bulkley, Z15 **OP**
Stoffregen, Charles Henry.....10515 Carnegie av., Z6 **ALR**
Stolzar, Irwin H.....831 Guardian bldg. **OP**
Stotter, Arthur L.....1148 Euclid av., Z15 **OALR**
Thoburn, Thomas W.....1020 Huron road., Z15 **ALR**
Thomas, Charles I.....10515 Carnegie av., Z6 **OP**
Tuckerman, W. H.....1020 Prospect av., Z15 **OALR**
Weidlein, Ivan F.....10515 Carnegie av., Z6 **ALR**
Wolpaw, Benjamin J.....2323 Prospect av. **OP**

### COLUMBUS

Beatty, H. G.....**150 E.** Broad, Z15 **ALR-PI S**
Brown, Harold Ethan.....581 Bulen Ave., Z9 **OP**
Brown, John E.....135 Preston rd., Z9 **OALR**
Clark, I. G.....188 E. State, Z15 **OALR**
Culler, Arthur M.....150 E. Broad st., Z15 **OP**
Davies, Morgan C.....269 E. State, Z15 **OP**
Davis, William C.....150 E. Broad, Z15 **OP**
Emswiler, Herbert D.....370 E. Town, Z15 **ALR**
Harris, Edward W.....240 East State st., Z15 **ALR**
Hauer, A. M.....350 E. State, Z15 **ALR**
Johnson, David M.....327 E. State St. **ALR**
Means, R. G.....40 S. Third, Z15 **ALR**
Perry, Claude S.....40 S. 3rd st. **OP**
Postle, C. D.....240 E. State, Z15 **ALR**
Prout, A. W.....380 E. Town, Z15 **OALR**
Sage, Harry M.....2194 N. Parkway Dr., Z8 **OP**
Sanor, Daniel G., Jr.....206 E. State, Z15 **ALR**
Silbernagel, C. E.....247 E. State, Z15 **OALR**
Stoutenborough, William A.....**21 E. State, Z15 OALR**
Thomas, Francis W.....**327** E. State, Z15 **OALR**
Troutman, Erwin W.....21 E. State, Z15 **ALR**

### CUYAHOGA FALLS

Stahl, Richard Holmes.....143 Portage Trail **ALR**

### DAYTON

Beavis, James O.....**900 Fidelity Medical bldg., Z2 ALR**
Binkley, Roy S.....Fidelity bldg., Z2 **OALR**
Boswell, Robert Echo.....Fidelity bldg., Z2 **ALR**
Clark, Willard C.....333 Rockwood Ave. **OP**
Dutrow, H. V.....426 Kramer rd. **OALR**
Harris, H. B.....Fidelity Medical bldg., Z2 **OALR**
Insel, Herman Herbert....770 Fidelity Medical Bldg. Z2 **OALR**
Kilbourne, P. H.....Fidelity Medical bldg., Z2 **OALR**
Thomas, Edward Robert.....60 Wyoming, Z9 **OP**
Webb, Walter Whayne.....3rd National bldg. **ALR**

### ELYRIA

Meredith, Lawrence Cornelius.....124 Middle av. **ALR**

### FINDLAY

Mowry, Ben.....First Nat'l Bank bldg. **ALR**

### FREMONT

Riddell, Malcolm M.....112½ Front **ALR**

## OHIO — Continued

**GALLIPOLIS**
Martin, N. A.....Gallipolis Clinic ALR

**GREENVILLE**
Shroder, Luther Null.....144 W. 4th St. ALR

**HAMILTON**
Chamberlin, Clyde.....300 Rentschler bldg. ALR

**LAKEWOOD**
Ellenberger, Carl.....14805 Detroit av., Z7 OP
Frackelton, Ralph James.....15701 Detroit av. ALR
McGannon, Francis Lambert.....14900 Detroit av. ALR
Merica, F. W.....14805 Detroit av., Z7 ALR
Miller, G. Leslie.....14805 Detroit av., Z7 OP
Sacheroff, Louis.....1288 Hird st., Z7 ALR

**LIMA**
Herr, Albert H.....129 N. Elizabeth st. ALR
Kniseily, Alan DeG.....722 Nat. Bank bldg. ALR
Page, Robert O.....722 Nat'l Bank bldg. ALR
Stueber, F. G.....209 W. North st. OALR
Stueber, Paul J.....209 W. North OALR

**LORAIN**
Monosmith, O. B.....O. P. S. bldg. OALR
Smith, Thomas Leonard.....800 Broadway ALR

**MANSFIELD**
Brown, Wilbur Max.....49 W. 2nd St. OP
Garber, J. M.....14 N. Mulberry OALR
Oakes, Milton C.....24 N. Mulberry st. OP
Smedal, Erling A.....456 W. Park av. OP
Wise, Ralph C.....News bldg. OALR

**MARIETTA**
Sauer, W. W.....231 Third OALR

**MARION**
Brickley, Daniel W.....Uhler-Phillips bldg. OALR
Brickley, Daniel W., Jr.....401 Uhler Phillips bldg. ALR

**MASSILLON**
Dowling, James Reginald.....508 Ohio Merchants bldg. ALR
Stewart, James V.....1st Nat'l Bank bldg. ALR

**MIDDLETOWN**
Williams, Walton H.....Castell bldg. OALR

**MONROEVILLE**
Bolman, Harold R.....6½ S. Main OP

**MT. VERNON**
Pumphrey, Gordon H.....100 N. Maine st. OP

**NEWARK**
Steele, Wendell M.....17 E. Locust st. OP

**OBERLIN**
Stephan, Lewis B.....207 E. College OP

**PAINESVILLE**
Hedlund, G. O.....616 Mentor av. ALR

**SALEM**
King, Lowell W.....470 E. State ALR

**SANDUSKY**
Kramer, Julius C.....409 Columbus av. ALR

**SHAKER HEIGHTS**
Phillips, Josephine D.....16130 Parkland drive, Z20 OP

**SIDNEY**
Alvis, Raymond W.....Ohio bldg. OALR

**SPRINGFIELD**
Burgman, James E.....8 W. Main ALR
Easton, J. C.....First Nat'l Bank bldg. OALR
Ernsting, Harry Carl.....8 W. Main st. OP
Hogue, D. W.....34 W. High OALR
Reuter, Carl H.....First Nat'l Bank bldg. OP
Yinger, Starling C.....First Nat'l Bank bldg. ALR

**STEUBENVILLE**
Quinn, John L.....401 Market ALR
Weinman, Edward B.....401 Market St. OP

**TOLEDO**
Alderdyce, William W.....513 Madison av., Z4 OALR
Boice, Ralph E.....316 Michigan, Z2 OP
Cooper, James H.....316 Michigan st., Z2 OP
Denyes, Gerald Foster.....316 Michigan, Z2 ALR
Gunderson, Harvey Clifford....2001 Collingwood Ave., Z2 ALR
King, Charles R.....Spitzer bldg., Z4 OALR
Lukens, Charles.....316 Michigan Z2 OALR
Lukens, John A.....316 Michigan, Z2 ALR
Minns, John Edward.....316 Michigan, Z2 ALR
Nippe, Leonard.....316 Michigan, Z2 OALR
Patterson, Sam H.....125-15th st., Z2 OP
Ravin, Louis C.....316 Michigan st., Z2 OP
Roberts, John L.....316 Michigan st. OP
Skow, John D.....2001 Collingwood av., Z2 OP
Steinfeld, A. L.....125 Fifteenth, Z2 OALR
Winger, Ira B.....709 Madison av., Z2 OALR

**UNIVERSITY HEIGHTS**
Weinberg, Herman Charles.....2101 S. Taylor Rd., Z18 OP

**VAN WERT**
Edwards, Gwilym Austin.....Home Guards bldg. ALR
Edwards, Stephen A.....Home Guards bldg. ALR

**WARREN**
Beale, Allen W.....308 N. Park av. ALR
Noonan, Paul Ecret.....238 N. Park ave. ALR
Stimson, George W.....580 Perkins drive N. W. OALR

**WILLOUGHBY**
Waugh, J. M.....Ridge road, R. F. D. No. 1 OALR

**XENIA**
Ray, Harold E.....Allen bldg. OP

**YOUNGSTOWN**
Evans, Wm. H.....510 Dollar Bank bldg., Z3 ALR
Goldcamp, Edward C.....Dollar Bank bldg., Z3 ALR
Goldcamp, John S., 810 Dollar Sav. & Trust Co. bldg., Z3 OP
Goodwin, Vernon L.....509 Central Tower, Z3 ALR
Hall, Raymond A.....514 Home Savings & Loan bldg. ALR
Myers, Stanley A.....810 Dollar Bank bldg. ALR
Odom, Robert E.....378 Lora av., Z4 ALR
Piercy, Frederick Franklin
613 Home Savings & Loan bldg., Z3 ALR
Piercy, Robert Lee.....613 Home Savings & Loan bldg. ALR
Walker, Orville J.....Home Savings & Loan bldg., Z3 ALR
Wenaas, Elmer J.....24 Wick av. OP

**ZANESVILLE**
Culbertson, L. R.....Masonic Temple OALR

## OKLAHOMA

ADA
Peterson, William G.....106 E. 13th ALR

BARTLESVILLE
Rucker, Ralph Weller.....Union National Bank bldg. ALR

MUSKOGEE
Coachman, Edwin H.....Manhattan bldg. ALR

OKLAHOMA CITY
Bonham, William L.....Medical Arts bldg., Z2 ALR
Cooper, F. Maxey.....Medical Arts bldg., Z2 OP
Coston, Tullos O.....Medical Arts bldg., Z2 OP
Emenhiser, Lee K.....Medical Arts bldg. ALR
Ford, Harry C.....1014 Medical Arts bldg., Z2 ALR
Macdonald, J. C.....301 N. W. 12th, Z3 ALR
McHenry, L. Chester.....Medical Arts bldg., Z2 ALR
Randel, Harvey O.....Medical Arts bldg., Z2 OP
Robertson, E. Norris, Jr.....301 Northwest 12th OP
Royer, Charles A.....717 Medical Arts bldg., Z2 OP
Wails, Theodore G.....Medical Arts bldg., Z2 ALR
Watson, O. Alton.....1200 N. Walker, Z3 ALR

OKMULGEE
Tracewell, George L.....300 Commerce bldg. ALR
Tulloch, George M.....410 McCulloch bldg. ALR

TULSA
Cook, W. Albert.....Medical Arts bldg., Z3 OALR
Crane, Donald Van Denburgh....Medical Arts bldg., Z3 OP
Davis, Arthur Henry.....Medical Arts bldg., Z3 ALR
Edwards, David Lloyd.....203 Stanolind bldg., Z3 OP
Haralson, Charles Herndon.....Medical Arts bldg., Z3 OP
Huber, Walter A.....Medical Arts bldg., Z3 OP
Mishler, D. L.....604 S. Cincinnati av., Z3 ALR
Smith, Ruric N.....Medical Arts bldg., Z3 ALR
Stuard, Charles G.....310 Medical Arts bldg., Z3 OP

## OREGON

ASTORIA
Spalding, Lansford M.....185 Fourteenth ALR

BAKER
Gordon, Glenn G.....Alexander bldg. ALR

BEND
Evers, Lorance Bixler.....1036 Wall st. ALR

EUGENE
Norton, Arthur H. .....1162 Willamette OP
Shepard, Willis B.....Miner bldg. ALR

LA GRANDE
Bouvy, L. B.....Foley bldg. OALR

MEDFORD
Young, Clarence M. .....P. O. Box 1506 ALR

PORTLAND
Adix, H. Victor, Jr.....425 Medical Dental bldg., Z5 ALR
Bailey, Paul.....1515 S. W. Myrtle st., Z1 ALR
Bolton, Wilbur Minnis.....1020 S.W. Taylor St., Z1 ALR
Bouvy, Harry M., 800 Pittock blk., 10th & Washington OALR
Boyden, Guy L.....1735 N. Wheeler, Z12 ALR
Browning, Andrew J.....Mayer bldg., Z5 OP

## OREGON—Continued

Carruth, Howard Ernest.....Medical Arts bldg., Z5 OALR
Davis, Ralph F.....Medical Arts bldg., Z5 OALR
Dykman, Augustus Bartram.....833 S. W. 11th, Z5 OP
Fenton, Ralph A.....Medical Arts bldg., Z5 OALR
Gaston, Ira E. .....Medical-Dental bldg., Z5 OALR
Henton, George E.....812 S. W. Washington, Z5 OP
Henton, George H.....812 S. W. Washington st., Z5 OP
Jones, Lester T.....1020 S. W. Taylor st., Z5 OALR
Jordan, Lewis W. .....Medical Arts bldg., Z5 ALR
Karkeet, Robert B.....833 S. W. 11th av., Z5 ALR
Kiehle, Frederick A.....1020 Southwest Taylor, Z5 OP
Kistner, F. B.....1216 S.W. Yamhill st. ALR
Kuhn, Clifford W.....9545 S.W. 62nd Dr. ALR
Larsell, Olaf., Ph.D., Sc.D.,.....University of Oregon
Lucas, Harold R.....Medical-Dental bldg., Z5 OALR
Lupton, Irving Martin.....Medical Arts bldg., Z5 OALR
Neely, Paul T.....Medical Arts bldg., Z5 ALR
Petheram, C. C.....Medical Arts bldg., Z5 ALR
Pulliam, Robert Lee, Jr.
Bodine-Contril Clinic, 1635 N. Wheeler ave., Z12 ALR
Simons, Max.....2580 N. W. Mildred st., Z10 ALR
Swan, Kenneth C., Dep't of Oph.,
University of Oregon Med. Sch., Marquam Hill rd., OP
Taylor, E. Merle.....Medical Arts bldg., Z5 OP
Ten Eyck, Thomas G.....808 N. E. 39th st., Z13 ALR
U'Ren, Harold Merle.....1735 N. Wheeler ave., Z12 OP
Weeks, John E. (Retired).....360 Edgecliff road, Z1 OP

SALEM
Baum, William W.....206 Guardian bldg. OALR
Clement, L. O.....Guardian bldg. OALR

THE DALLES
Scovis, I. John.....U. S. National Bank bldg. OP

WECOMA
Cutler, Franklin E.....ALR

## PENNSYLVANIA

ALIQUIPPA
Thel, Henry C.....387 Franklin ALR

ALLENTOWN
Hertz, William J.....125 N. 8th ALR
Phillips, Samuel A.....137 N. 8th OP
Rothenberger, Marvin K.....206 N. 8th st. ALR
Shoemaker, Robert Eugene.....1248 Hamilton St. OP
Uhler, S. Mann.....104 N. 8th ALR

ALTOONA
Buzzard, Josiah F.....1110 Thirteenth av. OALR
Glover, Lewis P.....1200 Fourteenth av. OP

ARDMORE
Bostwick, D. S.....106 Linwood av. ALR
Furlong, Thomas F., Jr.....Times Medical bldg. ALR
Longaker, Edwin P.....41 E. Montgomery av. ALR

BEAVER
Crumrine, Norman Ross.....595 Third OALR

BEAVER FALLS
Baldwin, Thomas M.....222 Brodhead bldg. ALR
Reed, Elmer McCready.....2835 4th ave. ALR

## PENNSYLVANIA — Continued

**BETHLEHEM**
Chase, W. D. ..... 230 E. Broad ALR
James, James E. ..... 128 W. Fourth st. ALR

**BLOOMSBURG**
Moser, G. P. ..... 455 Market st. ALR

**BRYN MAWR**
McAndrews, Leo F. ..... Mermont-Plaza OP
McGavic, John S. ..... 132 Morris ave. OP
Scarlett, Hunter W. ..... 825 Montgomery av. OP
Snyder, George Gordon ..... Mermont Apts ALR

**BUTLER**
Diess, Robert G. ..... 307 B.C.N.B. bldg. OP
Goehring, Donald Everett ..... Butler S. & T. bldg. ALR
Hunt, Guy Anderson ..... R.D. 5 OP

**CARBONDALE**
Marmelstein, Maurice ..... 1st Nat'l Bank bldg. OP

**CARLISLE**
George, Forney P. ..... 118 S. Hanover st. ALR
Stoner, Donald D. ..... 156 W. Pomfret st. ALR

**CHAMBERSBURG**
Himelfarb, H. M. ..... 180 E. Queen st. ALR

**CHARLEROI**
Rowland, Ivo E. ..... 5th and McKean ALR

**CHESTER**
Cross, G. H. ..... 525 Welsh OP
Hayes, Merrill B. ..... 710 Madison st. ALR
Lynch, Joseph S. ..... 302 E. 9th St. OP

**CLEARFIELD**
Shaffer, Harry Gard ..... 225 Cherry ALR

**CONNELLSVILLE**
Harrison, Fred H. ..... Second Nat'l Bank bldg. OALR
McLellan, Thomas G. ..... Second Nat'l Bank bldg. OP

**CORAOPOLIS**
Moon, Seymour B. ..... R.D. 2 OP

**DANVILLE**
Davison, Francis Wells ..... Geisinger Hospital ALR
Jacobs, Clyde Hillock ..... Geisinger Memorial Hosp. OP
Nebinger, Reid ..... 19 W. Market ALR

**DREXEL HILL**
Weber, H. C. ..... 1112 Mason Ave. OALR

**DU BOIS**
Beals, C. Wearne ..... 28 W Scribner av. ALR
Tyson, John McClure ..... 3 S. Brady ALR

**EASTON**
Bloom, C. Hugh ..... 65 N. Third ALR
Urban, Joseph T. ..... 204 Bushkill St. ALR

**ERIE**
Delaney, James H. ..... 138 W. 9th OP
Donovan, John E. ..... 265 W. 10th st. ALR
Fust, John H. ..... 231 W. 7th OALR
Jackson, J. D. ..... 232 W. 8th OALR
Russell, J. A. Merle ..... 813 Sassafras OALR
Simmons, J. Thomas ..... 316 W. 10th st. OP

**FRANKLIN**
Nordstrom, Chester Albert ..... Exchange Bank bldg. ALR

**GREENSBURG**
Fairing, John W. ..... 122 N. Main OALR

**GREENVILLE**
Jamison, Edgar L. ..... 19 Louisa av. ALR

**HANOVER**
Wetzel, Roy J. ..... 301 Broadway ALR

**HARRISBURG**
Benson, Kenneth H. ..... 1114 N. 2nd St. ALR
Dailey, Gilbert L. ..... 618 N. 3rd OALR
Gracey, George F. ..... 209 State OALR
Phillips, Warren Charles ..... 801 N. Second OP
Smith, Jay D. ..... 813 N. 2nd st. ALR
Weinstein, Lee ..... 1104 N. 2nd St. ALR
Zimmerman, J. Landis ..... 207 State OP

**HAVERTON**
Garlichs, Richard Walton ..... 216 North Manoa Rd. ALR

**HAZLETON**
Reiche, O. C. ..... Markle Bank bldg. OP

**JENKINTOWN**
Cornelius, C. E. ..... 119 Summit av. ALR
Follweiler, Frank L. ..... 423 York rd. ALR
Kauffman, M. Luther ..... Medical Arts bldg. OP

**JOHNSTOWN**
Cohen, Jerome H. ..... 604 Johnstown Trust bldg. OP
Flora, William Kenneth ..... 406 Main st. ALR
Griffith, Harold M. ..... Johnstown Bank & Trust bldg. OP
Harris, C. M. ..... Johnstown Bank & Trust bldg. OALR
Hays, Charles E. ..... U. S. Bank bldg. ALR
McCloskey, Paul ..... 338 Locust ALR
Meyers, Paul T. ..... 1109 U.S. Bank bldg. ALR
Parker, Ray ..... U. S. Bank bldg. ALR

**LANCASTER**
Fulton, Harry C. ..... 339 N. Duke OP
Hutchison, Lloyd S. ..... 45 E. Orange St. ALR
LeFevre, Willis H. ..... 284 N. Duke ALR
Smith, Ernest Gerard ..... 339 N. Duke OP

**LANSDOWNE**
McLaughlin, John J. ..... Owen & Lacrosse aves. ALR

**LATROBE**
Mather, Homer Raymond ..... 224 Depot st. ALR
Mather, Homer Raymond, Jr. ..... 1st Nat'l Bank bldg. OP

**LEBANON**
Diehl, William Harold ..... 701 Chestnut St. ALR
Gardiner, Charles Ernest ..... 701 Chestnut St. ALR
Strickler, Alfred D. ..... 26 N. 9th st. OP

**LEWISTOWN**
Allis, Paul M. ..... 6 N. Brown ALR

**MAHANOY CITY**
Ricchiuti, A. George ..... 316 E. Centre ALR

**MEADVILLE**
Hazen, S. Frank ..... 231 Chestnut ALR

## PENNSYLVANIA — Continued

**MEDIA**
Lewis, Fielding O. (Retired) ALR

**MERION STATION**
Hunt, Wm. T., Jr. 367 Brookway OALR

**MONESSEN**
Gemmill, Walter D. 130 Seventh ALR

**NANTICOKE**
Abbott, Albert J. 100 E. Main St. OP

**McKEESPORT**
Rittenhouse, Emory A. 203 Masonic Temple ALR

**NEW KENSINGTON**
Steim, J. M. First Nat'l Bank bldg. OP

**NORRISTOWN**
Beideman, Joseph Ellsworth. Montgomery Trust Arcade OP
LaClair, Charles Huston, Jr. 1021 W. Main St. ALR
Tait, Edwin Forbes. 1324 W. Main St. OP

**OIL CITY**
Magee, F. Earle. 222 Seneca ALR

**PALMERTON**
Kupp, John H. 582 Delaware av. ALR

**PHILADELPHIA**
Adler, Francis Heed. 313 S. 17th, Z3 OP
Annon, W. T. 235 S. 15th, Z2 ALR
Appleman, L. F. 308 S. 16th, Z2 OP
Atkins, Joseph Preston. 250 S. 18th St. ALR
Auslander, Milton Manuel. 1422 W. Erie av., Z40 ALR
Balentine, P. L. 1524 Chestnut, Z2 OP
Batson, Oscar V. 133 S. 36 st., Z4 ALR
Berner, George Emil. 5430 Greene, Z44 OP
Bertolet, John Allen. 1530 Locust, Z2 ALR
Blair, Mortimer W. N. E. cor. 20th and Chestnut, Z3 OP
Brav, Solomon S. 2027 Spruce St., Z41 OP
Bricker, Sacks. 1101 W. Wyoming av., Z40 ALR
Bridgett, Frank Aloysius 3332 Chestnut st., Z4 ALR
Briglia, Frank J. 819 Wharton, Z47 ALR
Brumm, Seth A. Medical Arts bldg., Z3 OALR
Butt, Miriam M. 333 Seymore st., Z44 OP
Campbell, Edward H.
Spruce st. Medical bldg., 19th & Spruce sts., ALR
Carpenter, Chapin. 1930 Chestnut, Z3 OP
Clerf, Louis Henry. 1530 Locust, Z2 ALR
Coates, George M. 1721 Pine, Z3 ALR
Cohen, Herman B. 255 S. 17th, Z3 ALR
Cohen, Louis B. 1320 Arrott, Z24 OP
Cohen, Samuel. Spruce St., Medical bldg., Z3 ALR-PIS
Cowan, Alfred. 1930 Chestnut, Z3 OP
Cowan, Thomas H. 1930 Chestnut St. Z3 OP
Creighton, William J. 37 S. 20th, Z3 OP
Davis, T. Carroll. 3128 N. Broad, Z32 ALR
Davis, Warren B. 135 S. 18th, Z3 ALR-PI S
Deichler, L. Waller. 1930 Chestnut, Z3 OALR
Dickson, Robert L. 4803 Penn, Z24 ALR
Dintenfass, Henry. 1305 Spruce, Z7 ALR
Donnelly, Edward J. 1321 Spruce, Z7 OP
Dublin, George Joseph. 1930 Chestnut, Z3 OP
Elfman, Louis Kenneth. 6330 Lebanon av., Z31 ALR
Ellis, Van M. 1528 Spruce st., Z2 OP
Ersner, Matthew S. 1915 Spruce, Z3 ALR
Fewell, A. G. 1924 Pine, Z3 OP
Fox, C. Calvin. 1806 Spruce, Z3 ALR
Fry, W. E. 1930 Chestnut, Z3 OP
Gettes, Bernard Charles. 2028 Pine st. OP
Gibson, Glen G. 255 S. 17th, Z3 OP
Gordon, William. 1721 Pine, Z3 ALR
Gouterman, Joseph I. 1930 Chestnut, Z3 OP
Gowens, Henry L. Jr. 2217 Delancey st., Z3 OP
Harkins, Herbert P. 1930 Chestnut st., Z3 ALR
Hawman, Erle G. 4051 N. Broad, Z40 OP
Heed, C. R. 1205 Spruce, Z7 OP
Hewson, William. 2031 Locust st., Z3 ALR
Hirst, Otto C. 255 S. 17th, Z3 ALR
Hitschler, William Jacob. 5 E. Chestnut av., Z18 ALR
Hoffman, N. Fulmer, Jr. 2126 Spruce st., Z3 ALR
Houser, Karl M. 2035 Delancey st., Z3 ALR
Howland, Alvin W. 255 So. 17th St., Z3 OP
Hunter, Robert John. 2035 Delancey st., Z3 ALR
Husik, D. N. 1820 Spruce, Z3 ALR
Irgens, Edwin R.
Wills Hosp., 1601 Spring GardenSt., Z30 ALR
Ivy, Robert H. 1930 Chestnut, Z3 ALR-PIS
Jackson, Chevalier. 255 S. 17th, Z3 L
Jackson, Chevalier L. 255 S. 17th, Z3 L
Jordan, Harvey Ernest, Jr. 1912 Spruce St., Z3 ALR
Kasper, Kelvin A. 135 S. 18th, Z3 ALR
Katz, Harry Kolman. 1206 Cottman st., Z11 OP
Kaufman, A. Spencer. 255 So. 17th St., Z3 ALR
Kelly, George F. J. 37 S. 20th, Z3 OP
Krall, J. Thomas. 1927 Spruce St., Z3 OP
Krewson, William Egbert, 3rd. 1930 Chestnut, Z3 OP
Lane, Franklin F., Capt. (MC) USN
7430 Boyer St., Mt.Airy OALR
Lawrence, Granville A. Medical Arts bldg., Z3 OP
Lehrfeld, Louis. 1321 Spruce, Z7 OP
Leopold, Irving H. 1930 Chestnut St., Z3 OP
Lieberman, George Emanuel. 1721 Pine St. ALR
Loftus, John Edward. 1930 Chestnut, Z3 ALR
Lukens, Robert McD
Suite 1001, Medical Arts bldg., 16th and Walnut, Z3 ALR
Luongo, R. A. 2054 Locust, Z3 ALR
Marden, Philip Ayer. 1912 Spruce St., Z3 ALR
McCarthy, C. T. 1810 Pine, Z3 ALR
McDonald, P. Robb. 255 S. 17th St. OP
Miller, M. Valentine, 114 W Phil-Ellena (Mt. Airy), Z19 ALR
Mitchell, E. K. 710 W. Lehigh av., Z33 ALR
Mullen, Carroll R. 2025 Locust, Z3 OP
Myers, David. 1834 Pine St. ALR
Norris, Charles Morgan. 3401 N. Broad St., Z40 ALR
O'Keefe, John Joseph. 255 S. 17th St., Z3 ALR
Olsho, S. L. 235 S. 15th, Z2 OALR
Ornston, Darius G. 36 Carpenter Lane, Z19 ALR
Persky, Abram H. 1737 Chestnut, Z3 ALR
Pryor, Charles A. 255 S. 17th st., Z3 ALR
Putney, Floyd Johnson. 255 S. 17th St., Z3 ALR
Reese, Warren S. 1901 Walnut, Z3 OP
Richardson, George A. 1532 Overington, Z24 ALR
Ridpath, Robert F. 1737 Chestnut, Z3 LR
Rommel, John C. 4601 N. Broad, Z40 OP
Ronis, B. J. 7141 Elmwood av. ALR
Rudolphy, Jay Besson. 230 S. 21st, Z3 OP
Scheie, Harold G. 313 S. 17th st., Z3 OP
Schenck, Harry P. 1912 Spruce st., Z3 ALR
Seltzer, Albert Pincus. 1332 N. Franklin st., Z22 ALR
Shannon, C. E. G. 1930 Chestnut, Z3 OP
Shuster, Benjamin Harrison. 1824 Pine, Z3 ALR

## PENNSYLVANIA — Continued

Silcox, Louis Edward.....255 S. 17th St. Z3 ALR
Simkins, James J.....1737 Chestnut, Z3 ALR
Skillern, S. R., Jr.....1734 Pine, Z3 ALR
Sloane, Henry O.....1717 Pine, Z3 OP
Smith, Austin T.....1830 Spruce st., Z3 ALR
Spaeth, Edmund B.....1930 Chestnut, Z3 OP-PI S
Spencer, W. H.....1830 S. Rittenhouse sq., Z3 ALR
Stamm, Carl.....1737 Chestnut St., Z3 ALR
Stockwell, Eunice LeBaron.....2018 Pine st., Z3 OP
Sutherland, William S.....1930 Chestnut, Z3 OP
Swalm, Charles Joseph.....1330 W. Rockland, Z41 ALR
Tassman, Isaac S.....Medical Arts bldg., Z2 OP
Towson, Charles E.....Germantown Prof. bldg., Z44 ALR
Trueman, Robert H.....600 N. 16th st., Z30 OP
Tucker, Gabriel.....250 S. 18th, Z3 LB
Wagers, Arthur J.....1429 Spruce, Z2 ALR
Waldman, Joseph.....216 E. Gorgas Lane, Z19 OP
Walsh, Thomas J.....2059 Parrish, Z30 ALR
Washick, Frank.....501 Cottman st., Z11 ALR
Weinstein, M. A.....1530 Locust, Z2 ALR
Weiss, Sidney.....1930 Chestnut st., Z3 OP
Whelan, G. L.....2100 Walnut, Z3 ALR
Whelan, William F.....2004 Walnut, Z3 ALR
Williams, Horace J.....5908 Greene (Germantown), Z44 ALR
Winston, Julius.....1737 Chestnut st., Z3 ALR
Wolf, Lewis R.....3474 Frankford av., Z34 OP
Zacks, Myron Albert.....1737 Chestnut, Z3 ALR
Zentmayer, William.....1930 Chestnut, Z3 OP

### PHOENIXVILLE

Sherman, Arthur E. Maj. (MC) .Valley Forge Gen. Hosp. PLS OP

### PITTSBURGH

Bernatz, C. F.....Park bldg., Z22 OP
Billings, Robert J.....500 Penn av., Z22 OP
Blair, W. W.....Woodland rd., Z6 OP
Boaz, Emmett D.....3710 Fifth av., Z13 ALR
Conway, John M.....Jenkins Arcade, Z22 ALR
Cooper, Roy C.....6929 Yorkshire rd., Z8 ALR
Curry, Glendon E.....1204 Pitt Bank bldg., Z22 OP
Day, Kenneth M.....121 University pl., Z13 ALR
De Stio, Daniel Stephen.....Highland bldg., Z6 ALR
Dunbar, John Charles.....Empire bldg., Z22 OP
Evans, Samuel D...1502 Park bldg., 355 Fifth av., Z22 OP
Fischer, N. Arthur.....Union Trust bldg., Z19 ALR
Friedman, Louis Leonard.....6078 Jenkins Arcade, Z22 ALR
Geeseman, George R.....634 Washington rd., Z16 OP
Goldberg, Sol.....8082 Jenkins Arcade Z22 OP
Hamilton, Richard G.....Medical Arts bldg., Z13 ALR
Hampsey, Joseph William.....806 May bldg., Z22 ALR
Hinderer, Kenneth H.....402 Medical Arts bldg., Z13 ALR
Jennings, Charles W.....121 University pl., Z13 OP
Jordan, Raymond Ellsworth.....121 University Pl., Z13 ALR
Krebs, Adolph.....500 Penn av., Z22 OP
Kreiger, Alexander A.....4101 Jenkins Arcade Z22 OP
Kutscher, Charles F.....520 Medical Arts bldg., Z13 OP
Lanson, F. Arthur.....646 Washington rd., Z16 ALR
Larimore, Fred Campbell.....500 Penn av., Z22 OP
Linhart, William Oran.....521 Medical Arts bldg. OP
Linn, J. G.....Jenkins Arcade, Z22 OP
MacLachlan, A. A.....May bldg., Z22 ALR
MacMillan, Karl D.....1206 Clark bldg., Z22 OP
Markel, J. Clyde.....806 Pitt Bank bldg., Z22 OP
Marshall, Watson.....121 University pl., Z13 ALR
McCaslin, Murray F.....Union Trust bldg., Z19 OP
McCollough, T. B.....121 University pl., Z13 ALR
McCready, J. Homer.....121 University pl., Z13 ALR
Miller, A. Boyd, 1006 Prof. bldg., 429 Penn av., Z22 ALR
Morgan, Irving Joseph.....Jenkins Arcade bldg., Z22 OP
O'Donnell, Merrill Charles.....725 Jenkins bldg., Z22 ALR
Pachtman, Isadore.....Jenkins Arcade bldg., Z22 OP
Patterson, Ellen J.....(Retired), 504 Neville, Z13 ALR
Perrone, Joseph Addison.....Mercy Hospital, Z19 ALR
Plumer, John S.....121 University pl., Z13 OP
Ratner, Simon Harry.....3401 Fifth av., Z13 ALR
Reed, Charles L.....Jenkins Arcade, Z22 OP
Rhodes, David Henry.....Clark bldg., Z22 OP
Rimer, Frank H.....Jenkins Arcade, Z22 ALR
Rohm, Jack Zeth.....7002 Jenkins Arcade ALR
Rohm, Robert Franklin.....7002 Jenkins Arcade, Z22 OP
Ruehl, William W.....7106 Jenkins Arcade, Z22 ALR
Seip, Walter R.....Professional bldg. OP
Shaffer, D. Howard.....7036 Jenkins Arcade, Z22 OP
Shepard, Warren Brundage.....Jenkins Arcade, Z22 ALR
Shuman, G. H.....351 Fifth av., Z22 OP
Simpson, John Reid.....Medical Arts bldg., Z13 ALR
Statti, Louis Wm.....Jenkins Arcade, Z22 OP
Taylor, M. C.....3000 Knolson av., Z26 OP
Thorpe, H. E.....Jenkins Arcade, Z22 OP
Todd, Grover Cleveland.....Jenkins Arcade, Z22 ALR
Turner, H. H.....Clark bldg., Z22 OP
Weiss, Louis.....7085 Jenkins Arcade ALR
Weisser, Chas. Wm.....806 May bldg., Z22 OP
Weisser, E. A.....May bldg., Z22 OP
Wible, LeRoy E.....3227 5th Ave. ALR

### PITTSTON

Gagion, Thomas Reed.....23 Broad OP
Gallizzi, Vincent D.....307 Dime Bank bldg. ALR

### POTTSTOWN

Hancox, Cecil William.....Bahr Arcade bldg. ALR

### POTTSVILLE

Bacon, Lewis H.....300 Mahantongo St. OP
Cress, William J.....318 W. Market ALR
Mengel, Sterling F.....612 W. Market St. OP

### READING

Bankes, Claude W.....212 N. 6th OP
Craig, Paul C.....232 N. 5th OP
High, Isaac B.....326 N. 5th ALR
Landis, James E.....232 N. 6th ALR
Penta, John J.....237 S. 5th st. ALR
Penta, Michael J.....312 N. 5th ALR
Quereau, James V.....206 N. 5th OP
Rhode, Homer Jonas.....220 N. 6th st. OP
Strause, Harold L.....826 N. 5th st. ALR
Wiest, Philip R.....1036 Franklin st. ALR
Wotring, John M.....220 N. 6th OP

### ROCHESTER

Miksch, Henry Fred.....209 S. Park ALR

### SCRANTON

Bishop, Frederick J.....Medical Arts bldg., Z3 ALR
Corser, J. B.....415 Mulberry, Z3 OP
Curtin, Eugene A.....506 Spruce, Z3 OALR
Guzek, Joseph T.....616 Connell bldg., Z3 OP
Jordan, James Shannon.....Medical Arts bldg., Z3 OP
Williams, M. M.....Medical Arts bldg., Z3 OP

## PENNSYLVANIA — Continued

SHARON HILL
Tomlinson, J. Wesley....900 Chester Pike OP

SHENANDOAH
Monahan, James J....31 S. Jordin ALR

SUMMIT HILL
Bonner, William Richard....3 W. White st. OP

TAMAQUA
Houser, Ben P....458 E. Broad St. ALR

TURTLE CREEK
Hunter, James F....503 Penn av. ALR

UNIONTOWN
Staman, Harry H....49 W. Church St. ALR

UPPER DARBY
Kennedy, Patrick James....6910 Market OP
Moore, George H....101 Long Lane OALR
Rankin, Charles A....Ludlow and Heather road OP

WARREN
Anderson, Edwin R....220 Liberty OALR

WASHINGTON
McCullough, Clarence Joseph....Washington Trust bldg. OALR
McKennan, James W....Washington Trust bldg. OP
McMurray, J. B....Washington Trust bldg. OALR
McMurray, John S....6 S. Main St. ALR

WELLSBORO
Laird, Archibald....12 Main st. OP

WILKES-BARRE
Buckman, Lewis T....83 S. Franklin OALR
Buckman, Samuel T....70 S. Franklin OALR
Connole, Joseph V....Miners Nat'l Bank bldg. OP
Dattner, Herman B....1000 Miners Nat'l Bank bldg. ALR
Marsden, W. C....99 N. Franklin OALR
Schappert, N. Louis....59 S. Washington OALR

WILLIAMSPORT
Decker, Philip Harold....25 W. 3rd st., Z5 ALR

WYOMISSING
Perfect, Fred Rolleston....1258 Penn. Ave. ALR

YORK
Fackler, C. L....Country Club rd. OALR
Fisher, Lawrence Christian....469 W. Market OP
Grove, Bruce Alexander....426 W. Market st. OP
Podboy, August J....28 N. Queen St. ALR

## RHODE ISLAND

BARRINGTON
Stephens, Henry F., Lt. Comdr. (M.C.), 295 Rumstick rd. OP

PROVIDENCE
Adams, Frank M....122 Waterman, Z6 ALR
Bernasconi, Ezio Joseph....716 River Ave. OP
Bolotow, Nathan A....126 Waterman, Z6 ALR
Dimmitt, Frank W....78 Waterman, Z6 OALR
Grossman, Herman P....210 Angell, Z6 OP
Happ, Linley C....170 Waterman st., Z6 ALR
Hanson, Frederick Charles....162 Angell St. OP
Harvey, N. Darrell....112 Waterman, Z6 OALR
MacCardell, Frank Cameron....193 Waterman, Z6 ALR
Messinger, H. C....210 Angell, Z6 OP
Muncy, William M....162 Angell, Z6 OALR
Porter, L. B....199 Thayer, Z6 OALR
Sannella, Lee George....124 Waterman St., Z6 OP
Sharp, Benjamin S....339 Thayer St., Z6 OALR
Winkler, Herman A....224 Thayer, Z6 ALR

## SOUTH CAROLINA

ANDERSON
Gaines, Thomas R. ....Professional bldg., OP

CHARLESTON
Best, L. Kent....107 G. Ashley ave., Z16 OP
Evatt, Clay W....91 Rutledge ave., Z16 ALR
Hanckel, Richard White, Jr....96-A Bull st., Z16 ALR
Hope, Robert Meek....108 Rutledge av., Z16 ALR
Price, F. Raymond....118 Rutledge Ave., Z16 OP
Smith, Josiah Edward....60 Meeting, Z1 OALR
Teasdale, Laurie Redmond....311 Professional bldg. ALR
Townsend, John Ferrars....85 Wentworth, Z6 OALR

COLUMBIA
Asbill, David S....1512 Marion st., Z49 OP
Bristow, Walter J....1517 Hampton, Z29 OALR
Lowestein, Harold H....Vet. Adm. Hospital OP
Workman, Joseph B. Jr....1515 Bull St. OP

FLORENCE
Mobley, Marion R. ....125 W. Cheves st., ALR

GREENVILLE
Harper, DeWitt Lawrence....111 East North st. ALR
Jervey, James W., Jr....101 Church St. OALR
Sanders, John Levis....222 N. Main ALR

ORANGEBURG
Gressette, James H....Box 2467, Z10 ALR

ROCK HILL
Macdonald, Roderick....330 E. Main ALR

SPARTANBURG
Anderson, Ruskin G....753 N. Church st. OP
Watkins, John O....314 Montgomery bldg. OP

SUMTER
Walker, R. M....115 N. Sumter St. ALR

## SOUTH DAKOTA

ABERDEEN
Bunker, Paul Gordon....Newberry bldg. ALR
Rudolph, Edward A....624 Citizens bldg. OP

BROOKINGS
Kershner, C. M....Watson Clinic OP

MITCHELL
Mabee, Oscar Judson....Medical Arts bldg. ALR

## SOUTH DAKOTA — Continued

PIERRE
Robbins, Clarence Edward.....111 S. Huron av. ALR

RAPID CITY
Newby, Harley D.....619½ Main st. ALR
Spain, Marceda L.....629½ Main st. ALR

SIOUX FALLS
Gregg, John B.....Security Nat'l Bank bldg. OALR
Keller, S. A.....303 S. Minnesota av. OALR

VERMILLION
Jordan, George T.....215 Court st. OALR

WATERTOWN
Crawford, James H.....7 Way-Penney bldg. OP

YANKTON
Hohf, Julius A.....Clinic bldg. OALR

## TENNESSEE

CHATTANOOGA
Benz, Alvin H.....Medical Arts bldg. OP
Hogshead, J. M.....602 Georgia av., Z3 OALR
Isbell, D.....552 McCallie av., Z3 ALR
Lassiter, Chester L.....Hamilton Nat'l Bank bldg., Z2 ALR
Lawwill, Stewart.....Medical Arts bldg., Z3 OALR
Long, Samuel Herman.....Medical Arts bldg., Z3 ALR
Steele, Willard.....721 Broad, Z2 OALR

CLARKSVILLE
Wilson, Paul Edwin.....408 Franklin OP

CLEVELAND
Ferguson, E. R.....404 Inman st. ALR

DYERSBURG
Brewer, John D.....Baird Brewer General Hospital OALR

JOHNSON CITY
Jones, U. G.....Cor. Market and Boone ALR
McKee, Thomas P.....207 E. Watauga av. OALR
Wilson, John McCullough.....207 Watauga Ave. OP

KINGSPORT
Hodge, James Vernon.....105 Market ALR
Vermillion, James S.....233 E. Sullivan st. ALR

KNOXVILLE
Christenberry, Henry Edward Jr.....501 W. Church St. ALR
Cunningham, Henry Kelso.....Medical Arts bldg., Z01 ALR
Irwin, Willard J.....Medical Arts bldg. ALR
Kincaid, J. H.....421 W. Church av., Z42 OALR
Leach, Robert S.....Medical Arts bldg., Z2 OP
Le Tellier, Forrest S.....Doctors bldg., Z30 OP
Miller, Edwin Ephron.....Medical Arts bldg., Z1 ALR
Montgomery, John L.....Medical Arts bldg., Z01 OP
Potter, William W.....601 Walnut, Z30 OALR

MARYVILLE
Burchfield, George W.....309 Court ALR

MEMPHIS
Anthony, Daniel Harbert.....2968 Iroquois rd. OP
Blassingame, C. B.....899 Madison av., Z3 ALR
Blue, J. B.....62 N. Main, Z3 OALR
Cecha, Albin H., Capt. (MC) USN..U.S. Naval Hosp., Z15 OP
Ellett, E. C.....Exchange bldg., Z3 OP
Evans, John Dorritte.....899 Madison Ave. Z3 ALR
Evans, S. S.....Physicians & Surgeons bldg., Z3 ALR
Harris, J. Harley..Methodist Hospital Doctors bldg., Z4 ALR
Hooker, James F., Capt. (MC) USN..U.S. Naval Hospittal. ALR
King, Charles M., Jr.....899 Madison Ave., Suite 116 OP
Klotz, Wm. F.....1879 Poplar ALR
Levy, Louis.....Commerce Title bldg., Z3 OALR
Lewis, Charles Kelley.....2109 Sterick bldg. ALR
Lewis, Philip Meriwether.....Exchange bldg., Z3 OP
Maiden, S. D.....Kennedy V. A. Hosp., Box 24, Z15 OALR
Manigan, Thomas P.....431 Patterson Z11 ALR
McKinney, James Wesley.....Exchange bldg., Z3 OALR
Minor, H. F.....Commerce Title bldg., Z3 OALR
Qualls, H. W.....1635 Exchange bldg., Z3 ALR
Rychener, Ralph O.....130 Madison av., Z3 OP
Sanders, Sam H.....1089 Madison av., Z3 ALR
Seligstein, Milton B.....Union Planters Bank bldg., Z3 OALR
Shea, J. J.....1018 Madison av., Z3 ALR
Simpson, W. Likely....Physicians & Surgeons bldg., Z3 OALR
Stanford, J. B.....899 Madison av., Z3 OP
Stinson, William David.....899 Madison av., Z3 ALR
Wilson, James Edward.....899 Madison Ave., Z3 OP

NASHVILLE
Bryan, James Leslie.....Bennie-Dillon bldg., Z3 OALR
Cullom, M. M.....Bennie-Dillon bldg., Z3 OALR
Hollabaugh, Charles F.....414 Doctors bldg., Z3 OP
Maness, Guy Milford.....119 7th av. N., Z3 ALR
Orr, Eugene.....Doctors bldg., Z3 ALR
Smith, Henry C.....Doctors bldg., Z3 OP
Sullivan, Robert E.....Doctors bldg., Z3 OP
Warner, Robert Jay.....706 Church, Z3 OP
Wilkerson, William W., Jr.....Bennie-Dillon bldg., Z3 ALR

## TEXAS

ABILENE
Burns, Coleman Craig.....Medical Arts bldg. ALR

AMARILLO
Crume, John James.....724 Polk ALR
Duncan, Frank Benbow.....301 Polk St. OP
Duncan, Robert Austin.....Amarillo bldg. ALR
Streit, August J.....724 Polk ALR

AUSTIN
Hilgartner, Henry L.....202 W. 13th st. OP
Key, Samuel N.....505 Capital Nat'l Bank bldg., Z16 OALR
Key, Samuel Newton, Jr.....120 W. 7th st., Z16 OP
Miears, Claude H.....713 Scarbrough ALR
Robison, J. T.....909 Capital Nat'l Bank bldg. Z16 ALR
Suehs, Oliver W.....120 W. 7th st. ALR
Woodson, B. Palmer.....817 Scarbrough bldg., Z22 OALR

BAYTOWN
Dolph, Chancey H.....317 Main ALR

BEAUMONT
Woodward, John F., Jr.....Goodhue bldg. ALR

BIG SPRING
Malone, P. W.....811 Main ALR
Wood, George Harden.....Petroleum bldg. ALR

## TEXAS — Continued

CORPUS CHRISTI
- Gill, Earl King....416 Chaparral st. ALR
- Kelly, Francis B....519 Medical Professional bldg. OP
- Stroud, Sanders Key....519 Medical Professional bldg. OP

CORSICANA
- Mountjoy, Philip S.... ALR

DALLAS
- Barr, William T....1327 Medical Arts bldg., Z1 ALR
- Brannin, Daniel....Medical Arts bldg., Z1 OP
- Cary, E. H....Medical Arts bldg., Z1 OALR
- Cox, Kelly....Medical Arts bldg., Z1 OP
- Daniel, Ruby K....Medical Arts bldg., Z1 OP
- Darrough, L. E....4105 Live Oak ALR
- Harrington, Silas Frederick....4227 Herschell, Z4 ALR
- Knowles, W. Mood....4227 Herschell, Z4 ALR
- Marchman, Oscar M. ....814 Medical Arts bldg., Z1 ALR
- Marchman, Oscar M., Jr....816 Medical Arts bldg., Z1 OP
- McCullough, Malcolm Kelley....Medical Arts bldg., Z1 OP
- McLaurin, J. G....4710 Munger av., Z4 ALR
- Nelson, Leo A....Box 28, Z1 ALR
- Newton, F. H....Medical Arts bldg., Z1 OALR
- Quinn, Lester Hale ....4105 Live Oak, Z5 OP
- Sellers, L. M....1719 Pacific av., Z1 ALR
- Singleton, John D....3704 Dickason ALR
- Thomas, W. Maxwell....206 Medical Arts bldg., Z1 OP
- Wilkinson, Wallace Bond....521 E. 10th, Z8 ALR

EL PASO
- Ringer, Merritt Gartley, Col. (MC) Wm. Beaumont Gen. Hosp. ALR
- Schuster, Frank P....First Nat'l Bank bldg. OALR
- Schuster, S. A....First Nat'l Bank bldg. OALR
- Spearman, Maurice P....First Nat'l Bank bldg. ALR
- Vandevere, Wm. E....1st Nat'l Bank bldg. OP

FORT WORTH
- Eschenbrenner, John W. Jr....306 W. Broadway, Z4 OALR
- Goodman, Thomas L....1601 Clover Lane, Z7 ALR
- Gough, Roy H....600 W. 10th, Z2 OALR
- Jackson, Atras E....Medical Arts bldg., Z2 OP
- Lees, Charles Ray....806 Medical Arts bldg. OP
- Mitchell, Douglas Gatlin....703 Medical Arts bldg. OP
- Rathgeber, Van D....1305 Medical Arts bldg. OP
- Richardson, James Joseph, Cook Hospital and Beall Clinic, Z3 ALR
- Schenck, C. P....Medical Arts bldg., Z2 OALR

GAINSVILLE
- Mead, Ernest Charles....106½ E. California ALR

GALVESTON
- McReynolds, Geo. S. Jr....Merimas Professional bldg. ALR
- Potter, William Bently....303 Merimas bldg. OP
- Sykes, Clarence Sherbourne....2201 Market OP

GREENVILLE
- Ward, James W....2607 Lee ALR

HOUSTON
- Alexander, Charles S....Med. Arts bldg., Z2 OP
- Barrett, John H. ....1304 Walker av., Z2 ALR
- Bressler, J. L....Medical Arts bldg., Z2 OP
- Cody, Claude C....1304 Walker av., Z2 ALR
- Cody, Claude Carr III....1304 Walker Ave. ALR
- Daily, Louis....Medical Arts bldg., Z2 OALR
- Daily, Ray K....Medical Arts bldg., Z2 OALR
- Dickson, J. Charles....Medical Arts bldg., Z2 ALR
- Elles, Norma B....Niels Esperson bldg., Z2 OALR
- Foster, J. H....1304 Walker av., Z2 ALR
- Goar, Everett Logan....1300 Walker av., Z2 OP
- Griffey, Edward W....Medical Arts bldg., Z2 OALR
- Haden, Henry C.....1914 Travis, Z3 OP
- Harris, Herbert H....1039 Mellie Esperson bldg., Z2 ALR
- Hooker, Lyle....446 Esperson bldg., Z2 OP
- Janse, Hector Mason....3717 Main, Z4 ALR
- Logue, Lyle J....1304 Walker av., Z2 ALR
- Potts, Charles R....1304 Walker av., Z2 OP
- Ralston, Wm. W....1304 Walker av., Z2 OP
- Robison, J. M....1304 Walker av., Z2 ALR
- Royce, Thomas L....904 Holman ALR
- Schultz, Jacob F....1304 Walker av. OP
- Snow, William J....1215 Walker av. ALR
- Spencer, Walter C. E....Medical Arts bldg., Z2 OP
- Stough, John Trew....4501 Fannin St., Z14 OP
- Vanzant, T. J....Medical Arts bldg., Z2 OP

KERRVILLE
- Simpson, Robert K....P.O. Box 789 OP

LONGVIEW
- Hurst, V. Reeves....Hurst Clinic OALR
- Phillips, James Winston....Hurst Clinic ALR
- Roberts, Joseph D., Jr....204 N. Green ALR

LUBBOCK
- Hutchinson, Ben B....Lubbock Memorial Hospital OP
- Malone, F. B....2609 19th ALR
- Thompson, Chase S. The Standefer & Canon Clinic, West Texas Hospital ALR

LUFKIN
- Percy, Archibald Edward....North Raguet & Frank aves. ALR

PAMPA
- High, Clifton E....Combs Worley bldg. OP

PARIS
- Hunt, Thomas Ewell....First Nat'l Bank bldg. ALR

PORT ARTHUR
- Heare, Louis C....Box 312 OP
- Vaughan, Benjamin H....Adams bldg. ALR

SAN ANGELO
- Windham, Robert Edward....234 W. Beauregard blvd. OALR

SAN ANTONIO
- Atkinson, D. T....Medical Arts bldg., Z5 OALR
- Boehs, Charles John....Medical Arts bldg., Z5 ALR
- Brunner, George Harmon....603 Navarro st. OP
- Champion, Albert N....Medical Arts bldg., Z5 ALR
- Dumas, Edward DeVan....Medical Arts bldg., Z5 ALR
- Gill, William Davis....705 E. Houston, Z5 OALR
- Matthews, John L....414 Navarro st., Z5 OP
- Muldoon, Wilfred E....Nix Professional bldg., Z5 OP
- Parrish, Robert E....Medical Arts bldg., Z5 OALR
- Russell, Daniel A....Medical Arts bldg., Z5 ALR
- Sykes, Edwin Meredith....Nix Professional bldg., Z5 ALR

STEPHENVILLE
- Terrell, Vance....Stephenville Hospital ALR

## TEXAS—Continued

TAYLOR
Alexander, Margaret Henrietta.....720 Talbot ALR

TEMPLE
Bonner, Wm. F.....McCloskey V.A.H. OALR
DeBord, Bert Jr.....Scott-WLitee Clinic ALR
McDavitt, Bertha Schenck Stecker ....Main and av. A ALR
McKay, Edward D.....Scott & White Clinic OP
Veirs, E. R.....Scott & White Clinic OALR
Woodson, W. Burbank.....601-22 Professional bldg. OALR

TEXARKANA
Fuller, T. E.....209 Pine OALR
Roberts, A. Warren.....209 Pine OALR

TYLER
Vaughn, E. H.....Peoples Nat'l Bank bldg. OALR

WACO
Aynesworth, H. T.....Box 1191 OALR
Burgess, John L.....904 Medical Arts bldg. ALR
Spencer, Shelby Cruthirds.....Professional bldg. OALR
Woolsey, William Jinkens.....212 N. 7th st. OP

WAXAHACHIE
Donnell, Herbert.....507 W. Main ALR

WHARTON
Black, Vernon Anderson.....105 Hawes St. OP

WICHITA FALLS
Nail, James B.....1300 Eighth ALR

## UTAH

LOGAN
Riter, Kersey C.....The Budge Clinic, 3 N. Main ALR

OGDEN
Beck, Frederick W.....218 First Security Bank bldg. ALR
Hetzel, C. C.....Eccles bldg. OALR
Hetzel, Clarence Charles Jr.....721 Eccles bldg. ALR
Pugmire, Ralph W.....2404 Washington bldg. OP

PROVO
Oaks, Lewis Weston.....33 E. 2nd S. OALR

SALT LAKE CITY
Berman, Harry.....128 Lincoln St., Z2 ALR
Cleary, James A.....804 Boston bldg. ALR
Crandall, Alan Slade.....141 East 2nd South, Z1 OP
Dolowitz, David A.....1152 Glimer Drive, Z5 ALR
Le Compte, E. D.....9 Exchange place, Z1 OALR
Maw, Raymond B.....699 East South Temple, Z2 ALR
Merrill, Rowland Hyde.....1010 First National Bank OP
Muirhead, Robert Mowatt.....8 East Broadway, Z1 ALR
Palmer, Bascom W.....804 Boston bldg., Z1 OP
Phillips, Earl H.....1704 Harrison Ave., Z5 ALR
Rigby, Ralph G.....837 Boston bldg., Z1 ALR
Ruggeri, Charles, Jr.....1120 Boston bldg., Z1 OP
Smith, Homer Edwards.....1005 Medical Arts bldg. OP
Smith, William Leroy.....Medical Arts bldg., Z1 OP
Spear, Dean.....516 Boston bldg., Z1 OP
Stauffer, F. Leaver.....52 East South Temple, Z1 ALR
White, Vivian P.....524 S. 8th East, Z2 ALR

## VERMONT

BARRE
Jarvis, De Forest C.....Quarry Bank bldg. ALR
Kingston, Louis C.....91 Tremont St. ALR

BENNINGTON
Mahoney, Frank A., Jr.....213 Union st. ALR
Ramm, Carl H.....508 Main st. OP

BRATTLEBORO
Noyes, Leighton Helmuth.....67 Main St. ALR

BURLINGTON
Arnold, Francis J.....182 Pearl OALR
Cunningham, John C.....49 S. Winooski ave. OP
Twitchell, Emmus George.....266 Main OALR

MONTPELIER
Bertrand, George Joseph.....73 Main st. OP

RUTLAND
Marshall, George Guerin.....Gryphon bldg. OALR
Seeley, Ralph Hunt, Jr.....79 Center St. ALR

ST. JOHNSBURY
Cramton, Edward Allen.....33 Main OALR

## VIRGINIA

ALEXANDRIA
Nicholson, Charles T.....422 Tyler Place ALR

ARLINGTON
Gilligan, John Henry.....1831 Wilson blvd. ALR
Kulesher, Henry T.....3825 Pershing Dr. ALR

BRISTOL
Harris, Herman Lee.....Reynolds Arcade bldg. ALR

CHARLOTTESVILLE
Burton, Edwin W.....307 E. Market St. OP
Fitz-Hugh, Glassell Slaughter.....104 E. Market st. ALR
Woodward, Fletcher Drummond.....104 E. Market ALR

COVINGTON
Winn, Thomas Meredith.....129½ Maple av. OALR

DANVILLE
Dickerson, Wm. E.....802 Masonic Temple ALR
McGovern, Francis H.....Masonic Temple bldg. ALR

FREDERICKSBURG
Holloway, J. Minor.....1200 Prince Edward ALR

HARRISONBURG
Preston, H. Grant.....Professional bldg. ALR

LYNCHBURG
Gorman, James R.....Allied Arts bldg. ALR
Holland, John Gill.....725 Church ALR
Morrison, Frederick M.....717 Church st. OP

NEWPORT NEWS
Hankins, George Geddy.....Medical Arts bldg. ALR
Phillips, James Wm.....Box 632 OP
Poindexter, Frank Wilmore.....Medical Arts bldg. ALR

## VIRGINIA—Continued

**NORFOLK**

**Cooley, Carl Conrad**.....Medical Arts bldg., Z10 **OALR**
**Diehl, James E**.....813 Wainwright bldg. **ALR**
**Gowen, Thomas F.**, Comdr. (MC) USNR.N.A.S. Dispensary **ALR**
**Kennon, Beverley Randolph III**..908 Med. Arts bldg., Z10 **OP**
**White, J. Warren**.....Medical Arts bldg., Z10 **OALR**

**NORTON**

Henderson, Charles Henry.....800 Park Ave. **OP**

**PORTSMOUTH**

**Hoxie, Derrick**, Comdr. (MC) USN....U.S. Naval Hosp. **ALR**

**PULASKI**

**Smith, Richard O**.....73 Third av. N. W. **ALR**

**RICHMOND**

**Courtney, Robert H**.....Professional bldg., Z19 **OP**
**Gatewood, E. T**.....Professional bldg., Z19 **ALR**
Guerry, DuPont III.....503 Professional bldg. **OP**
**Hughes, T. E**.....1000 W. Grace, Z20 **ALR**
**Pastore, Peter Nicholas**.....5503 Riverside Drive, Z24 **ALR**
Perkins, Edward Williamson.....312 Medical Arts bldg. **OP**
Rein, Walter John.....501 E. Franklin St., Z19 **OP**
**Sheppard, Louis B**.....Suite 301, Medical Arts bldg. **OP**
Smith, Mason.....1100 W. Franklin St., Z20 **OP**
**Smith, Maynard P**.....206 Professional bldg., Z19 **ALR**
**Wallerstein, Emanuel U**.....Professional bldg., Z19 **ALR**
**Wellford, B. R**.....510 Medical Arts bldg., Z19 **ALR**

**ROANOKE**

**Gill, E. G**.....711 S. Jefferson, Z10 **ALR**
**Hamlin, Fred Eugene**.....P.O. Box 2276 **ALR**
**Hatcher, Wm. F**.....Medical Arts bldg., Z11 **OP**
**Stone, Harry B**.....Medical Arts bldg., Z11 **ALR**
**Stone, Harry B., Jr**.....811 Medical Arts bldg., Z11 **ALR**
**Williams, Mortimer H**.....711 Medical Arts bldg. **ALR**
**Young, Charles A**.....409 Medical Arts bldg. **OP**

**STAUNTON**

**Fisher, Guy Rothwell**.....1 E. Beverley st. **ALR**

**WINCHESTER**

**Hawthorne, Allen T**.....110 N. Braddock **ALR**
**McGuire, H. H**.....105 N. Braddock **OP**
**McGuire, William P**.....105 N. Braddock st. **OP**
Smith, George H. Jr.....114 W. Boscawen St. **ALR**

## WASHINGTON

**BELLINGHAM**

Kaiser, Robert F.....1705 Knox ave. **ALR**

**BREMERTON**

Benson, C. E.....515 6th St. **OALR**

**COLFAX**

**Henry, Robert T**.....Grady bldg. **OP**

**ELLENSBURG**

**Hicks, William Wynne**.....202 E. 6th st. **ALR**

**EVERETT**

**Murphy, Albert B**.....Medical and Dental bldg. **ALR**
**Secoy, Harry R**.....Central bldg. **ALR**
Waltz, Harold D.....510 Medical Dental bldg. **ALR**

**LONGVIEW**

**Barton, John F**.....1346 Fourteenth av. **ALR**

**RICHLAND**

Davis, Leo L.....1509 Farrell Lane **OP**

**SEATTLE**

Chase, E. Frank.....Cobb bldg., Z1 **OALR**
Drell, Maurice Joseph.....630 Medical Arts bldg., Z1 **OP**
**Firestone, Charles**.....326 Medical Dental bldg. **ALR**
Goss, Harold Leroy.....1305 4th Ave., Z1 **OP**
Hanson, A. George.....1528 Medical Dental bldg., Z1 **OP**
Hoffman, W. F.....1317 Marion, Z4 **OP**
Horsfield, Russell Thomas, 715 Medical & Dental bldg., Z1 **OP**
**Jensen, Carl Dana**.....1315 Medical & Dental bldg. Z1 **OP**
Johnson, Roger Harry.....522 Cobb bldg. **OP**
McCoy, L. L.....1317 Marion, Z4 **OP**
**Miller, Alvin R**.....Fourth and Pike bldg., Z1 **ALR**
**Peden, Barton Edward**.....Stimson bldg., Z1 **ALR**
Powell, Archie C.....Stimson bldg., Z1 **ALR**
Shannon, C. W.....New 4th and Pike bldg., Z1 **OP**
Stillson, H.....Seabord bldg., Z1 **OALR**
Tolan, John F.....1115 Terry, Z5 **ALR**
Weber, Julius Albert.....Stimson bldg., Z1 **ALR**

**SPOKANE**

deRoetth, Andrew F.....508 Old Nat'l Bank bldg. **OP**
Greene, Philip Burkland.....517 Old Nat'l Bank bldg. **OP**
Hagan, Cornelius E., Jr.....1918 S. Oneida **ALR**
Hogsett, Smith Fuller.....1581 Paulsen Medical bldg. **OP**
Lower, Raymond A.....1455 Paulsen Med. Arts bldg. **ALR**
Parsell, Louis A.....Paulsen Med. and Dental bldg., Z8 **ALR**
Pohl, Robert L.....755 Paulsen Med. & Dental bldg., Z8 **ALR**
Rott, O. M.....1828 W. 1st av., Apt. C, Z9 **OALR**
Smith, Carroll L.....Paulsen Med. and Dental bldg., Z8 **OALR**
Veasey, C. A., Sr.....Paulsen Med. and Dental bldg. Z8 **OALR**
Veasey, C. A., Jr.....Paulsen Med. and Dental bldg., Z8 **OALR**
Wilmes, M. N.....960 Paulsen Medical & Dental bldg. **ALR**

**TACOMA**

Busby, William, Jr.....Madigan Gen. Hosp. **ALR**
Cameron, Walter Courtney.....1103 Medical Arts bldg. **OP**
**Howe, A. W**.....Medical Arts bldg., Z2 **OALR**
**Ludwig, William Henry**.....Medical Arts bldg., Z2 **ALR**
**Lundvick, Cyril V**.....Medical Arts bldg., Z2 **OP**
Maier, Haskel Levitt.....936 Medical Arts bldg., Z2 **ALR**
**Paul, William C**.....Medical Arts bldg., Z2 **ALR**

**VANCOUVER**

**Dunnavan, Floyd Leighton**.....Arts bldg. **OP**

**WALLA WALLA**

Ralston, Frank L.....120 E. Birch St. **OALR**
**Stevens, Ralph W**.....302-12 Baker bldg. **OP**

**WENATCHEE**

**Miller, Claude K**.....Widby-Hadley bldg. **OP**

**YAKIMA**

Thompson, James P.....532 Miller bldg. **OP**

## WEST VIRGINIA

**BECKLEY**

**Peter, Beverly K**.....Beckley Hospital **ALR**

**BLUEFIELD**

**Blaydes, J. E**.....First Nat'l Bank bldg. **OP**
**McGuire, John**.....Peery bldg. **ALR**

## WEST VIRGINIA—Continued

CHARLESTON
Armentrout, Lewis H.....Medical Arts bldg. ALR
Breisacher, Carl F.....432 Medical Arts bldg. Z1 OP
Chandler, Arthur C.....1562 Virginia st. E., Z11 OP
Holcombe, Virgil Eugene.....Medical Arts bldg., Z1 ALR
Lance, Verne L.....1113 Quarrier ALR
McLaughlin, Ralph S.....805 Atlas bldg. OP
Reel, Frederick Clifton.....432 Medical Arts bldg., Z1 ALR
Seletz, Abraham.....Medical Arts bldg., Z1 ALR

CHARLES TOWN
Barlow, Roy.....R. F. D. No. 2 ALR

CLARKSBURG
Hall, Sobisca S.....Empire Bank bldg. ALR
Thomas, Harry Vernon.....Empire Bank bldg. OP

ELKINS
Goodwin, Thomas M.....Golden Clinic ALR

FAIRMONT
Johnson, H. R.....320 Jefferson OALR
Maxwell, Joseph S.....323 Adams St. OP
Traugh, George H.....309 Cleveland av. ALR

HUNTINGTON
Beckner, Wm. Forrest.....West Virginia bldg., Z1 ALR
Hoitash, Frederick J.....P. O. Box 1066 Z13 OP
Krimsky, Joseph.....2924 Winters Rd. OALR
McGehee, Melvin W.....425 11th st. ALR
Moore, J. Hallock..1229 1st Huntington Nat'l Bank bldg. ALR
Moore, T. W.....1050 Fifth av., Z1 OP
Polan, Charles M.....1042 6th Ave., Z1 OP

MORGANTOWN
Wylie, C. B.....Box 224 OALR

PARKERSBURG
Gilmore, Milton A.....Box 172 OP
Hartman, Eugene C.....610½ Market OALR

WELCH
Schiefelbein, Harry T.....Stevens Clinic Hospital OP

WESTON
Long, Albert Emanuel.....169 Main St. OP

WHEELING
Fawcett, Ivan.....75 Twelfth st. OALR
Jones, Edmund Lloyd.....Wheeling Clinic OALR
Joseph, Nime K.....40-14th st. ALR
Stewart, James K.....Wheeling Clinic ALR
Tomassene, R. A.....1144 Market OALR
Zubak, Matthew F.....704-9 Central Union bldg. OALR

## WISCONSIN

APPLETON
Brooks, E. H.....Zuelke bldg. OALR
Rector, A. E.....Zuelke bldg. OALR

ASHLAND
Grigsby, Roll O.....100 E. Second ALR

BELOIT
Shearer, Harry Arthur.....405 E. Grand av. ALR

EAU CLAIRE
Payne, A. L.....119 S. Barstow OALR
Russell, Sidney Benteen.....Midelfart Clinic OALR
Tanner, James Wallace.....131 S. Barstow ALR

FOND DU LAC
Cerny, Frank Joseph.....160 Bischoff st. OP
Folsom, W. H.....11 N. Main OALR

GREEN BAY
Ford, William W.....109 East Walnut ALR
Nadeau, E. G.....Bellin bldg. OALR
Troup, Wilson J.....306 Cherry st. OALR

JANESVILLE
Pember, Aubrey H.....508 W. Milwaukee OALR

KENOSHA
Ashley, Richard Warner.....5811 8th Ave. OP
Ashley, T. W.....5711 Eighth av. OALR
Hartgraves, Hallie.....St. Mary's Convent OP

LA CROSSE
Douglas, Frederick Alexander.....State Bank bldg. OALR
Ridout, George B.....1836 South av. ALR
Smith, D. S.....State Bank bldg. OALR

MADISON
Aageson, Carl W.....1 South Pinckney st. OP
Brindley, Benjamin I.....304 W. Washington, Z3 OP
Davis, Frederick Allison....224 W. Washington av., Z3 OP
Dimond, Waldo B.....701-710 Gay bldg. ALR
Duehr, Peter Alexander.....224 W. Washington av., Z3 OP
Neff, E. Eugene.....224 W. Washington av., Z3 OP
Nesbit, Mark E.....616 1st Nat'l Bank bldg., Z3 OP
Nesbit, Wellwood.....1 S. Pinckney, Z3 ALR
Schubert, Clarence K.....1 S. Pinckney, Z3 OP
Smith, R. B.....224 W. Washington av., Z3 ALR

MANITOWOC
Andrews, M. P.....826 S. 8th OALR

MARINETTE
May, J. V.....1703 Main OALR

MARSHFIELD
Copps, Lyman Alden.....Marshfield Clinic OALR

MILWAUKEE
Bach, Edwin Clarence.....324 E. Wisconsin av., Z2 ALR
Blankstein, Samuel S., Bankers bldg., 208 E Wisc. av., Z2 OP
Brook, Jeffrey J.....231 West Wisconsin av., Z3 ALR
Cannon, Harold J.....536 W. Wisconsin av., Z3 ALR
Carl, Evan Elsworth.....606 W. Wisconsin av., Z3 OP
Fox, Meyer S.....208 E. Wisconsin av., Z2 ALR
Grossman, Erwin E.....238 W. Wisconsin av. OP
Grove, W. E.....324 E. Wisconsin av., Z2 OALR
Haessler, F. Herbert.....Wells bldg., Z2 OP
Heeb, Harry J.....212 W. Wisconsin av., Z3 OALR
Higgins, S. G.....411 E Mason, Z2 OALR
High, Howard Charles.....324 E. Wisconsin ave., Z2 ALR
Hitz, John B.....411 E. Mason st., Z2 OP
Judge, Thomas Ambrose.....735 N. Water, Z2 ALR
Light, George.....709 N. 11th, Z3 ALR
Martin, Hilmar George.....231 W. Wisconsin av., Z3 OP
McCormick, Thomas F.....Wells bldg., Z2 ALR
Mings, Dwain Edward.....1684 N. Prospect Ave. OP

### WISCONSIN — Continued

**Molsberry, Jasper Merl**.....425 E. Wisconsin av., Z2 **OP**
Mulsow, Jack E.....Shorecrest Hotel **OALR**
**Schmidt, Herbert G.**.....411 E. Mason, Z2 **OALR**
**Schoofs, Orlando P.**.....324 E. Wisconsin av., Z2 **ALR**
**Sproule, Ralph P.**.....208 E. Wisconsin av., Z2 **OALR**
Straus, Gerhard D.....4459 N. Murray Ave. **ALR**
**Tolan, Thomas L.**.....Wells bldg., Z2 **ALR**
**Treskow, Frank G.**.....411 E. Mason, Z2 **ALR**
Truitt, John William.....161 W. Wisconsin **ALR**
**Waldeck, Edward A.**.....238 W. Wisconsin av., Z3 **OALR**
Warner, Raymond C.....1684 N. Prospect Ave., Z2 **OP**
Wild, Joseph Peter.....161 W. Wisconsin Ave., Z3 **ALR**
Wynn, Sidney K.....759 No. Milwaukee St., Z2 **ALR**

MONROE
**Kundert, Fred W.**.....The Monroe Clinic **ALR**

OSHKOSH
**Ebert, Reinhold O.**.....64 Mt. Vernon st. **OP**
Emrich, Paul Stanley.....64 Mt. Vernon **ALR**

RACINE
Covell, Kermit Wendell.....610 Main Street **ALR**

SHEBOYGAN
**Neumann, William H.**.....Sheboygan Clinic **OALR**

STEVENS POINT
**Anderson, George H.**.....111½ Strongs av. **ALR**
Dunn, Austin Gibbons.....111½ Strongs av. **ALR**

SUPERIOR
Myers, Elmer A.....1708 Hammond av. **OALR**

WAUSAU
**Trumbo, James Keeley**.....P. O. Box 485 **ALR**
Wilson, Otis M.....502 3rd st. **OP**

WHITEHALL
**Simons, Neal S.**.....MacCornack Clinic **ALR**

WISCONSIN RAPIDS
Barnet, Emmanuel.....214 W. Grand Ave. **ALR**

### WYOMING

CASPER
**James, George Russell**.....Wyoming Nat'l Bank bldg. **OP**
Nelson, John Russell.....Wyoming Nat'l Bank bldg. **ALR**

CHEYENNE
**Newman, Erwin W.**.....1606 Capitol av. **OP**
Stump, Robert Byron.....408 Hynds bldg. **OP**
**Williams, Russell I.**.....422 Hynds bldg. **ALR**

SHERIDAN
**Veach, Oscar Lloyd**.....Whitney Trust bldg. **OP**

## U. S. TERRITORIES

### HAWAII

HILO
**Crawford, H. E.**.....139 Kinoole st. **OP**

HONOLULU
**Corboy, Phillip M.**.....565 Young Bldg. Z9 **OP**
Lynam, Patrick J.....1415 Kalakaua Ave., Z19 **ALR**
Moffat, Harold F.....1415 Kalakaua Ave., Z19 **OP**
Pinkerton, Forrest Joy.....Young Hotel bldg., Z9 **OALR**
**Trexler, C. W.**.....50 Dowsett av. **ALR**
Van Poole, G. McD.....Young bldg., Z9 **OALR**
Wong, Robert Tuck.....1147 Union St. **OP**

### PUERTO RICO

SAN JUAN
**Fernandez, Luis J.**.....Box 2206, Z10 **OP**
Fernandez, Ricardo F.
15 Ponce de Leon av., Box 2206 Z10 **OP**
Font, Juan H., P. O. Box 5363,
Puerta de Tierra Station, Medical Arts bldg. **ALR**

SANTURCE
Munoz-MacCormick, Carlos Eduardo
Monterrey bldg., Park Terrace **ALR**

UTUADO
Carrasquillo, Honorio Fernando.....P. O. Box 128 **OP**

## CANADA

### ALBERTA

CALGARY
Dowling, Harvey E.....315 Southam bldg. **OP**
Miller, Meyer S.....213 Fitzpatrick bldg. **ALR**

### BRITISH COLUMBIA

VANCOUVER
Boucher, R. B.....Medical-Dental bldg. **OALR**
Brown, W. Harold ..... 326 Medical-Dental bldg., **OALR**
Davies, Charles Edgar.....925 West Georgia St. **OP**
Grimmett, Roger Skelton.....5038 Newton Wynd. **ALR**
Paton, Walter M.....919 Medical Arts bldg. **ALR**

VICTORIA
Grieve, Harry Gordon.....315 Scollard bldg. **OALR**

### MANITOBA

BRANDON
McDiarmid, H. Oliver.....35 Clement block **OALR**

WINNIPEG
Macneil, Frank A.....Medical Arts bldg. **ALR**
Ramsey, Robert M.....1237 Wellington Crescent **OP**

### NEW BRUNSWICK

ST. JOHN
Hayes, Robert T.....42 Coburg **ALR**

### NOVA SCOTIA

HALIFAX
**Schwartz, Hugh William**.....183 S. Park **ALR**

WOLFVILLE
Chipman, L. DeV.....44 Westwood ave. **OALR**

### ONTARIO

CAMP BORDEN
McLeod, Angus M., Capt. (MC)
**Camp Borden Military Hospital ALR**

HAMILTON
**Macfarlane, P. B.**.....Medical Arts bldg. **OALR**
**Rabinowitz, Paul**.....Mountain Sanitarium **ALR**

## ONTARIO — Continued

LONDON
McFarlane, Donald C.....380 Queens av. ALR

OTTAWA
Fraser, W. G.....Medical Arts bldg. OALR
Smart, Robert Edwin.....Medical Arts bldg. OP

PETERBOROUGH
Buchanan, Norman D.....129 Murray OALR

SARNIA
Biehn, J. Telford.....282 Wellington St. OALR

TORONTO
Alexander, Seeyman L.....Medical Arts bldg., Z5 ALR
Atkins, John.....345 Bloor st. W. ALR
Barnes, Lancelot.....170 St. George, Z5 OP
Bochner, Maxwell K.....329 Bloor st. W. OP
Burnham, Howard Hampden.....Medical Arts bldg., Z5 ALR
Campbell, A. A.....86 Bloor st. W., Z5 ALR
Davies, T. A.....578 Sherbourne OALR
Elliot, Alfred J.....170 St. George st. OP
Goldsmith, Perry G.....Medical Arts bldg., Z5 ALR
Gollom, Joseph.....425 Bloor St. W., Z5 ALR
Henry, George Arnold.....Medical Arts bldg. Z5 ALR
Hill, C. E.....160 Bloor st W., Z5 OP
Ireland, Percy E.....330 Medical Arts bldg. ALR
Luke, William R. F.....Medical Arts bldg., Z5 OP
McCart, Howard W. D.....170 St. George st., Z5 ALR
McCulloch, Robert J. P.....Medical Arts bldg., Z5 OALR
McEachern, Margaret Aileen.....Medical Arts bldg., Z5 ALR
McGregor, Gregor William.....170 St. George, Z5 ALR
Morgan, Alonzo Lloyd.....170 St. George, Z5 OP
Risdon, E. F.....Medical Arts bldg., Z5 ALR-PI S
Robert, Joseph X.....170 St. George, Z5 ALR
Smith, I. Ruebert.....116 College st. ALR
Snell, George C.....Medical Arts bldg., Z5 ALR
Stark, W. Berkeley.....Medical Arts bldg., Z5 ALR
Sullivan, J. A., Wing Comdr. (RCAF),
Medical Arts bldg., Z5 ALR
Vaile, Ivan Royle, .....384 Bloor st. W. ALR
Veitch, Ambert H.....170 St. George, Z5 ALR
Wishart, David Edmund Staunton.Medical Arts bldg., Z5 ALR
Wright, Walter Walker.....Medical Arts bldg., Z5 OP

WELLAND
Briant, Thomas Ernest.....28 East Main ALR

## QUEBEC

DRUMMONDVILLE
Duclos, Gaston Normand.....3 DuMoulin St. ALR

MONTREAL
Ballon, David H.....1022 Medical Arts bldg., Z25 ALR
Freedman, Abraham O.....1396 St. Catherine st. W. ALR
Hodge, George E.....1414 Drummond ALR
Hutchison, Keith O.....1509 Sherbrooke st. W. ALR
Johnston, Kenneth B.....1509 Sherbrooke st. W. OP
Lapointe, Jean.....300 Carre, St. Louis ALR
Lundon, A. E.....1463 Bishop st., Apt. C-3 ALR
MacMillan, J. A.....1410 Stanley, Z2 OP
McAuley, Albert G.....1301 Dorchester st. W., Z25 OP
McNally, William James.....Linton Apartments ALR
Ramsey, G. Stuart.....1496 Mountain OP
Rogers, J. T.....Medical Arts bldg. ALR
Rosenbaum J.....1396 St. Catherine st. W. OP
Steine, Ben Zion.....Medical Arts bldg. ALR
Tremble, G. Edward.....1390 Sherbrooke St. W. ALR
Viger, Roland Joseph.....1414 Drummond St. OP
Wright, R. Percy.....1414 Drummond st. ALR

## SASKATCHEWAN

REGINA
Haughton, Thomas J.....313 McCallum-Hill bldg. ALR

# ASIA

## INDIA

MUNGELI, CENTRAL PROVINCES
Rambo, Victor Clough
Christian Hospital via Bilaspur B.N. Ry. OP

# EUROPE

## AUSTRIA

WIEN
Lauber, Prof. Dr. Hans.....Grillparzerstrabe 7/15 OP

## ENGLAND

LEEDS
Parsons, Sir John Herbert..Meads, Wigton Lane, Alwoodley OP

## HOLLAND

LEIDEN
van der Hoeve, Prof. J.....Rijnsburgerweg 11 OALR

## SWITZERLAND

ZURICH
Nager, Prof. F.....Freiestrasse 30 OALR

# LATIN AMERICA

## ARGENTINA

CORDOBA
Zavalia, Alberto Urrets.....27 de Abril 24 OP

## BRAZIL

SAO PAULO
Alvaro, M. E.....1151 Consolacao OP

## COSTA RICA

SAN JOSE
Aguero, Alexis ..... OP

## MEXICO

MEXICO, D. F.
Tapia y Fernandez, Ricardo.....4a de Londres, 67 ALR

## URUGUAY

MONTEVIDEO
Alonzo, Justo M.....Mercedes 1233 ALR

## MEMBERS SERVING WITH THE ARMED FORCES OVERSEAS

Baird, Charles G., Col. (MC)
AAG Nanking China, APO 909 c/o PM, San Francisco, Cal.OALR

Loy, A. W., Capt. (MC) USN
Navy No. 100, c/o FPO, New York, N.Y. OP